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INTRODUCTION

Several diseases have contributed to limit the bean crop yield. Among the diseases white mold (*Sclerotinia sclerotiorum*) and anthracnose (*Colletotrichum lindemuthianum*) stand out. One way to reduce losses caused by both *S. sclerotiorum* and *C. lindemuthianum* is using resistant cultivars. Recurrent selection allows improving quantitative traits such as white mold resistance, as well as improve several characters simultaneously, if the selected parents have alleles for these traits. LEITE et al. (2016) and DIAS (2015) performed recurrent selection for resistance to white mold intercrossing several parents using the circulant diallel procedure and, some of them had anthracnose resistance alleles (*Co-4²* and *Co-5*). Although there was no selection for those alleles, they may have remained in the population after successive selective cycles. The objective of this study was to verify anthracnose resistance among the selected progenies in the cycle VII, VIII and IX from recurrent selection program for white mold resistance, and to identify the presence of resistance alleles *Co-4²* and *Co-5*.

MATERIAL AND METHODS

Twenty-two progenies of cycle VII, 23 of cycle VIII and 21 of cycle IX, all of them of the $S_{0:4}$ generation and derived of recurrent selection program for resistance to white mold, and the controls Pérola (susceptible) and M20 (resistant due to *Co4²* allele) were evaluated. The experiments were set up using a completely randomized design, with two replicates and plots with 16 plants in greenhouse. The races 65 and 1609 were inoculated in all experiments according to PIO-RIBEIRO & CHAVES (1975) and, the anthracnose reaction was evaluated visually using a descriptive scale with scores from 1 to 9 (SCHOONHOVEN & PASTOR-CORRALES 1987).

In order to verify the presence of the *Co-4²* resistance allele, DNA of the progenies and the controls were extracted through a procedure similar to the used by PARRELLA et al. (2008), and the PCR was carried out using the primer SAS13, which is linked to *Co-4²* allele at the distance of 0,34cM.

RESULTS AND DISCUSSION

The experiments were set up with high experimental precision with accuracy value higher than 90%. There were selected 54% of resistant progenies to anthracnose, with mean lower than the control M20. Some progenies and the controls are present in the Table 1. The cycle VII had a higher number of resistant progenies than cycles VIII and IX, probably because in cycle VII five new white mold sources were intercrossed and, three of them were also anthracnose resistant. In cycles VIII and IX the frequency of the resistant alleles for anthracnose should have become more diluted in the population, as long as the selection was based only on white mold resistance.

Table 1. Reaction of progenies from cycles VII, VIII and IX of recurrent selection to the *C. lindemuthianum* races 65 and 1609.

Cycle VII	Race 65			Race 1609		
Progeny	Group	Mean	% of R*	Group	Mean	% of R*
81/15	a	1.00	91 (M<3)	a	1.15	74 (M<3)
81/13	a	1.00		a	1.07	
81/17	a	1.18		a	1.00	
M20	b	2.10		b	3.85	
Pérola	d	5,22		d	5,76	
Cycle VIII						
8\04	a	1.00	65 (M<3)	a	3.25	0 (M<3) 35 (M<M20)
5\23	a	1.04		a	4.41	
14\17	a	1.32		a	4.43	
M20	b	2.44		a	4.57	
Pérola	d	6,13		b	6,19	
Cycle IX						
34\3	a	1.10	64 (M<3)	a	1.74	3 (M<3) 47 (M<M20)
28\11	a	1.47		a	1.71	
24\14	a	2.40		b	3.30	
M20	a	2.57		b	4.27	
Pérola	c	5,55		c	5,44	

* % of resistant progenies with mean less than 3 (M<3) and mean less than resistant control (M<M20) according to *Scott Knott* grouping ($p<0.05$).

Although large number of the selected progenies had less infection than M20 control, the *Co4²* allele could not be identified by molecular marker SAS13 like in M20. Therefore, the resistant progenies should have the *Co5* allele, known to be present in some original parents. However, it is matched by the race 1605 and responsible for resistance only to 65 race. So, those progenies resistant to both races should also have other alleles that are present in the parents. We can realize that recurrent selection was efficient in contributing to generate variability, and consequently, to make feasible the selection of resistant progenies, both to the white mold and anthracnose.

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