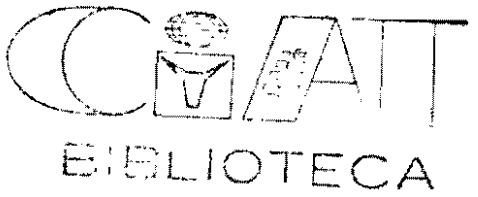

PROCEEDINGS OF FIRST

AFRICAN BEAN PATHOLOGY WORKSHOP

KIGALI, RWANDA
14-16 November 1987

CIAT African Workshop Series No. 20

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P R E F A C E

This volume reports the proceedings of the First Pan-African Pathology Workshop held in Kigali in Rwanda from 14 to 16 November 1987. The workshop was intended to review existing information on bean disease in Africa, identify gaps in our knowledge and develop co-ordinated research programmes to supply needed information. It is just one of a series of workshops that have been organized through the African bean research network with the aim of focusing and co-ordinating research on different aspects of bean production.

To avoid becoming swamped by trivialities, discussions were confined to what are considered the most damaging diseases of common bean in Africa: angular leaf spot, anthracnose, rust, ascochyta blight and common bacterial blight. In these proceedings existing knowledge of each of these diseases is reviewed separately, followed by a consideration of more general topics of interest to pathologists and breeders, such as evaluation methods, the value of varietal mixtures and chemical and cultural disease control. The discussions and conclusions relating to all of these topics are then summarised in a final section. The long delay in publication of these proceedings, arising from staff changes and late submission of a few papers, reduces their value and is much regretted. Nevertheless, it is considered that the costs to the sponsors and the time and effort expended by organizers and participants justify their publication. We hope they will also contibribute to the advancement of our knowledge of diseases of bean in Africa.

The workshop was organized by the Programme Regional pour l'Amelioration du Haricot dans la Region des Grands Lacs and the Institut des Sciences Agronomiques du Rwanda (ISAR) at Butare in Rwanda in collaboration with the CIAT Regional Programme on Beans in Eastern Africa, Debre Zeit, Ethiopia and the SADC/CIAT Regional Programme on Beans in Southern Africa, Arusha, Tanzania. The latter project forms the bean component of the Grain Legume Improvement Programme of the Southern African Centre for Cooperation in Agricultural Research and Training (SACCAR) of the Southern African Development Community (SADC). Funding was provided by: national programmes; the Canadian International Development Agency (CIDA); the Swiss Development Cooperation (SDC); and the United States Agency for International Development (USAID).

Further information on research activities on bean in Africa that are part of these projects is available from:

Pan-Africa Coordinator, CIAT, P.O. Box 23294, Dar es Salaam, Tanzania.

Coordinateur Regional, CIAT, Programme Regional pour l'Amelioration du Haricot dans la Region des Grands Lacs, B.P. 259, Butare, Rwanda.

PUBLICATIONS OF THE NETWORK ON BEAN RESEARCH IN AFRICA

Workshop Series

No. 1. Proceedings of the Bean Fly Workshop, Arusha, Tanzania, 16-20 November 1986.

No. 2. Proceedings of a Workshop on Bean Research in Eastern Africa, Mukono, Uganda, 22-25 June 1986.

No. 3. Proceedings of a Workshop on Soil Fertility Research for Bean Cropping Systems in Africa, Addis Ababa, Ethiopia, 5-9 September 1988.

No. 4. Proceedings of a Workshop on Bean Varietal Improvement in Africa, Maseru, Lesotho, 30 January-2 February 1989.

No. 5. Actes du Troisieme Seminaire Regional sur L'Amelioration du Haricot dans la Region des Grands Lacs, Kigali, Rwanda, 18-21 Novembre 1987.

No. 6. Proceedings of First SADCC Regional Bean Research Workshop, Mbabane, Swaziland, 4-7 October 1989.

