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INHERITANCE OF RESISTANCE TO COMMON BACTERIAL BLIGHT IN COMMON BEAN

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ABSTRACT

The common bean (*Phaseolus vulgaris* L.) is an important grain legume crop in Malawi where it is grown by small holder farmers for food as well as for sale. Among the many diseases that limit crop productivity is the common bacterial blight caused by *Xanthomonas axonopodis* pv *phaseoli (Xap).* Effective breeding for resistance to *Xap* requires understanding of the model of inheritance for resistance. A study to determine the inheritance of resistance to *Xap* in common bean was carried out in Malawi. Two established bean varieties originating from local landraces in Malawi (Chimbamba and Nasaka), plus one line (RC 15) from the breeding programme at Bunda College of Agriculture, were used as recipient (susceptible) parents; while Vax 6 from CIAT was the donor (resistant) parent. The progenies were advanced to F_2 generations in greenhouses. The F_2 populations were evaluated for resistance to *Xap*. The results showed that one recipient parent, Chimbamba, which is supposedly homogeneous, behaved like a segregating population and, therefore, modified the phenotypic ratios of the progenies. A Chi-square test using data generated from populations resulting from the three recipient parents showed that the inheritance of resistance to *Xap* was controlled by two major genes with possible minor genes involvement. The same was true when a Chi-square test was used to analyse the pooled data across populations generated from the three recipient parents (Chimbamba, Nasaka and RC 15), suggesting that inheritance of resistance to *Xap* was controlled by two major genes.

Key Words: *Phaseolus vulgaris, Xanthomonas axonopodis*

RÉSUMÉ

Le haricot commun (*Phaseolus vulgaris* L.) est une importante légumineuse cultivée par les petits fermiers au Malawi aussi bien pour la consummation que pour la vente. Parmi de nombreuses maladies qui limitent sa productivité se trouve la bactérie commun de causé par *Xanthomonas axonopodis* pv *phaseoli (Xap).* Une amelioration effective pour la résistance au *Xap* exige la compréhension du modèle d'acquisition de la résistance. Une étude était conduite pour déterminer l'acquisition de la résistance au *Xap* dans le haricot commun au Malawi. Deux variétés indigènes de haricot au Malawi (Chimbamba and Nasaka), plus une lignée (RC 15) provenant du programme d'amélioration au Collège d'Agriculture de Bunda, étaient utilisées comme parents recepteurs (susceptibles); pendant que Vax 6 fourni par CIAT était parent donneur (résistant). Les descendants étaient portés aux générations F2 en serre. Les populations F2 étaient évalués pour résistance au *Xap*. Les résultats ont monté qu'un parent recepteur, Chimbamba, supposé homogène, s'était comporté comme une population ségrégante et, par conséquent, avait modifié les rapports phénotypiques des descendants. Un test de Chi-Carré utilisant des données des populations résultant des trois parents recepteurs ont montré que l'acquisition de la résistance au *Xap* était controllé par deux gènes majeurs avec implication possible de gènes mineurs. Ceci était de même vrai lorsqu'un test Chi-carré était utilisé pour l'analyse de données à travers les populations générées de trois parents recepteurs (Chimbamba, Nasaka and RC 15), suggérant qu'une acquisition de résistance au *Xap* était controllé par deux gènes majeurs.

Mots Clés: *Phaseolus vulgaris, Xanthomonas axonopodis*

INTRODUCTION

Common bacterial blight caused by *Xanthomonas axonopodis pv phaseoli* (*Xap*) is a disease of economic importance in common bean (*Phaseoulus vulgaris* L.) worldwide (Zaumeyer, 1957). In the tropical and sub-tropical areas, it can be severe because of high temperatures and alternating wet and dry conditions. Weather conditions, susceptibility of the cultivars and disease pressure determine the extent of loss of grain yield and quality, resulting in losses of 20-60% (Lema-Marquez *et al.*, 2007). The pathogen is seed borne and this poses serious implications on seed distribution within and between producing countries. In addition to being a seed borne pathogen, Mkandawire *et al.* (2004) reported great genetic diversity and co-evolution for *Xap* across geographic regions and bean gene pools (Mesoamerican and Andean), which is a challenge in breeding for disease resistance.

