# Characterization of the Genetic Resistance to *Colletotrichum lindemuthianum* in Common Bean Differential Cultivars

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## ABSTRACT

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The inheritance of genetic resistance to bean anthracnose in genotypes Catrachita and SEL 1360 derived from two anthracnose differential cultivars, AB 136 and G 2333, respectively, is described. Segregation data from three different $F_2$ populations and their respective $F_{2:3}$ families indicated that a single dominant gene is responsible for the anthracnose resistance in Catrachita. In the test for allelism, chi-square test confirmed that the single dominant resistance gene in Catrachita was situated at a different locus from previously characterized resistance genes *A*, *Are, Mexique 1, Mexique 2,* and *Mexique 3*. It is proposed that the single dominant resistance gene present in Catrachita be assigned the genetic symbol *Co-6*, *Co* for *Colletotrichum* and *6* because it is the sixth major anthracnose resistance gene characterized and reported in the literature. Segregation in the three $F_2$ populations where SEL 1360 was used as the resistant parent fitted a 3:1 (R-:rr) ratio and a 1:2:1 (RR:Rr:rr) ratio in the $F_{2:3}$ families. Segregation data suggested that a single dominant gene was conditioning resistance to anthracnose in SEL 1360. The test for allelism involving SEL 1360 indicated that the single dominant gene in SEL 1360 is independent from *A* (*Co-1*), *Are* (*Co-2*), *Mexique 1* (*Co-3*), and *Mexique* 2 (*Co-4*) genes. However, the dominant gene in SEL 1360 did not segregate independently from the resistance gene *Mexique 3* in the differential cultivar TU, demonstrating that both dominant alleles are located at the same locus. Deployment of major genes of Middle American origin, such as *Co-6* and *Mexique 3* (*Co-5*), in different combinations with other characterized genes of Andean origin is possible and should contribute to more durable anthracnose resistance in common bean.


Anthracnose, caused by *Colletotrichum lindemuthianum* (Sacc. & Magnus) Lams.-Scrib., is one of the most widespread and economically important fungal diseases of common bean (*Phaseolus vulgaris* L.). Complete yield loss can occur with susceptible genotypes when conditions favorable for the pathogen are present during the growing season (20). Since *C. lindemuthianum* is a seedborne pathogen that exhibits extensive physiological variability, the use of genetic resistance has been the most effective control strategy implemented in Europe and North America (5,9,24). The extensive genetic variation in common bean is associated with two main centers of domestication known as the Andean (South America) and Middle American (Mexico and Central America) gene pools (23). In the United States, the control of bean anthracnose by means of genetic resistance has relied primarily on the deployment of two genes, *A* (Andean gene) and *Are* (Middle American gene) (9). The single dominant *A* gene was the first genetically characterized gene reported to confer resistance to race alpha (2). The *Are* gene, which conditions resistance to six distinct physiological races (alpha, beta, delta, epsilon, gamma, and lambda) of *C. lindemuthianum* (24), was described as a monogenic dominant factor by Mastenbroek (13). In Europe, Fouilloux (5) identified three additional single dominant resistance genes in a collection of Mexican germ plasm. Allelism analysis showed that these Middle American genes segregated independently from each other and from the *Are* gene, and they were designated *Mexique 1, Mexique 2,* and *Mexique 3*.

Breakdown of the genetic resistance, leading in some cases to disease epidemics caused by races of *C. lindemuthianum* virulent on the *Are* or *A* resistance genes, has been observed in different parts of the world, including North America (9,12,15,25). Because the protection conferred by single genes is potentially short-term, a need for diverse sources of genetic resistance is widely recognized among bean breeders. Using a diverse collection of isolates of the pathogen, scientists at CIAT (Centro Internacional de Agricultura Tropical), Colombia, South America, screened several thousand bean germ plasm accessions and identified new sources of genetic resistance (19,20,22). Two members of the differential set of 12 cultivars, AB 136 and G 2333, used to categorize isolates of *C. lindemuthianum* (17), were consistently among the germ plasm accessions that showed the broadest resistance. Independent studies on the genetic characterization of these two differential cultivars have been conducted. According to Gonçalves-Vidigal (7), a monogenic dominant factor present in AB 136 was responsible for the resistance to races alpha, delta, and kappa, while two independent dominant genes conditioned resistance to race 521 in G 2333 (18). Neither study reported on the independency of the individual genes nor on the relationship of these dominant genes to other previously characterized resistance genes.