No. 7. Proceedings of Second Workshop on Bean Research in Eastern Africa, Nairobi, 5-8 March 1990.

No. 8. Actes de l'Atelier sur la Fixation Biologique d'Azote du Haricot en Afrique, Rubona, Rwanda, 27-29 October 1988.

No. 9. Actes du Quatrieme Seminaire Regional sur L'Amelioration du Haricot dans la Region des Grands Lacs, Kigali, Rwanda, 21-25 Novembre 1988.

No. 10. Proceedings of a Workshop on National Research Planning for Bean Production in Uganda, Kampala, Uganda, 28 January-1 February 1991.

No. 11. Proceedings of the First Meeting of the Pan-African Working Group on Bean Entomology, Nairobi, Kenya, 6-9 August, 1989.

No. 12. Proceedings of African Bean Research Workshop, Morogoro, Tanzania, 17-22 September, 1990.

No. 13. Pan-Africa Working Group Meeting on Virus Diseases of Beans and Cowpea in Africa, Kampala, Uganda, 17-21, January 1990

No. 14. Proceedings of the First Meeting of the SADCC/CIAT Working Group on Drought in Beans, Harare, Zimbabwe, 9-11, May 1991

No. 15. First Pan-African Working Group Meeting on Anthracnose of Beans, Ambo, Ethiopia, February 17-23, 1991.

No. 16. Cinquieme Seminaire Regional sur l'Amelioration du Haricot dans la Region des Grands Lacs, Bujumbura, Burundi, 13-17 Novembre, 1989.

No. 17. Sixieme Seminaire Regional sur l'Amelioration du Haricot dans la Region des Grands Lacs, Kigali, Rwanda, 21-25 Janvier, 1991.

No. 18. Conference sur Lancement des Varietes, la Production et la Distribution de Semaines de Haricot dans la Region des Grands Lacs, Goma, Zaire, 2-4 Novembre 1989.

No. 19. Recommendation of Working Groups on Cropping Systems and Soil Fertility Research for Bean Production Systems, Nairobi, Kenya, 12-14 February, 1990.

Occasional Publications Series

No. 1. Agromyzid Pests of Tropical Food Legumes: a Bibliography.

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No. 3B. First African Bean Yield and Adaptation Nursery (AFBYAN I): Part II. Performance across Environments

No. 4. Assessment of Yield Loss caused by Biotic Stress on Beans in Africa.

Reprint Series

No. 1. Bean Production Problems in the Tropics: Common beans in Africa and their constraints.

No. 2. Bean Production Problems in the Tropics: Insects and other pests in Africa.

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OBJECTIVES

The objectives of the workshop are to bring together the principal scientists working on bean diseases for Africa to:

a) collect information, past and present, about relevant research on the following diseases:

   Angular leaf spot caused by *Phaeoisariopsis griseola*
   Anthracnose caused by *Colletotrichum lindemuthianum*
   Rust caused by *Uromyces appendiculatus*
   Ascochyta blight caused by *Phoma exigua* var. *diversispora*
   Common bacterial blight caused by *Xanthomonas campestris* pv. *phaseoli*

b) identify gaps in the present knowledge, determine research needs and set research priorities;

c) improve coordination of research among pathologists working on bean diseases in Africa so as to maximize the use of limited available resources;

d) discuss the most appropriate evaluation methods, inoculation techniques, and experimental designs to screen germplasm;

Eight different subjects which are regarded as the most essential have been selected for in-depth discussion:

1) identification and collection;
2) distribution, prevalence and economic importance;
3) pathogenic variation;
4) epidemiology in pure stands and mixtures;
5) survival and spread;
6) non-genetic disease control strategies for Africa;
7) sources of resistance for Africa; and
8) evaluation/screening for resistance.

This workshop is intended to be practically orientated, particularly in relation to disease control and research priorities for Africa.

Some important topics, such as clean seed production, have been omitted due to lack of time. However, it is hoped that this workshop is but the first in a series, covering the most important plant diseases and aspects of plant protection relevant to common bean in Africa.