Breeding for high levels of resistance remains the most appropriate and cost effective means of managing *Xap*. In order to effectively breed for resistance in the adaptable cultivars, knowledge of the mode of inheritance and type of gene action for resistance are of paramount importance. The number of genes involved in resistance to *Xap* is not clearly known, but suggestions vary from one to several genes, with varying degrees of action and interactions (CIAT, 1981; Beebe and Pastor-Corrales, 1991; Zapata *et al.*, 2009; 2010). Quantitative inheritance was observed by Honna (1956) after making original interspecific crosses between resistant *P. acutifolius* 'tepary 4' and susceptible *P. vulgaris*. It is also critical to have durable sources of resistance to *Xap*. Sources of resistance to *Xap* in common bean have been reported (Zapata *et al.*, 2004; Miklas *et al.*, 2005). Other sources of resistance have been identified in tepary bean (*P. acutifolius*) (Schuster *et al.*, 1983; Drijfhout and Blok, 1987), and runner bean, (*P. coccineus*) (Mohan, 1982).

The Centro Internatcional de Agricultura Tropical (CIAT) has developed several lines which are used as good sources of resistance to *Xap*: Vax 1, Vax 2, Vax 3, Vax 4, Vax 5 and Vax 6 (Singh *et al*., 1999), but the mode of inheritance and type of gene action for resistance to *Xap* remain to be clearly understood. This study sought to determine the inheritance of resistance to *Xap* under field conditions in Malawi.

MATERIALS AND METHODS

Crosses were made between three recipient (susceptible to *Xap*) parents: Chimbamba, Nasaka and RC 15; and one donor (resistant to *Xap*) parent – Vax 6. Chimbamba is a local land race, climbing bean cultivar of Type IV, with indeterminate growth habit, which is adapted in Malawi. It is normally grown with stakes or in association with maize for support. Nasaka is a local land race, bush bean cultivar of Type I with determinate growth habit, which is adapted in Malawi. RC 15 is a bush bean line of Type I which originated from the Bean Breeding Programme at Bunda College of Agriculture in Malawi. Vax 6 is a bush bean line of Type I which originated from CIAT in Colombia. It was developed from G40020 *via* Xan 159, Xan 160, Xan 161, Xan 263 and Xan 309. Xan 263 and Xan 309 derive their resistance to *Xap* from tepary bean and this is the possible source of the resistance genes to *Xap* in Vax 6 (McElroy, 1985). Crosses were generated in the greenhouses at Bunda College of Agriculture and Bvumbwe Research Station.

Field evaluation. The parental and F_2 plants were evaluated at the Bunda Crop and Soil Science Research Farm and Dedza Bean/Cowpea research site. Bunda is located at 14^0 12' S; 33^0 46 E in the Lilongwe plains, with an elevation of 1200 meters above sea level (masl), and the soils are sandy clay loam. The crop at Bunda received moderate rainfall, about 378 mm, with average daily temperatures of 27 ^{o}C (Max) and 18 ^{o}C (Min). Dedza is located in the Kirk Range highlands at 14^0 20' S and 34^0 18' E, with an elevation of 1500 masl, and the soils are clay loam. The crop growing conditions at Dedza were wetter, receiving 528 mm of rainfall, with slightly cooler average daily temperatures of 25 ^{o}C (Max) and 15 ^{o}C (Min).