In the present study, the inheritance of resistance to different races of bean anthracnose in lines derived from two differential cultivars, AB 136 and G 2333, is described. The relationship of the resistance factors in AB 136 and G 2333 with other previously characterized resistance genes is analyzed. Finally, strategies for improving the long-term durability of genetic resistance to bean anthracnose are discussed.

## MATERIALS AND METHODS

**Plant material.** Three breeding lines derived from crosses involving two differential cultivars (AB 136 and G 2333) were used to characterize the genetic resistance. A CIAT breeding line RAB 205 (BAT 1225/AB 136), released in Honduras as the cultivar Catrachita, was used as the source of resistance from AB 136 (1) (Table 1). CIAT breeding lines SEL 1360 and SEL 1308 (Talamanca * 2/G 2333), were used as the source of the resistance genes from the differential cultivar G 2333 (4) (Table 1). It was observed that SEL 1308, when challenged with a wide group of races of the pathogen, displayed a different resistance reaction from SEL 1360. This suggested that SEL 1308 carried a resistance factor(s) different from SEL 1360 (Table 1). The characterization of the resistance in SEL 1308 is still underway, and those findings will be reported elsewhere. The derived lines were used because of their adaptation to growing conditions at East Lansing, MI. Synchronization of flowering for crossing purposes was facilitated using Catrachita and the SEL lines instead of the original parental genotypes, which are photoperiod sensitive. The Michigan cultivars—Black Magic (8), Raven (10), and Blackhawk (6)—were each crossed to Catrachita and SEL 1360. Black Magic is

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susceptible to race 64 (alpha) and race 73; Raven carries the *A* gene for resistance to races 64 and 73 but is susceptible to race 7; and Blackhawk carries the *Are* gene for resistance to races 7, 23 (delta), and 64 but is susceptible to race 73 (Tables 1 and 2). The six $F_2$ populations developed and their respective $F_{2:3}$ families were used to determine the inheritance of genetic resistance in both derived lines. For the test of allelism, 10 $F_2$ populations were developed using Catrachita and SEL 1360 in test crosses with a series of cultivars—Raven, Blackhawk, Mex 222, TO, and TU—carrying independently characterized genes *A*, *Are*, *Mexique 1*, *Mexique 2*, and *Mexique 3*, respectively; (5,6,10). The genetic independence of the new resistance sources was also evaluated in the cross of Catrachita × SEL 1360.

**Preparation of races of *C. lindemuthianum*.** Inoculum of each race used in the present study was obtained from monosporic cultures grown and maintained in stocks of fungus-colonized filter paper for long-term storage. The identity of each race was first confirmed by the phenotypic reaction displayed with the anthracnose differential set (17). The numerical system used to identify different races is based on the sum of the binary values assigned to each of the 12 differential cultivars on which the specific race is pathogenic (3). Parental genotypes, original (AB 136 and G 2333) and derived (Catrachita, SEL 1360, and SEL 1308) sources of resistance were screened with a collection of races of the pathogen (Table 1). Based on the different phenotypic reactions observed on these genotypes, specific races of *C. lindemuthianum* were chosen for study of the genetic characterization of the resistance. Different races were used for the allelism test (Table 2). Race 64 (ATCC 18987), race 23 (ATCC 18989), and race 73 (ATCC 96512) were selected to determine the inheritance of resistance in Catrachita and SEL 1360 when crossed to the cultivars Black Magic, Raven, and Blackhawk, respectively. Race 64 was selected to test for allelism in all segregating populations, except in Mex 222 × Catrachita and Mex 222 × SEL 1360 populations, where race 7 (ATCC 96390) was used. In addition to race 64, race 7 was used in an independent test for allelism in the TU × SEL 1360 population. Inocula of all races except race 73, which was grown on Mathur's agar (14), were prepared by placing fungus-colonized filter paper in petri dishes containing potato-dextrose agar. All cultures were incubated for 10 days under complete darkness at 24°C. Spore suspensions were prepared by flooding the plates with 5 ml of 0.01% Tween 20 in sterile distilled water and scraping the surface of the culture with a spatula.