The agenda has been structured to include, where necessary, an initial ten minute review of the literature of each individual disease from an African perspective. Time has also been allocated for participants to supplement the information presented in these literature reviews. Following the reviews, is a general discussion of current research relevant to the topic. From these discussions, it is hoped that conclusions can be drawn on research needs; which subjects warrant priority; what research strategies should be pursued; and, if possible, who should do the work.

The proceedings of this workshop will be published in a document, so it is important that each person giving a presentation provide a written copy of his or her talk. The rapporteurs are expected to provide accurate accounts of the rest of the proceedings.

SECTION 1. ANGULAR LEAF SPOT (*Phaeoisariopsis griseola* (Sacc.) Ferr.)

DISTRIBUTION, PREVALENCE AND ECONOMIC IMPORTANCE

Mukishi Pyndji

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Angular leaf spot disease of the common bean (*Phaseolus vulgaris* L.) caused by *Phaeoisariopsis griseola* (Sacc.) Ferr. is common in almost all regions where common bean is grown. It is distributed from low altitude areas to more than 2,000 masl. The disease has been reported in South America (Brazil, Colombia, Costa Rica, Guatemala, Mexico, Peru and Venezuela), the United States of America, Africa, Australia, Europe, India, Iran, Israel and Japan.

The disease is particularly frequent in tropical and subtropical regions, where high humidity and moderate temperatures (20-25°C) favour epidemic development. Angular leaf spot is found not only in hot, but also in temperate regions. Humidity seems to be the most critical factor producing epidemics in tropical and temperate zones. In the Great Lakes, the disease predominates in southern Rwanda and in the mountainous Kivu Region of Zaire, according to surveys and field observations.

Although angular leaf spot is common in farmers' fields, its severity varies among fields and seasons. It is particularly devastating on susceptible varieties, on which it can provoke premature defoliation often leading to reduced yields due particularly to smaller grains. The importance of the disease has been demonstrated by several studies carried out in various bean producing regions. Harvest losses of 50% or more have been reported in farmers' fields in the U.S.A. In Colombia, losses have been estimated at between 40 and 60%, while they reached 80% in Mexico. In Brazil, average losses of 31% have been obtained after two years. Trials carried out in Kivu (Zaire) have shown that angular leaf spot, alone or in association with other leaf infections, can cause average yield losses of between 20 and 60% depending on season.

Due to association of angular leaf spot with other diseases, *P. griseola*'s rôle in yield reduction in the Great Lakes region is difficult to determine. In exploratory trials in Rwanda, where *P. griseola* was among the predominant pathogens (together with *Ascochyta phaseolorum* and *Colletotrichum lindemuthianum*), yield losses of between 57 and 59% were recorded.

In conclusion, *P. griseola* is distributed over a wide area. Its occurrence and its severity are high in regions where conditions are favourable for the development of symptoms. The yield losses recorded represent only estimates made from protected versus unprotected plots. It is therefore not possible to extrapolate the results to field conditions. In addition, *P. griseola* is always found in association with other pathogens, especially in the Great Lakes, complicating the measurement of the yield losses it causes.

PATHOGENIC VARIATION

Mukishi Pyndji

Station de Recherches Agronomiques, Mulungu, Zaire

Evaluations of the disease in the field show that there is variability within the angular leaf spot fungus, *P. griseola*. For example, the variety BAT 332 shows an immune reaction or mild symptoms of the disease in Colombia, whilst it is severely attacked in several sites in Brazil. This observation shows that isolates of *P. griseola* differ among regions (CIAT, 1984).

Villegos (1959) identified thirteen different pathogenic races, but the genetic purity and uniformity of the differential varieties used were not certain. Moreover, Hocking (1967) found an isolate in Tanzania producing atypical (circular) lesions. This isolate was highly virulent at a concentration of 10^2 spores/ml, whereas in most work (Alvarez-Ayala and Schwartz, 1979), isolates of *P. griseola* are virulent at a concentration of 10^4 spores/ml. The origin of this isolate in Tanzania is due, according to the author, to a simple mutation in natural isolates.