The segregating progenies in F_2 generations resulting from a common donor and recipient parents were assigned to a block which had 3

sub-plots: F_2, recipient (susceptible) parent and donor (resistant) parent. Each sub-plot had a single row of 6 meters, and the rows were spaced at 75 cm apart. Seeds were planted at a spacing of 10 cm for bush beans (Vax 6, Nasaka and RC 15) and their resulting progenies, while Chimbamba, a climbing bean type, and the resulting progeny populations were planted at a spacing of 15 cm, apart because climbing beans need more space.

Evaluation for *Xap* was done at R6 (flowering) and R8 (pod filling) for both parents and progenies. The reaction of individual plant canopy to *Xap* was evaluated based on the 1-9 scale (CIAT, 1987), where 1= immune and 9 = very susceptible. The scores were grouped into 3 categories: 1-3 for resistant plants, 4-6 for intermediate reaction and 7-9 for susceptible plants.

A Chi-square test, using the Statistical Package for Social Scientists (SPSS) Version 9.0 was used to determine the mode of inheritance for resistance to *Xap*. The frequency distributions of parental plants based on disease reaction were plotted to determine the distribution pattern. The phenotypic classes were tested for goodness of fit to postulated ratios based on the possible number of genes involved.

RESULTS

Parental reaction and F_2 plants segregation for resistance to *Xap*

Bunda and Dedza as separate sites. There was low *Xap* disease infection pressure at Bunda and as a result, one of the three recipient (susceptible) parents, Chimbamba, showed high levels of resistance. The other two parents (Nasaka and RC 15) were susceptible, whereas the donor parent (Vax 6) was resistant (Table 1).

The pattern of segregation in F_2 progenies from Chimbamba x Vax 6 showed a 15:1 ratio. This result indicates a moderate probability (χ^2= 0.384; P= 0.535) for two genes with duplicate dominant epistasis (Table 1). In the second cross, Nasaka x Vax 6, the F_2 progenies segregated in a 13:3 ratio. In the third cross, RC 15 x Vax 6, the F_2 progenies segregated in the ratio of 9:3:3:1 (χ^2 = 1.253; P= 0.740).

The mode of gene action varied depending on the recipient parent: Chimbamba (duplicate dominant epistasis), Nasaka (dominant and recessive epistasis), and RC 15 (complete dominance). Heterogeneity test of progenies from the three different crosses failed to confirm homogeneity of F_2 progenies and, hence, the data from the different crosses could not be pooled together. This meant that progenies from three crosses segregated differently although the parents were considered to be homozygous for the genes controlling resistance to *Xap*.

Unlike Bunda, the disease pressure was more at Dedza, because the climatic conditions were conducive for disease development, but again Chimbamba was not severely attacked by common bacterial disease as were Nasaka and RC 15. Consistently, Vax 6 showed resistance to common bacterial blight (Tables 1 and 2). F_2 progenies from Chimbamba x Vax 6, Nasaka x Vax 6 and RC 15 x Vax 6 were consistent with the expected ratios of 9:3:3:1 (χ^2 = 2.56, P=0.46); 9:3:4, (χ^2 = 1.77, P= 0.41), and 9:3:4 (χ^2 = 1.59, P= 0.45), at Dedza site, respectively.

Heterogeneity test of progenies from the three different crosses confirmed homogeneity of F_2 plants in their reaction to *Xap* and, hence, could be pooled together. The Chi-square value for additivity (χ_d^2) for the 9:3:4 ratio showed that the F_2 plants from all the recipient parents were homogeneous (χ_d^2 = 0.003; P>0.99) and, hence, pooled χ^2 value could be used at 3 degrees of freedom in determining compliance of the observed to expected 9:3:4 ratio. The pooled χ^2 value confirmed the existence of two genes interacting in a recessive epistasis manner.

Bunda and Dedza combined. When the parents were assessed for reaction to *Xap* across the two sites (Bunda and Dedza), Chimbamba behaved like a segregating population with some plants showing good levels of resistance, and others susceptible (Fig. 1), while Nasaka and RC 15 were homogeneous and susceptible (Figs. 2 and 3). Vax 6 the resistant parent was also homogeneous showing high levels of resistance to *Xap* across sites (Figs. 1- 3).