**Disease phenotypic characterization.** Parental lines, $F_1$, $F_2$, and $F_{2:3}$ families were inoculated with a spore suspension ($1.2 \times 10^6$ spores $ml^{-1}$) of the pathogen. The race of *C. lindemuthianum* used to characterize the resistance factors segregating in each cross and the respective generations tested are shown in Table 2. The protocol for spore inoculation was as follows: The first incompletely expanded trifoliate leaf of 15- to 18-day-old parental $F_1$ or $F_2$ plants was sprayed on the lower and upper leaf surfaces. Spore suspensions were applied with a camel-hair paint brush. Conversely, $F_2$ populations for the allelism test and $F_{2:3}$ families were spray-inoculated with the spore suspension on the abaxial and stem surface of unifoliate leaves (10- to 12-day-old plants). Different inoculation methods were used because $F_2$ individual plants (particularly susceptible plants), unlike $F_{2:3}$ plants and $F_2$ populations used for the allelism test, were saved for $F_3$ seed production. Inoculated plants were placed in a mist chamber at 100% RH and 22 to 25°C for 48 h, then transferred to greenhouse conditions under supplemental lighting (18-h day length). Seven days postinoculation, symptom expression was evaluated and phenotypic conditions were assigned. Host reaction was rated on a 1 to 9 scale in which 1 = no visible symptoms and 9 = severely diseased or dead (27). Resistant (R) phenotype was assigned to plants with no or limited disease symptoms (scores 1 to 3); whereas plants with numerous enlarged lesions or sunken cankers on the lower side of leaves or hypocotyls were considered susceptible (S) (scores 4 to 9).

## RESULTS

Differential cultivars and derived lines possessing anthracnose resistance were evaluated for their phenotypic reaction to a collection of races of the pathogen (Table 1). Different races (7, 23, 64, 73, 1545, and 2047), representing a wide range of virulence, were used to confirm whether Catrachita and SEL lines were comparable to the anthracnose resistance present in AB 136 and G 2333 differential cultivars. The cultivars Catrachita and AB 136 exhibited similar phenotypic patterns: both showed resistance to all races except 1545 and 2047. None of the races tested were pathogenic on G 2333 and its derived line SEL 1308. However, SEL 1360 exhibited a different phenotypic pattern from the G 2333 parent. Two races, 1545 and 2047, overcame the resistance factor in this derived line. Since SEL 1308 and SEL 1360 were derived from G 2333 through backcrossing to the susceptible variety Talamanca, it is assumed that the resistance

**Table 1.** Phenotypic characterization of parental (Black Magic, Raven, and Blackhawk) and differential (Mex 222, TO, TU, AB 136, and G 2333) cultivars and derived (Catrachita, SEL 1360, and SEL 1308) lines for their reactions to different races of *Colletotrichum lindemuthianum*

| | Race and phenotypic reaction[a] | | | | | |
|---|---|---|---|---|---|---|
| Genotype | 7 | 23 | 64 | 73 | 1545 | 2047 |
| Black Magic | S | R | S | S | S | S |
| Raven | S | S | R | R | R | S |
| Blackhawk | R | R | R | S | S | S |
| Mex 222 | R | R | S | S | R | S |
| TO | R | R | R | R | R | S |
| TU | R | R | R | R | S | S |
| AB 136 | R | R | R | R | S | S |
| Catrachita | R | R | R | R | S | S |
| G 2333 | R | R | R | R | R | R |
| SEL 1308 | R | R | R | R | R | R |
| SEL 1360 | R | R | R | R | S | S |

[a] R = resistant; S = susceptible.