Studies have shown that spore concentration is no more important than age of plant in the expression of symptoms and the development of the disease. Cardona-Alvarez and Walker (1956) reported that plant age was not important for development of symptoms. Olave (1958) found disease symptoms appeared eight days after infection but were more characteristic at 12 days. No special difference was observed in the degree of infection of plants infected at 20 and 30 days of age. Verma and Sharma (1984) observed that variation in lesion size was due to temperature variation and not to differences in isolates.

In the Great Lakes, lines from the BALSIT (Bean Angular Leaf Spot International Trial), evaluated in Zaire and Rwanda, expressed similar reactions indicating that the isolates in these areas are similar.

In conclusion, the existence of different pathogenic races of *P. griseola* implies the development of varieties resistant to several strains (races) of the pathogen. The sources of resistance identified should be exposed to different populations of the pathogen in order to improve breeding strategy and to create varieties with multiple resistance to the races existing in a given region. Moreover, the differential varieties should be well-defined so as to determine with some precision whether the isolates found in various regions are really different. In this case, the technique used to determine the resistance of the host should be the same as that used to identify strains of the pathogen.

SOURCES OF RESISTANCE FOR AFRICA

Mukishi Pyndji

Station de Recherches Agronomiques, Mulungu, Zaire

Local materials with resistance to angular leaf spot are not well known in Africa. In regions where the disease is rife, evaluations have sometimes been made with material collected in the areas concerned in order to determine their resistance. In Tanzania, the following local accessions were judged to have resistance to angular leaf spot : TMO 320, TMO 333, TMO 334, TMO 335, TMO 341, TMO 354, TMO 357 and TMO 191. In Kenya two accessions have been identified as resistant to the disease: GLP-24 and GLP-x.92.

In other African countries, where the disease is more important, notably Zaire, Rwanda and Zambia, sources of resistance have been identified from the Bean Angular Leaf Spot International Trial (BALSIT) received from the Centro Internacional de Agricultura Tropical (CIAT). In Zaire, the material in this nursery was evaluated during five growing seasons, 1985B to 1987B. A large number of lines showed resistance during these five seasons (Table 1).

Table 1: Some sources of resistance to *Phaeoisariopsis griseola* identified in the field at Mulungu during five growing seasons (1985B-1987B).

Entries	Growth habit	Seed colour	Seed size
A 140	111	Cream	Small
A 216	111	Black	Small
A 221	11	Black	Small
A 345	11	Cream	Small
A 285	111	Cream/striped	Small
A 367	11	Yellow	Small
A 75	11	Cream/striped	Small
A 300	11	Cream	Small
A 339	11	Cream	Medium
A 163	11	Cream	Small
A 240	11	Cream/striped	Small
A 384	11	Brown	Small
A 212	11	Black	Small
BAT 76	11	Black	Small
BAT 434	11	Black	Small
BAT 1432	11	Black	Small
BAT 67	11	Black	Small
G 2676	11	Black	Small
G 4169	111	Black	Small
G 4459	1	White	Small
G 2959	1	Black	Small
G 5473	11	White	Small
XAN 37	11	Pink	Small
XAN 68	11	Cream	Small
EMP 81	111	Cream	Small

Other lines showed variable reactions during the evaluations. Most of the material had either a resistant or intermediate and sometimes even a susceptible reaction. Several entries from the BALSIT identified as resistant in Zaire were found to be equally resistant in Rwanda. Among these entries we can cite: A 240, A 367, A 339, A 285, A 75, A 300, A 384, BAT 67, BAT 76, G 2676, G 4459 and XAN 37. Materials resistant in Africa are of intermediate resistance or susceptible in South America (Julia Kornegay, personal communication).