The evaluation of F_2 progenies showed segregation patterns ranging from complete

TABLE 1. Number of bean plants for parental lines and F_2 progenies, showing different levels of resistance to *Xap* at Bunda in Malawi

Parent/crosses	Frequency distribution of plants on a 1-9 scale									Observed R:S[a] ratio	Expected R:S ratio	X^2	P
	1	2	3	4	5	6	7	8	9				
Chimbamba	1	17	6	1	2	0	0	0	0				
Vax 6	18	14	1	0	0	0	0	0	0				
F_2 (Chimbamba/Vax 6)	4	18	9	3	0	0	0	0	0	31:3	15:1	0.38	0.54
Nasaka	0	0	0	0	1	6	6	9	8				
Vax 6	9	22	0	0	0	0	0	0	0				
F_2 (Nasaka/Vax 6)	4	22	1	0	0	1	2	0	4	27:7	13:3	0.075	0.78
RC 15	0	1	0	1	3	7	7	6	16				
Vax 6	3	23	7	2	2	0	0	0	0				
F_2 (RC 15/Vax 6)	0	11	12	5	6	2	5	3	0	23:11:7:3	9:3:3:1	0.25	0.74

[a] R:S = resistant : susceptible

TABLE 2. Number of bean plants for parental lines and F_2 progenies, showing different levels of resistance to *Xap* at Dedza in Malawi

Parent/crosses	Frequency distribution of plants on a 1-9 scale									Observed R:S[a] ratio	Expected R:S ratio	X^2	P
	1	2	3	4	5	6	7	8	9				
Chimbamba	1	0	2	3	1	3	6	2	0				
Vax 6	6	2	2	1	0	0	1	0	0				
F_2 (Chimbamba/Vax 6)	5	3	2	2	2	2	3	1	2	8:4:4:6	9:3:3:1	2.56	0.46
Nasaka	2	0	1	2	3	3	5	7	18				
Vax 6	29	1	1	0	0	0	0	0	0				
F_2 (Nasaka/Vax 6)	8	1	4	4	2	0	3	2	5	13:6:10	9:3:4	1.77	0.42
RC 15	0	0	0	0	3	2	7	12	16				
Vax 6	14	3	1	1	1	1	0	0	0				
F_2 (RC 15/Vax 6)	6	3	1	2	1	1	1	5	2	10:4:8	9:3:4	1.60	0.45
F_2 s across crosses	18	8	7	9	5	3	7	7	8	36:16:24	9:3:4	3.78	0.15
Chi-square value for additivity in F_2												0.003	0.999
Pooled Chi-square value (total in F_2)												3.621	0.305

