Journal of Animal & Plant Sciences, 2009. Vol. 2, Issue 2: 89 - 95.
Publication date: 03 March 2009, http://www.biosciences.elewa.org/JAPS; **ISSN 2071 - 7024**

Inheritance of resistance to *Fusarium* wilt in pigeonpea {*Cajanus cajan* (L.) Millsp.}

D.A. Odeny[1a♣], S.M. Githiri[1b] and P.M. Kimani[1]

[1] University of Nairobi, College of Agriculture and Veterinary Sciences, Upper Kabete Campus, P.O. Box 29053, Nairobi, Kenya; [a] Present address: Max Planck Institute for Plant Breeding Research, Carl-von-Linne Weg 10, 50829 Cologne, Germany; [b]Present Address: ACCI, University of KwaZulu Natal , P/Bag X01, Scottsville, 3209, Kwa Zulu Natal, South Africa.

♣ *Corresponding author email: da_odeny@yahoo.com*

Key words
Pigeonpea, *Cajanus cajan*, *Fusarium udum*, wilt

1 SUMMARY

***Fusarium* wilt caused by *Fusarium udum* Butler is a common and destructive disease of pigeonpea. The development and use of resistant cultivars remains the most effective, economical and environmentally sound strategy for disease control. The objective of this study was to determine the inheritance of resistance to *Fusarium* wilt in pigeonpea, which remains unknown, and to assess whether its genetic control would differ between African and Indian germplasm. Two resistant lines; one from African germplasm (ICEAP00040) and another of Indian origin (ICP8863) were used to make three different crosses (NPP670 x ICEAP00040, ICPL87091 x ICP8863, and KAT60/8 x ICP8863). Tests of F_1, F_2 and backcross generations under controlled conditions indicated involvement of one recessive gene in ICEAP0040 and 2 recessive genes in ICP8863. Our results not only suggest a greater mechanistic complexity of the genes but also provide an insight into the possible differences in genetic basis of resistance to *Fusarium* wilt between cultivars in different regions, which will need to be considered in future breeding programs.**

2 INTRODUCTION

Kenya is the world's third largest pigeonpea producer after India and Myanmar, with over 200,000 ha cultivated annually. Pigeonpea in Kenya is second only to field beans (*Phaseolus vulgaris*) as pulse and as a food legume in acreage and production (FAOSTAT, 2007). *Fusarium* wilt (*Fusarium udum* Butler) is a soil borne fungus that affects the plant at all stages of development resulting in up to 100% yield loss (Reddy *et al*., 1990). Previous studies (Songa *et al*., 1991; Khonga & Hillocks, 1996) highlighted *Fusarium* wilt as one of the most important and widespread diseases in Kenya with wilt incidence estimated at 60% (Kannaiyan *et al*., 1984) in Kenya alone and total annual loss of over US$ 5 million in Eastern Africa. Although no recent surveys have been done, wilt incidence and crop damage is likely to have worsened in the region.

Pigeonpea is mainly grown by small-scale poor farmers in dry areas of the Eastern and Coast provinces of Kenya. Although fungicides can reduce wilt damage to some extent, these chemicals are unaffordable to the peasant farmers and their use would be environmentally damaging in such dry areas. The soil borne nature of the fungus also makes the use of fungicides highly impracticable. It has been

Journal of Animal & Plant Sciences, 2009. Vol. 2, Issue 2: 89 - 95.
Publication date: 03 March 2009, http://www.biosciences.elewa.org/JAPS; **ISSN 2071 - 7024**

suggested that wilt incidence could be reduced by various crop management practices, e.g. pigeonpea-cereal rotation, pigeonpea-tobacco rotation, fallow, green manuring, zinc application, biological control with *Bacillus* (Harish *et al.*, 1998) and early planting. However, host resistance would be the most effective and cheapest management practice.

A lot of research has been conducted on *Fusarium* wilt since the 1930s, especially in India, yet the genetics of resistance to this disease remains to be understood (Saxena, 2008). Some of the reports available (Shaw, 1936; Joshi, 1957; Jain & Reddy, 1995; Pandey *et al.*, 1996; Singh *et al.*, 1998) are conflicting and inconclusive regarding the genetics of this destructive disease. Pal (1934) reported that resistance to wilt in pigeonpea was controlled by multiple factors while Shaw (1936) observed two complementary genes. Later studies by Pathak (1970) confirmed the presence of two complementary genes while Pawar and Mayee (1986) reported the control of this trait by a single dominant gene. Clearly, a better understanding of the genetics of this disease is urgently needed to enable efficient development of resistant cultivars that are suited to various pigeonpea growing regions.