**Table 2.** Crosses, generations, and races of *Colletotrichum lindemuthianum* used for the genetic characterization of resistance in Catrachita and SEL 1360

| Cross | | Generation[a] | Race[b] |
|---|---|---|---|
| Black Magic × Catrachita | (S/R)[c] | P, $F_1$, $F_2$, $F_{2:3}$ | 64 |
| Raven × Catrachita | (S/R), (R/R) | P, $F_1$, $F_2$, $F_{2:3}$ | 23, 64 |
| Blackhawk × Catrachita | (S/R), (R/R) | P, $F_1$, $F_2$, $F_{2:3}$ | 73, 64 |
| Mex 222 × Catrachita | (R/R) | P, $F_1$, $F_2$ | 7 |
| TO × Catrachita | (R/R) | P, $F_1$, $F_2$ | 64 |
| TU × Catrachita | (R/R) | P, $F_1$, $F_2$ | 64 |
| Black Magic × SEL 1360 | (S/R) | P, $F_1$, $F_2$, $F_{2:3}$ | 64 |
| Raven × SEL 1360 | (S/R), (R/R) | P, $F_1$, $F_2$, $F_{2:3}$ | 23, 64 |
| Blackhawk × SEL 1360 | (S/R), (R/R) | P, $F_1$, $F_2$, $F_{2:3}$ | 73, 64 |
| Mex 222 × SEL 1360 | (R/R) | P, $F_1$, $F_2$ | 7 |
| TO × SEL 1360 | (R/R) | P, $F_1$, $F_2$ | 64 |
| TU × SEL 1360 | (R/R), (R/R) | P, $F_1$, $F_2$ | 64, 7 |
| Catrachita × SEL 1360 | (R/R) | P, $F_1$, $F_2$ | 64 |

[a] Parental, $F_1$, $F_2$, and $F_{2:3}$ families, respectively.
[b] Races of *C. lindemuthianum* used for the genetic characterization and the allelism test, respectively.
[c] Parental resistant (R) or susceptible (S) reaction to the respective races shown in column four.

factor(s) observed in both lines came from the G 2333 parent. Since G 2333 carries two dominant resistance genes (18), it is possible that only one of the major genes was inherited in SEL 1360; thus a different phenotypic pattern was observed. Although similar disease reactions were observed between SEL 1308 and G 2333, it is not certain, in the absence of test crosses, if SEL 1308 inherited both genes responsible for anthracnose resistance of G 2333.

The nature of the resistance in Catrachita and SEL 1360 was determined after analyzing the observed segregating ratios obtained from the disease phenotypic characterization of the three different $F_2$ populations and their respective $F_{2:3}$ families. In the crosses of Catrachita as a parent with Black Magic, Raven, and Blackhawk, the chi-square test performed on $F_2$ populations supported a fit to the 3:1 expected ratio of resistant to susceptible plants (R-:rr), respectively (Table 3). The chi-square test performed on the $F_{2:3}$ families confirmed a 1:2:1 (RR:Rr:rr) ratio in each $F_2$ population, indicating that a single dominant gene is responsible for the anthracnose resistance in Catrachita (Table 3). The allelism study performed to test the independence of the single dominant resistance gene in Catrachita with the other five reported genes (*A, Are, Mexique 1, Mexique 2,* and *Mexique 3*) is shown in Table 4. In all five $F_2$ populations, the observed segregation fits a 15:1 (R-:rr) ratio, indicating that the single dominant gene conditioning resistance to anthracnose in Catrachita segregated independently from the *A, Are, Mexique 1, Mexique 2,* and *Mexique 3* dominant genes.