[a] R:S = resistant : susceptible

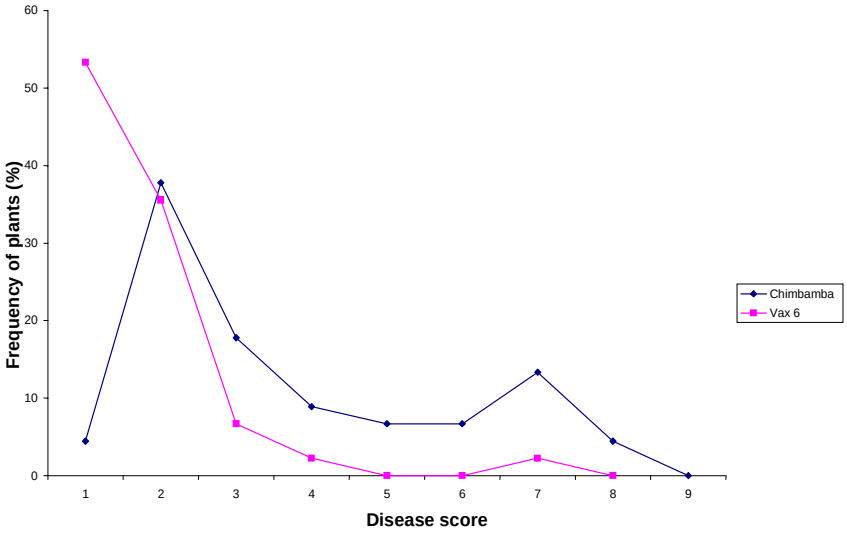

Figure 1. Percentage distribution of Chimbamba and Vax 6 plants with different scores (1-9) for resistance to *Xap* in Malawi.

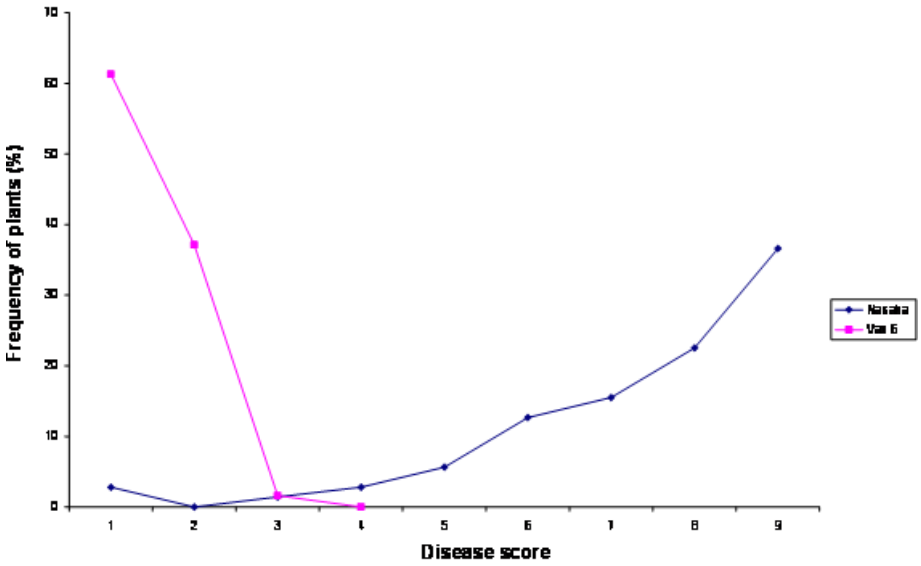

Figure 2. Percentage distribution of Nasaka and Vax 6 plants with different scores (1-9) for resistance to *Xap* in Malawi.

resistance to susceptibility. The Chi-square test of additivity indicated that the data sets were heterogeneous across populations.

The phenotypic segregation of the F_2 progenies for reaction to *Xap*, showed that plants from Chimbamba x Vax 6 population largely segregated in the ratio of 12:3:1 (X^2=2.024, P=0.364). The F_2 progenies from a cross between Nasaka x Vax 6 suggested the presence of two genes with recessive epistasis (X^2=2.553, P=0.279) (Table 3). In RC 15 x Vax 6 population, the F_2 plants' segregation supported the hypothesis that resistance to *Xap* was governed by two genes with recessive epistasis (X^2 =2.175, P=0.337).

DISCUSSION

Chimbamba, one of the susceptible parents with a climbing growth habit (type IV), behaved like a