In Eastern Africa in particular, research on *Fusarium* wilt started in 1980s and focused on the identification and testing of newly bred and imported sources of resistance (Kimani *et al.*, 1994; Songa & King, 1994). Two sources of resistance to *Fusarium* wilt have so far been identified in the region while confirmation of resistance in imported cultivars has also been done. Recent molecular characterisation of East African isolates of *Fusarium udum* (Kiprop *et al.*, 2002) suggested the existence of different virulence groups. To develop high yielding *Fusarium* wilt resistant varieties of pigeonpea, it is essential not only to identify sources of resistance, but also to understand the genetics of inheritance. This study was carried out to determine the mode of inheritance of resistance to *Fusarium* wilt in pigeonpea varieties commonly grown in the Eastern African region.

3 MATERIALS AND METHODS

3.1 Experimental site and plant materials: The studies were conducted under greenhouse conditions at Kabete Field Station of the University of Nairobi in 2000. Three wilt resistant and three susceptible pigeonpea lines (Table 1) that had been maintained as pure lines through self pollination were obtained from the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Nairobi, Kenya. Seeds from the 5 cultivars were sown in 10 litre pots filled with sterilized soil collected from a field where pigeonpea had not been grown previously. The potted plants were watered regularly to avoid moisture stress.

The late maturing genotypes were planted first followed by medium and eventually early maturing genotypes, with an interval of one month for every maturity group to ensure synchronization of flowering. At flowering, the lines were hand-pollinated to make 3 crosses (NPP670 x ICEAP00040, ICPL87091 x ICP8863, KAT60/8 x ICP8863), each cross involving a susceptible and a resistant line. Tightly closed buds of the female parent were emasculated by removing anthers from the staminal column with fine forceps one day before they were due to open. About 2 – 10 buds were emasculated per branch and all smaller buds removed to prevent competition within the inflorescence. Pollination was done immediately after emasculation using unopened buds of the male parent for which the anthers would dehisce on the same day. Both emasculation and pollination were done in the morning before 10.00 am to avoid heat, which would otherwise rapture the stigma of the emasculated flower. At maturity, the pods were harvested and F_1 seeds divided into three lots. The first lot was planted and allowed to self into F_2. The second lot was planted and backcrossed to both the resistant (BC_1) and susceptible parents (BC_2). The remaining seeds were kept in store.

Journal of Animal & Plant Sciences, 2009. Vol. 2, Issue 2: 89 - 95.
Publication date: 03 March 2009, http://www.biosciences.elewa.org/JAPS; ISSN 2071 - 7024

Table 1: Maturity, origin and reaction to *Fusarium* wilt of pigeonpea cultivars used for crosses.

Accession	Maturity[1] (duration)	Origin	Response to *Fusarium* wilt
ICP 8863	Medium	India	Resistant
ICEAP 00040	Long	Kenya	Resistant
KAT 60/8	Short	Kenya	Susceptible
NPP 670	Short	Kenya	Susceptible
ICPL 87091	Short	India	Susceptible

[1]*Short, medium and long duration pigeonpea take less than 6 months, 6-8 months and 9-11 months respectively to maturity.*

3.2 Evaluation for resistance: The parents, F_1, F_2, BC_1 and BC_2 generations of all crosses were evaluated in pots under greenhouse conditions. On average, 40 seeds for each of the parents and F_1, 200 seeds for the F_2 and 40 seeds for each backcross generation were used. The seeds for use in the study were pre-germinated in sterile river-bed sand in the glasshouse 10 days before the anticipated date of inoculation. Seeds were sterilised for 3 min in 5% (v/v) Clorax solution and then rinsed twice in distilled water before sowing. The root-dip transplantation technique (Kiprop *et al*., 2002) was used. An isolate of *Fusarium udum* was obtained from Kiboko, a major pigeonpea growing area located in Eastern province. The isolation and culture of a single conidium was done as previously described (Kiprop *et al*., 2002). A mixture of soil and sand (1:3) was steam-sterilized for 24 hours, allowed to cool and then placed in 6-inch diameter pots in the glasshouse. The potted mixture was thoroughly watered a day before transplanting. Moisture level was thereafter maintained at 15-20%.