Sources of resistance can also exist among local materials found on farm. For this reason, a collection was made in northern and southern Kivu. The local mixtures collected were sorted according to the different cultivars making up the mixture. Next, these cultivars were sown in a disease nursery in order to screen them for resistance to angular leaf spot. The accessions identified as resistant will in addition be subjected to artificial infection with different isolates of the region before their use by breeders.

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SECTION 3: ANTHRACNOSE
(Colletotrichum lindemuthianum (Sacc. & Magn.) Bri.& Cav.)

DISTRIBUTION, PREVALENCE AND ECONOMIC IMPORTANCE

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INTRODUCTION

Anthracnose, caused by *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav. is one of the most common and destructive of the diseases of beans (*Phaseolus vulgaris*), occupying an important position among the constraints to bean production in Africa (CIAT, 1980). The disease is found widespread in susceptible cultivars throughout all the regions where beans are produced, being most devastating where relative humidities are high and temperatures lie between 13 and 27°C. Damage is much less under dry conditions, which are unfavourable for disease development. Anthracnose is the principal disease of common bean in eastern Africa (Leakey, 1970; Leakey and Simbwa-Bunnya, 1972) particularly at altitudes of 1200-2400 masl near to the equator.

The disease is seen in the crop approximately 4-6 weeks after sowing. The symptoms occur on leaves, stems and pods. They appear first on the lower surfaces of the leaves as a dark veinal necrosis. Dark coloured, depressed cankers form subsequently on stems and pods - conidial masses appear as salmon-pink patches in the pod lesions. The pod lesions penetrate to the seeds, which become infected and serve to propagate the disease.

ECONOMIC IMPORTANCE

Anthracnose can cause total crop loss where contaminated bean seeds are sown in conditions favourable for disease development. Severe losses have been recorded in Africa, Australia, North America, Europe and the Latin American countries, Mexica, Costa Rica, Guatemala, Venezuala, Colombia and Brazil (Schwartz and Galvez, 1980).

TIME OF INOCULATION AND YIELD

In trials at Rubona, yield losses of three cultivars (Kilyumukwe - resistant; Rubona 5 - intermediate: and Ikinyange - susceptible) were greatest following inoculation with *Colletotrichum lindemuthianum* at the cotyledonary stage and progressively declined with delay in inoculation (Table 2). Yield loss was greatest when plants were inoculated at all four stages of growth. The yields of the resistant cultivar (Ikinkange) were reduced less by inoculation than those of the other cultivars.

INCIDENCE OF ANTHRACNOSE IN MALAWI

Anthracnose was present in bean crops in all areas of Malawi that were sampled confirming its importance in this country (Table 3).

Table 2. Effects of time of inoculation with *Colletotrichum lindemuthianum* on the percentage losses in yields of three cultivars of *Phaseolus vulgaris*.

Stage of inoculation[1]	Cultivar			Mean
	Kilyumukwe	Rubona 5	Ikinyange	
Uninoculated	0	0	0	0
I	13.1	25.5	30.2	22.9
II	10.7	19.2	24.4	18.1
III	7.9	15.4	18.7	14.0
IV	4.8	8.9	11.6	8.4
I+II	17.7	32.6	37.4	29.2
II+III	14.6	22.8	26.9	21.4
III+IV	11.5	22.4	24.6	19.5
I+II+III	23.4	38.3	41.2	24.3
II+III+IV	20.0	23.2	28.8	24.0
I+II+III+IV	25.3	40.7	43.5	36.5

[1] I = cotyledonary; II = second trifoliolate leaf; III = flower bud formation; IV = pod formation

Table 3. Incidence of anthracnose in bean crops in Malawi.