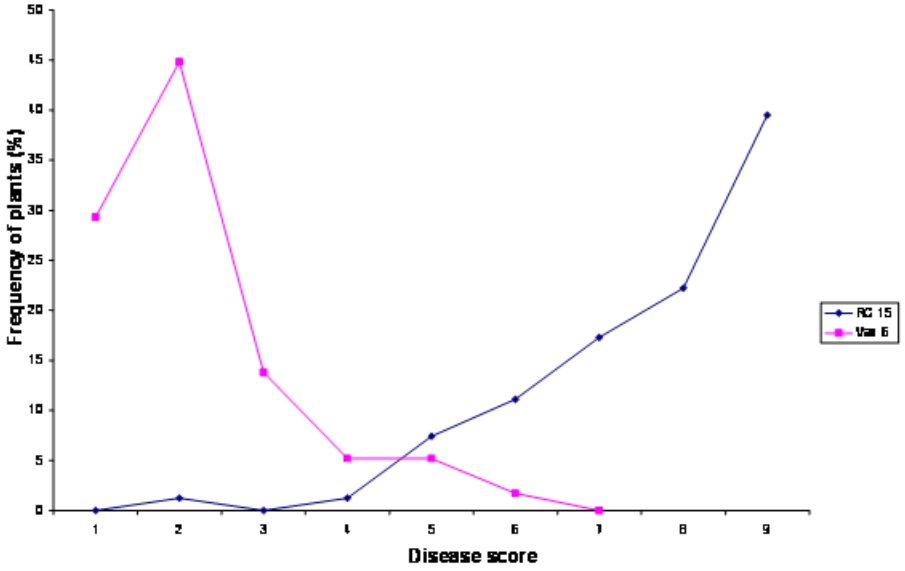

Figure 3. Percentage distribution of RC 15 and Vax 6 plants with different scores (1-9) for resistance to *Xap* in Malawi.

segregating population in its reaction to *Xap* while the other two susceptible parents, Nasaka and RC 15 with a bush growth habit (type I), were homogeneous in their reaction (Table 3). The reason for the low infection rate in Chimbamba may be associated with its growth habit. This is possibly because of its vigorous climbing growth habit, which was also reported by Coyne and Schuster (1974) and Beebe and Pastor-Corrales (1991) suggesting that plant architecture including growth habit may influence disease severity. The observation may also imply that choice of parents is an important factor in genetic studies.

The results from Bunda showed that the progenies originating from all the three susceptible parents: Chimbamba, Nasaka and RC 15 indicated that two genes were involved in conferring resistance to *Xap*. However, the mode of gene action varied depending on the recipient parent: Chimbamba (duplicate dominant epistasis), Nasaka (dominant and recessive epistasis), and RC 15 (complete dominance). The results from Dedza also showed a two gene model of inheritance, where progenies of Nasaka and RC 15 supported the hypothesis that resistance to *Xap* is controlled by two genes with recessive epistasis. Chimbamba, however, suggested that the resistance to *Xap* was controlled by 2 genes with dominant epistasis. The cross site analyses confirmed the two gene model, where Nasaka and RC 15 suggested two genes with recessive epistasis, but Chimbamba showed two genes with dominant epistasis.

This study suggests that genetic resistance to *Xap* in common bean genotypes is controlled by more than one gene with varying degrees of gene action. These findings are similar to those reported by several authors that have reported *Xap* to be controlled by one or more genes (Adams *et al*., 1988; Silva *et al.*, 1989; Beebe and Pastor-Corrales, 1991; Zapata *et al*., 2009). Other authors have reported quantitative trait inheritance for resistance to *Xap* (Kelly *et al*., 2003; O'Boyle and Kelly, 2007). However, Zapata *et al.* (2010) were the first ever to report a single gene for resistance to *Xap* in common bean. They found that the resistance gene derived from line PR0313-58 in the cross PR0313-58 (resistant) x Rosa Nativa (susceptible) supported the hypothesis that resistance to *Xap* strain 3353 is conferred by a single dominant gene.