During transplanting, the seedlings were gently removed from the sand, the roots cleaned, then trimmed with a sterile surgical blade and dipped into the *F.* wilt inoculum solution for 10 minutes before finally transplanting into the pots. Controls of both susceptible and resistant lines were used for every batch. The controls included inoculated and non-inoculated lots. Pots were kept in the greenhouse for two months and wilting of the plants observed. The pathogen was re-isolated from the wilted plants and its pathogenicity re-confirmed.

3.3 Plot design and data collection: All the test lines were grown in a randomised complete block design with four replicates. The disease on-set and progress was monitored and the wilted plants recorded every week for two months. A 1-9 disease scale was used, where 1 - no visible symptoms and 9 - very severely diseased or dead. Chi-square analyses were done to test goodness-of-fit between expected and observed segregation ratios of resistant to susceptible plants (Snedcor and Cochran, 1989).

4 RESULTS

The root dip technique proved to be a relatively quick and reliable procedure for characterising response of various genotypes to *Fusarium udum*. The pathogen was re-isolated and confirmed by infecting previously healthy plants. All the parental lines were relatively stable in their response to infection.

Progenies of a cross involving cv. ICEAP00040, which is the most *Fusarium* wilt resistant genotype of African origin (Gwata *et al*., 2006) and NPP670 were all susceptible suggesting recessive nature of the genes involved in resistance. Further selfing to F_2 led to a segregation giving a good fit for 1 (resistant):3 (susceptible). Progenies of a back cross (BC_1) to the resistant parent segregated at 1:1 while those of a back cross to the susceptible (BC_2) parent were all susceptible (Table 2).

All the F_1 progenies of 2 crosses that involved a common resistant parent from India were susceptible, which indicates a recessive type of resistance. The F_2 progenies segregated into 9:7 (susceptible: resistant) while most of the backcross to the susceptible parent were susceptible (Table 3). On the other hand, the backcross to the resistant parent resulted in 3:1 (susceptible: resistant) segregants. The homogeneity chi-square value (Table 3) was well within the acceptable limit for both crosses resulting in a good fit for the expected 9:7 segregation in the pooled data.

Journal of Animal & Plant Sciences, 2009. Vol. 2, Issue 2: 89 - 95.
Publication date: 03 March 2009, http://www.biosciences.elewa.org/JAPS; **ISSN 2071 - 7024**

Table 2: Segregation ratios, expected ratios and probability *(P)* within a cross made between pigeonpea cultivars NPP 670 x ICEAP 00040.

Pedigree	Observed		Expected		Expected ratio	c^2	*P*
	R[1]	S[2]	R[1]	S[2]			
NPP 670 (P1)	-	65	-	65			
ICEAP 00040 (P2)	24	-	24	-			
NPP 670 X ICEAP 00040 (F_1)	-	71	-	71			
NPP 670 X ICEAP 00040 (F_2)	30	147	44.25	132.75	1:3	3.46	0.06
F_1 X ICEAP 00040 (BC_1)	6	11	8.5	8.5	1:1	0.75	0.39
F_1 X NPP 670 (BC_2)	-	35	-	35		-	

[1]*Resistant* [2] *Susceptible*
df=1, c^2 = 3.84 at the 0.05 probability level

Table 3: Segregation ratios, expected ratios and probability *(P)* within pigeonpea crosses made to resistant lines of Indian origin.

Pedigree	Observed		Expected		Expected ratio	c^2	*P*
	R	S	R	S			
ICPL 87091 (P1)	-	21	-	21			
ICP 8863 (P2)	25	-	25	-			
ICPL 87091 X ICP 8863 (F_1)	-	44	-	44			
ICPL 87091 X ICP 8863 (F_2)	96	143	105	134	7:9	1.37	0.24
F_1 X ICP 8863 (BC_1)	21	34	13.75	41.25	1:3	5.10	0.02**
F_1 X ICPL 87091 (BC_2)	2[1]	33	-	35			
KAT 60/8 (P1)	-	20	-	20			
ICP 8863 (P2)	51	-	51	-			
KAT 60/8 X ICP 8863 (F_1)	-	17	-	17			
KAT 60/8 X ICP 8863 (F_2)	82	135	94.9	122.06	7:9	3.12	0.08
F_1 X ICP 8863 (BC_1)	7	37	11	33	1:3	1.94	0.16
F_1 X KAT 60/8 (BC_2)	4[1]	13	-	33			
Pooled							
F1	-	61	-	61			
F2	178	278	200	256	7:9	4.31	0.04**
BC1	28	71	24.75	74.25	1:3	0.57	0.45
BC2	6*	46	-	52			