Segregation in the three $F_2$ populations where SEL 1360 was the resistant parent fits a 3:1 ratio of R- to rr, respectively. A 1:2:1 ratio was confirmed in $F_{2:3}$ families from each $F_2$ population evaluated, suggesting that a single dominant gene was conditioning resistance to anthracnose in SEL 1360 (Table 3). Within four $F_2$ populations tested for allelism, the segregation ratio fitted a 15:1 (R-:rr) ratio for the crosses of SEL 1360 with Raven, Blackhawk, Mex 222, and TO. This allelism test indicated that the single dominant resistance gene in SEL 1360 is independent and located at a different locus from the *A, Are, Mexique 1,* and *Mexique 2* genes (Table 4). However, no segregation was observed in the $F_2$ population derived from the cross TU × SEL 1360 after two independent inoculations with races 64 and 7. This result indicated that the dominant gene in SEL 1360 is located at the same locus as the *Mexique 3* gene in TU. In addition, similar phenotypic reactions were observed in the differential cultivar TU and SEL 1360 when evaluated with a group of anthracnose races (Table 1), supporting the finding that SEL 1360 and TU both carry the *Mexique 3* dominant allele. The test of allelism performed on the $F_2$ population of the cross Catrachita × SEL 1360 segregated in a ratio of 15:1 ($P$ = 0.09), indicating that the dominant resistance gene in Catrachita is independent from the resistance gene in SEL 1360 (Table 4).

**Table 3.** Phenotypic characterization and observed ratio of $F_2$ progeny and $F_{2:3}$ families for their reactions to *Colletotrichum lindemuthianum*

| | Anthracnose reaction | | | | | | |
|---|---|---|---|---|---|---|---|
| | $F_2$[b] (no. plants) | | | $F_{2:3}$[c] (no. plants) | | | |
| Cross[a] | R- | rr | P | RR | Rr | rr | P[d] |
| Black Magic × Catrachita[e] | 74 | 32 | 0.26 | 23 | 50 | 32 | 0.41 |
| Raven × Catrachita[f] | 76 | 34 | 0.19 | 28 | 47 | 34 | 0.26 |
| Blackhawk × Catrachita[g] | 79 | 32 | 0.41 | 23 | 55 | 31 | 0.55 |
| Black Magic × SEL 1360[e] | 84 | 27 | 1.00 | 29 | 54 | 27 | 0.95 |
| Raven × SEL 1360[f] | 89 | 22 | 0.25 | 35 | 51 | 21 | 0.14 |
| Blackhawk × SEL 1360[g] | 84 | 27 | 1.00 | 33 | 49 | 27 | 0.41 |

[a] Susceptible × resistant crosses in all cases.
[b] 3:1 (resistant, R- : susceptible, rr).
[c] 1:2:1 (resistant, RR : resistant, Rr : susceptible, rr) expected ratio, respectively.
[d] $P$ = estimated probability value.
[e] Phenotypic reaction to race 64 of *C. lindemuthianum.*
[f] Phenotypic reaction to race 23 of *C. lindemuthianum.*
[g] Phenotypic reaction to race 73 of *C. lindemuthianum.*

**Table 4.** Allelism test for genetic characterization of the resistance to *Colletotrichum lindemuthianum* in Catrachita (AB 136) and SEL 1360 (G 2333)

| | Anthracnose reaction | | | |
|---|---|---|---|---|
| | No. $F_2$ plants | | | |
| Cross[a] | R- | rr | $X^2$[b] | P[c] |
| Raven × Catrachita[d] | 114 | 3 | 2.12 | 0.15 |
| Blackhawk × Catrachita[d] | 119 | 14 | 3.45 | 0.06 |
| Mex 222 × Catrachita[e] | 104 | 5 | 0.27 | 0.60 |
| TO × Catrachita[d] | 93 | 9 | 0.76 | 0.38 |
| TU × Catrachita[d] | 96 | 6 | 0.00 | 1.00 |
| Raven × SEL 1360[d] | 95 | 9 | 0.66 | 0.42 |
| Blackhawk × SEL 1360[d] | 69 | 4 | 0.00 | 1.00 |
| Mex 222 × SEL 1360[e] | 96 | 6 | 0.00 | 1.00 |
| TO × SEL 1360[d] | 80 | 4 | 0.11 | 0.74 |
| TU × SEL 1360[f] | 102 | 0 | 5.78 | 0.02 |
| Catrachita × SEL 1360[d] | 91 | 11 | 2.85 | 0.09 |