It is worthy noting from the findings of this study, the differences in gene expression for resistance to *Xap* from the same donor parent when in the background of recipient parents with different growth habits: Nasaka and RC15 (bush and determinate) *versus* Chimbamba (climbing and

TABLE 3. Number of bean plants for parental lines and F_2 progenies, showing different levels of resistance to *Xap* pooled over two locations (Bunda and Dedza) in Malawi

Parent/crosses	Frequency distribution of plants on a 1-9 scale									Observed R:S[a] ratio	Expected R:S ratio	X^2	P
	1	2	3	4	5	6	7	8	9				
Chimbamba	2	17	8	4	3	3	6	2	0				
Vax 6	24	16	3	1	0	0	1	0	0				
F_2 (Chimbamba/Vax 6)	9	21	11	5	2	2	3	1	2	41:9:6	12:3:1	2.02	0.36
Nasaka	2	0	1	2	4	9	11	16	26				
Vax 6	38	23	1	0	0	0	0	0	0				
F_2 (Nasaka/Vax 6)	12	23	5	4	2	1	5	2	9	40:7:6	9:3:4	2.55	0.27
RC 15	0	1	0	1	6	9	14	18	32				
Vax 6	17	26	8	3	3	1	0	0	0				
F_2 (RC 15/Vax 6)	6	14	13	7	7	3	6	8	2	13:17:16	9:3:4	2.17	0.33

[a] R:S = resistant : susceptible

indeterminate). The gene action for resistance to *Xap* in the background of bush bean cultivars as recipient parents was recessive epistasis versus dominant epistasis in the background of a climbing bean cultivar as a recipient parent. While that for climbing bean cultivar was two genes with dominant epistasis. This could be due to the differences in plant growth habit as Chimbamba is a type IV climbing bean with vigorous vegetative growth, which was clinging on to stakes, spreading its canopy in the aerial space. The other two cultivars (Nasaka and RC15) are bush with their canopy crowded close to the ground level, and experiencing a different microclimate.

Singh *et al*. (1999) suggested that the growth habit of the bean plant and delayed maturity, influenced expression on plant resistance to *Xap*. This might also explain why Chimbamba as a susceptible parent behaved differently from the other two, Nasaka and RC15. Singh *et al.* (1999) also cited instability of *Xap* resistance, differential *Xap* reaction of leaves *versus* pods and the association of resistance with stages of plant development as among the factors posing challenges in breeding for *Xap* resistance. The existence of genetic diversity and pathogen variation of *Xap* also poses challenges in breeding for resistance to *Xap*. Zapata (2006) and Zapata *et al*. (2010) suggested that the existence of pathogenic races in *Xap* raises the question of the number of races that might exist, and the stability of varietal resistance.

Mkandawire *et al.* (2004) reported that there was a possibility that *Xap* in Middle America and African regions had co-evolved with the bean germplasm grown in the respective regions. Their findings also showed that although the Middle American beans had resistance to *Xap* induced by East African strains, the results also supported earlier findings by Sigh and Muñoz (1999) that high levels of resistance to *Xap* were not found in common beans. As such, it is important for plant breeders to identify the prevalent type or types of *Xap* in the region in order to better target the breeding programme when developing resistant varieties against the predominant virulent *Xap* pathogens as suggested by Mkandawire *et al*. (2004). Miklas *et al.* (2005) and Zapata *et al.* (2004) have reported release of germplasm with resistance to *Xap* in addition to the Vax lines developed at CIAT.

Fortunately, the Vax lines, which combine various sources of resistance to *Xap* have shown high levels of resistance to most of the strains in Middle America, Andean and Africa regions, offering plant breeders some promising sources of resistance for use in the breeding programmes.

CONCLUSION

This study suggests that genetic inheritance for resistance to *Xap* in common bean is controlled by two genes with varying degrees of gene actions: recessive epistasis for Nasaka and RC 15, and dominant epistasis for Chimbamba. It has also revealed the importance of parental selection in breeding for resistance to *Xap,* due to the differences in reaction to diseases associated with the differences in plant growth habit. This is particularly important when selecting parental lines for genetic studies on inheritance for resistance to diseases. The environmental and plant architectural effects on the reaction to *Xap,* makes breeding for resistance more challenging. Marker assisted breeding (MAB) may provide opportunities for overcoming such challenges, and effort to use markers in bean breeding is already underway.

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