[1]*Resistant individuals even though expected to be all susceptible*
df=1, c^2 = 3.841 at the 0.05 probability level

5 DISCUSSION

Our results provide evidence that resistance to *Fusarium* wilt (Kiboko isolate) in pigeonpea is controlled by recessive genes; a single recessive gene in cv. ICEAP00040, which is of East African origin and duplicate recessive genes in the Indian resistant source, ICP8863. The genetic basis of resistance in the cross involving resistant Indian genotypes could be elucidated by assuming a set of 2 independent loci, i.e AABB – Susceptible parent, and aabb – Resistant parent. The 9:7 ratio indicates dihybrid segregation with complementary interaction between the 2 dominant genes. We also propose a model involving qualitative gene action with susceptibility being controlled by a dominant gene for cv. ICEAP00040 .

Although most plant resistance genes have been reported to be controlled by dominant genes, the presence of recessive genes has also been recognized in many plant-pathogen relationships (Upadhyaya *et al*., 1983; Barbetti *et al*., 2005; Sharma *et al*., 2005). Indeed, a recent study in pigeonpea identified two Random Amplified Polymorphic DNA (RAPD; Williams *et al*., 1990) markers linked to a recessive allele of a *Fusarium* wilt resistance gene

(Kotresh *et al.*, 2006). The control of resistance by recessive genes suggests a greater mechanistic complexity (Deslandes *et al.*, 2002) but can be largely attributed to mutations. The *Mlo* recessive mutation (Büschges *et al.*, 1997), which confers broad spectrum resistance to several isolates of the fungus *Erysiphe graminis* f. sp. *hordei* in barley (*Hordeum vulgare* L.) is a good example.

The complexity in the genetics of resistance to *Fusarium* wilt is also suggested by inconsistent results from earlier studies. There have been reports of single dominant (Joshi, 1957; Pawar & Mayee, 1986; Pandey *et al.*, 1996; Singh *et al.*, 1998) and duplicate dominant genes (Okiror, 2002), single recessive genes (Jain & Reddy, 1995), duplicate complimentary genes (Shaw, 1936; Pathak, 1970), or polygenes (Pal, 1934) controlling this trait. Differences in experimental methodology as well as using isolates that differ in virulence could also contribute to such contradictory results. In chickpea (*Cicer arietinum* L.), Tekeoglu *et al.* (2000) reported that lines resistant to one isolate of *Fusarium* wilt (*Fusarium oxysporium* Schlechtend.: Fr. f.sp. *ciceris*) could be susceptible to another isolate. Other sources of error could be the variability in test conditions or the scoring and classification of resistance. Use of uniform procedures, controlled environments and larger sample sizes are therefore critical for increased consistency in results between different experiments. However, in the current investigation, the method of inoculation, the isolate, disease scoring and environmental conditions were similar for all individuals tested.

Our results further suggest that germplasm from Asia and Africa may possess different genetic mechanisms for resistance to *Fusarium* wilt. This is to be expected since major character differences have been shown to occur between Asian and African pigeonpea types (Saxena & Sharma, 1990). An association between the patterns of inheritance and evolutionary relationships has been shown in previous investigations (Salgado *et al.*, 1995). Such an association would suggest that the two resistant lines used in this study have evolved separate and different forms of resistance to *Fusarium* wilt.

In field beans for example, resistance to *Fusarium* wilt (*Fusarium oxysporium* Schlechtend. f.sp. *phaseoli*) was reported to be controlled by major genes among germplasm of races Durango while polygenes controlled resistance in the Mesoamerican types (Salgado *et al.*, 1995; Cross *et al.*, 2000). In beans, just like pigeonpea, there are clear phenotypic differences between genotypes of the respective gene pools (Singh *et al.*, 2001). Whereas studies of field beans have confirmed the differences in genetic control of various traits, similar studies are needed for pigeonpea to conclude this argument.