[a] Resistant × resistant crosses in all cases.
[b] 15:1 (resistant, R- : susceptible, rr) expected ratio for two independently assorting genes.
[c] P = estimated probability value.
[d] Phenotypic reaction to race 64 of *C. lindemuthianum.*
[e] Phenotypic reaction to race 7 of *C. lindemuthianum.*
[f] Phenotypic reaction to two independent inoculations with races 64 and 7 of *C. lindemuthianum*, respectively.

## DISCUSSION

Stable resistance to plant pathogens with extensive physiological variability, such as *C. lindemuthianum*, requires continual evaluation of germ plasm and eventual introgression of diverse genetic resistance into commercial cultivars. The appearance of new races of anthracnose in Michigan (race 7 and 73) (9) and Canada (alpha-Brazil) (25) that overcome the *Are* (race 73 and alpha-Brazil) and *A* (race 7) genes has forced breeders to seek alternative sources of resistance. Studies on the inheritance of anthracnose resistance in Catrachita and SEL 1360 derived from AB 136 and G 2333 cultivars, respectively, were conducted because the broad-based resistance observed in these differential cultivars could be of value in breeding for long-term anthracnose resistance in beans (15,19,20,22). A unique monogenic dominant gene segregated in three different $F_2$ populations derived from crosses between resistant (Catrachita) and susceptible (Black Magic, Raven, and Blackhawk) parents. It was concluded that this single dominant gene in Catrachita was responsible for the resistance to races 23 (delta), 64 (alpha), and 73. A coupling-phase random amplified polymorphic DNA (RAPD) marker tightly linked to the resistant allele in Catrachita was also present in AB 136 and absent in all susceptible parents (Black Magic, Raven, and Blackhawk) (28), confirming that the single dominant resistance gene in Catrachita is the same resistance gene present in AB 136. A monogenic factor controlling anthracnose resistance in AB 136 was also described by Gonçalves-Vidigal (7). In that work, segregating populations derived from crosses involving AB 136 as the resistant parent were chal-

lenged with three different races of the pathogen. The symbols *B*, *A'*, and *Q* were assigned to describe the dominant gene governing the resistance to alpha, delta, and kappa, respectively. It is assumed that *B*, *A'*, and *Q* symbols refer to the same gene, since no experimental evidence suggests the contrary. Duplicate independent and dominant genes were observed to segregate in all populations derived from crosses of Catrachita and the cultivars Raven, Blackhawk, Mex 222, TO, and TU, which carry previously characterized resistance genes *A*, *Are*, *Mexique 1*, *Mexique 2*, and *Mexique 3*, respectively. In all instances, chi-square tests confirmed a 15:1 ratio, suggesting that the single dominant gene in Catrachita was situated at a different locus from *A*, *Are*, *Mexique 1*, *Mexique 2*, and *Mexique 3* resistance genes. In addition, independent segregation of two dominant genes was also observed in the cross Catrachita × SEL 1360, indicating that these genes are located at different loci.

Characterization of the genetic resistance in Catrachita determined that one dominant gene conditions resistance to bean anthracnose in this cultivar. The major gene in Catrachita is independent from the characterized genes *A* (*Co-1*), *Are* (*Co-2*), *Mexique 1* (*Co-3*), *Mexique 2* (*Co-4*), and *Mexique 3* (*Co-5*). It is proposed, therefore, that the single dominant resistance gene present in Catrachita be assigned the genetic symbol *Co-6*, *Co* for *Colletotrichum* and *6* because it is the sixth major anthracnose resistance gene characterized and reported in the literature (11).