This study would have benefited from larger populations, particularly in the backcrosses. The backcross and F_1 population sizes in some cases were lower than the minimum population size required (Hanson, 1959) even though the number of parental plants and F_2 populations tested was adequate. Final confirmation of the results reported here would require screening of F_3 plant families and carrying out allelism tests to determine the genetic nature with certainty. Further studies that will include more diverse pigeonpea lines with resistance to specific virulent isolates (Kiprop *et al.*, 2000) should be undertaken in order to confirm or reveal additional resistance genes.

6 ACKNOWLEDGEMENTS: This study was funded by the European Union through an INCO-DC project, contract number ERBIC18CT960130.

7 REFERENCES

Barbetti M., Ping S. and Nichols P: 2005. Genetic basis for and inheritance of resistance to race 1 and 2 of *Kabatiella caulivora* in *Trifolium subterraneum* ssp. *subterraneum* and ssp. *yanninicum*. Euphytica 144: 237 - 246.

Büschges R., Hollricher, K., Panstruga, R., Simons, G., Wolter, M., Frijters, A., van Daelen, R., van der Lee, T., Diergaarde, P., Groenendijk, J., Töpsch, S., Vos, P., Salamini, F. and Schulze-Lefert, P: 1997. The Barley *Mlo* gene: A novel control element of plant-pathogen resistance. *Cell* 88: 695 - 705.

Cross H., Brick M.A., Schwartz H.F., Panella L.W. and Byrne P.F: 2000. Inheritance of resistance to *Fusarium* wilt in two common bean races. *Crop Sci*ence 40: 954 - 958.

Deslandes L., Olivier J., Theuliéres F., Hirsch J., Feng D.X., Bittner-Eddy P., Beynon J. and Marco Y: 2002. Resistance to *Ralstonia solanacearum* in *Arabidopsis thaliana* is conferred by the recessive *RRS1-R* gene, a member of a novel family of resistance

gene. *Proceedings of the National Academy of Sciences USA* 99: 2404 - 2409.

FAOSTAT data: 2007. http://faostat.fao.org/faostat/collections?version=ext&hasbulk=0&subset=agriculture. Last accessed December 2008.

Gwata E.T., Silim S.N. and Mgonja, M: 2006. Impact of a new source of resistance to *Fusarium* wilt in pigeonpea. *Journal of Phytopathology* 154: 62 - 64.

Hanson, W.D: 1959. Minimum family sizes for the planning of genetic experiments. *Agronomy Journal* 51: 711 - 716.

Harish S., Manjula K. and Podile A.R: 1998. *Fusarium udum* is resistant to the mycolytic activity of a biocontrol strain of *Bacillus subtilis* AF1. *FEMS Microbiology Ecology* 25: 385 - 390.

Jain K.C. and Reddy M.V: 1995. Inheritance of resistance to *Fusarium* wilt in pigeonpea (*Cajanus cajan* L.). *Indian Journal of Genetics and Plant Breeding* 55: 434 – 437.

Joshi A.R: 1957. Genetics of resistance to disease and pests. *Indian Journal of Genetics and Plant Breeding* 17: 305 – 317.

Kannaiyan J., Nenen Y.L., Reddy M.V., Ryan J.G. and Raju T.N: 1984. Prevalence of pigeonpea diseases associated with crop losses in Asia, Africa and Americas. *Tropical Pest Management* 30: 62 - 71.

Khonga E.B. and Hillocks R.J: 1996. Soil borne diseases in maize-based cropping systems in southern Malawi: incidence and farmer's perceptions. *African Plant Protection* 2: 131 - 138.

Kimani P.M., Nyende A.B. and Silim, S: 1994. Development of early maturing *Fusarium* wilt resistant pigeonpea cultivars. *African Crop Science Journal* 2: 35 - 43.

Kiprop E.K., Baudoi, J.P., Mwangombe, A.W., Kimani, P.M., Mergea, G. and Maquet, A: 2002. Characterisation of Kenyan isolates of *Fusarium udum* from pigeonpea (*Cajanus cajan* (L.) Millsp.) by cultural characteristics, aggressiveness and AFLP analysis. *Journal of Phytopathology* 150: 517 - 525.

Kotresh H., Fakrudin B., Punnuri S.M., Rajkumar B.K., Thudi M., Paramesh H., Lohithaswa H. and Kuruvinashetti, M.S: 2006. Identification of two RAPD markers genetically linked to a recessive allele of a *Fusarium* wilt resistance gene in pigeonpea (*Cajanus cajan* L. Millsp.). *Euphytica* 149: 113 - 120.

Okiror M.A: 2002. Genetics of resistance to *Fusarium udum* in pigeonpea (*Cajanus cajan* (L.) Millsp.). *Indian Journal of Genetics and Plant Breeding* 62: 218-220.

Pal B.P: 1934. Recent Progress in Plant Breeding at Pusa. *Agr. Livest. India* 4: 505 - 515.

Pandey R.N., Pawar S.E. and Bhatia C.R: 1996. Inheritance of wilt resistance in pigeonpea. *Indian Journal of Genetics and Plant Breeding* 56: 305 – 308.

Pathak G.N: 1970. Red Gram. In: Pulse crops of Indian Council of Agricultural Research. New Delhi, India. Pp 14 – 53.

Pawar N.B. and Mayee C.D: 1986. Reaction of pigeonpea genotypes and their crosses to *Fusarium* wilt. *Indian Phytopathology* 39: 70 - 74.

Reddy M.V., Nene Y.L., Kannaiyan J., Ruju T.N., Saka V.W., Daudi A.T., Songa W.A. and Omanga, P: 1990. Pigeonpea lines resistant to wilt in Kenya and Malawi. *International Pigeonpea Newsletter* 12: 25 - 26.

Salgado M.O., Schwartz H.F. and Brick, M.A: 1995. Inheritance of resistance to a Colorado race of *Fusarium oxysporium* f.sp. *phaseoli* in common beans. *Plant Diseases* 79: 279 - 281.

Saxena K.B: 2008. Genetic Improvement of pigeonpea - A review. *Tropical Plant Biology* 1: 159 - 178.

Saxena K.B., Sharma D: 1990. Pigeonpea: Genetics. In: *Nene, Y.L., Hall, S. D., Sheila, V.K. (Eds), The Pigeonpea.* ICRISAT, Patancheru, India. Pp 137 – 157.

Sharma K.D., Chen W. and Muehlbauer F.J: 2005. Genetics of chickpea resistance to five races of *Fusarium* wilt and a concise set of race differentials for *Fusarium oxysporium* f.sp. *ciceris*. *Plant Disease* 89: 385 - 390.

Shaw F.L.F: 1936. Studies in Indian pulses: the inheritance of morphological characters and of wilt resistance in Arhar (*Cajanus indicus* Spreng). *Indian Journal of Agricultural Sciences* 6: 139 – 187.

Singh I.P., Vishwa D., Chaudhary R.C. and Pandey D.K: 1998. Genetics of *Fusarium* wilt resistance in pigeonpea. In: *National Symposium on Management of Biotic and Abiotic Stresses in Pulse Crops*, 26–28th June 1998, IIPR Kanpur, India. Pp 15.

Singh S.P: 2001. Broadening the genetic base of common bean cultivars: A review. *Crop Science* 41: 1659 - 1675.

Snedecor G.W. and Cochran, W.G: 1989. Statistical Methods. Iowa State University Press.

Songa W.A., Omanga P. and Reddy, M.V: 1991. Survey of pigeonpea wilt and other diseases in Machakos and Kitui districts of Kenya. *International Pigeonpea Newsletter* 14: 25-26.

Songa W.A. and King S.B: 1994. Pigeonpea pathology research in Kenya. In: *Silim, S.N., Tuwafe, S., Laxman, S. (Eds.), Pigeonpea Improvement in Eastern and Southern Africa.* ICRISAT, Patancheru, India. Pp 45-50.

Tekeoglu M., Tullu A., Kaiser W.J. and Muelbauer F.J: 2000. Inheritance and linkage of two genes that confer resistance to *Fusarium* wilt in chickpea. *Crop Science* 40: 1247 - 1251.

Upadhyaya H.D., Smithson J.B., Haware M.P. and Kumar J: 1983. Resistance to wilt in chickpea. II. Further evidence for two genes for resistance to race I. *Euphytica* 32: 749 - 755.

Williams J.G.K., Kubelik A.R., Livak K.J., Rafalski J.A. and Tingey, S.V: 1990. DNA polymorphisms amplified by arbitrary primers are useful as genetic markers. *Nucleic Acids Research* 18: 6531 - 6535.

Young N.D: 1996. QTL mapping and quantitative disease resistance in plants. Annual Rev. *Phytopathology* 34: 479-501.