The resistance factor present in SEL 1360 was shown to be a monogenic dominant gene based on segregation ratios in three different $F_2$ populations. A difference in inheritance patterns between G 2333 and the derived line SEL 1360 was expected, since G 2333 and SEL 1360 displayed different phenotypic reactions after inoculation with anthracnose races 1545 and 2047 (Table 1). Considering that SEL 1360 is a selection derived from two backcrosses with the donor parent G 2333, it can be assumed that only one of the two dominant genes was transmitted to SEL 1360. The test for allelism involving SEL 1360 demonstrated that the segregation ratio in four populations (Raven, Blackhawk, Mex 222, and TO) followed a 15:1 (R-:rr) ratio, indicating that the single dominant gene in SEL 1360 is independent from *A*, *Are*, *Mexique 1*, and *Mexique* 2 genes. However, the dominant gene in SEL 1360 did not segregate independently from the resistance gene (*Mexique 3*) in the differential cultivar TU, indicating that both dominant alleles are located at the same locus. Since none of the anthracnose races tested (Table 1) could effectively differentiate between the two alleles, the data suggest that the resistance gene in SEL 1360 is the same dominant *Mexique* 3 gene present in TU. Additional evidence supporting this hypothesis comes from a coupling-phase RAPD marker linked (5.9 ± 1.7 cM) to the SEL 1360 allele, which was also present in TU and the G 2333 parent (28). Since G 2333 and TU belong to the same Middle American *Phaseolus* gene pool, it is possible that these cultivars share common resistance genes. Race 521 was shown to discriminate between the resistance gene in TU and the two genes present in G 2333 (18). However, results of the present study suggest the following genetic model. The dominant gene in SEL 1360 is the *Mexique 3* gene and is a third independent gene present in G 2333. This third gene was not detected by Pastor-Corrales et al. (18), since it did not condition resistance to race 521, which was used in that study. Support for this model is based on the RAPD marker evidence (28) and in the susceptible reaction of SEL 1360 to race 521 (data not shown).

Gene pyramiding has been suggested as a potential strategy for maintaining long-term durable disease resistance (16). The accumulation of major resistance genes in a cultivar would delay the appearance of new races of the pathogen. The basis for the stabilization of resistance resides in the reduction in fitness of a pathogen when the number of virulence genes necessary to overcome host resistance increases (26). The decreased probability of occurrence of a mutant or recombinant race that could overcome several resistance genes is a major factor contributing to the durability of host resistance (21). The differential cultivar G 2333 constitutes an example of the effectiveness of gene pyramiding in controlling bean anthracnose when more than one major resistance gene is present in the host. A collection of 380 isolates of *C. lindemuthianum* from different parts of the world were avirulent on this cultivar (18). To date, no isolate has been reported with the ability to overcome G 2333 resistance. Diversity in the sources of resistance used would also contribute to the stability of anthracnose resistance. For example, Kelly et al. (9) suggested that pyramiding *A* and *Are* genes would afford protection against all known anthracnose races in North America. It is noteworthy that the *A* gene is of Andean origin, in contrast to the *Are* gene of Middle American origin. The introgression of both Andean and Middle American genes seems to be an effective genetic combination for broad-based resistance. For instance, Pastor-Corrales et al. (18,19) reported that the *A* gene present in the differential cultivar Michigan Dark Red Kidney conferred resistance to a group of highly virulent races (73, 129, 133, 136, 385, 448, 521, 901, 905, 1409, and 1473) of the pathogen. These races and race 1545 (Table 1) are pathogenic on most of the resistant sources of Middle American origin. It was observed that some of the differential cultivars of Andean origin showed susceptibility to several isolates from South America; whereas resistance to Middle American isolates was observed. In contrast, Middle American differential cultivars were usually immune to isolates of Andean origin (19). Therefore, deployment of major genes such as *Co-6* and *Mexique 3* (*Co-5*) in different combinations with other characterized genes such as the *A* (*Co-1*) gene should contribute to more durable anthracnose resistance in common bean.


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