Location	Number of samples	Percentage of samples infected
Chitipa	311	6.4
Karonga	119	5.9
Rumphi	183	2.7
Mzimba	545	9.5
Dedza	234	6.8
Dowa	135	5.2
Lilongwe	150	10.7
Ntcheu	15	6.7
Ntchisi	2	50.0
Chikwaw	80	8.8
Thyolo	164	4.3
Mangochi	190	10.5
Mulanje	129	6.2
Nsanje	512	3.7

IMPORTANCE OF ANTHRACNOSE IN AFRICA

Anthracnose is considered the most important disease of common bean in five of the six countries listed in Table 4. Rust usually ranks second or third; angular leaf spot is third to fifth; BCMV is considered next most important; while halo blight is especially important in Kenya. Ascochyta is considered least important of all these diseases except in Rwanda, where it causes a certain amount of damage.

Table 4. Country priorities in Africa for diseases of common bean.

Country	Anthr-acnose	Halo blight	BCMV	Angular leaf spot	Rust	Bacterial blight	Ascochyta blight
Kenya	3	1	2	4	5	-	-
Malawi	1	2	-	5	4	3	-
Rwanda	1	-	3	3	2	3	2
Tanzania	1	-	4	3	2	-	-
Uganda	1	-	-	4	3	2	-
Zambia	1	-	4	3	3	-	-

CONCLUSIONS

Considering the constraints to bean production imposed by anthracnose, the release of resistant cultivars and application of other methods of control of the disease should be emphasised in the African countries surveyed.

SOURCES OF RESISTANCE FOR AFRICA

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INTRODUCTION

The development of resistant cultivars is the most practical means of reducing damage to bean crops in Africa through anthracnose. Knowledge of the distribution of pathogenic variation in Africa points to areas where genetic resistance may be identified and provides the information needed for its effective deployment.

Research by CIAT in collaboration with national programmes (CIAT, 1987) reveals that isolates CL-1 AFR, CL-3 AFR and CL-AFR of *Colletotrichum lindemuthianum* from Kenya, Tanzania and Zaire are Race Beta, Group XII; two isolates (CL-2AFR from Zaire and CL-4AFR from Burundi) belong to Race Brasil I, Group IV; and another isolate from Kenya (C1-6AFR) is Race Mexico II, Group IV (Table 5).

Table 5. Reactions[1] of differential varieties to isolates of *Colletotrichum lindemuthianum* from Africa.

	Isolates					
Cultivar	KEN CL-1	TAN CL-3	ZAI CL-5	ZAI CL-2	BUR CL-4	KEN CL-6
Michelite	1.1	1.7	2.8	5.4	9	9
M.D.R.K.	9	9	9	1.7	1.6	9
Perry Marrow	1.5	1	2.8	4.7	6.5	2.4
Cornell 49242	1.1	1	1.1	1.6	1.5	1.3
Widusa	1	1	1.7	1	1	1
Kaboon	1	1	1.1	1.7	1.7	1
Samilac	1	1	2.2	8	9	1
To	1	1	1	6.2	7.6	1
Tu	1	1	1	1	1	1
P.I. 207262	1	1	1	7.7	8	1
AB 136	1	1	1	1	1.1	1
A 475	3.1	3	5.9	1/9[a]	1/9[a]	3.1
G 2328	1	1	1	1	1	1
Race/group	Beta	Beta	Beta	Brazil I	Brazil I	Mx II

[1] on scale of 1-9, where 1 indicates no symptoms and 9 indicates highly susceptible; means of ten plants
[a] some plants were immune and others highly susceptible

In attempting to incorporate resistance in the Great Lakes, it was surprising to find three African cultivars with resistance to all the isolates of anthracnose used. They were Habyalimana, Cyunyu and C 10 (Table 6) - all from Rwanda. BAT 1386 was also resistant to all isolates. Urubonobono, which is considered susceptible to anthracnose in Burundi, its country of origin, was resistant to all isolates except Group VI from Burundi and Zaire. Mutiki 2 from Uganda and Kibungo 2 from Rwanda were susceptible to all isolates except for those of Group IV. It should be possible to combine genes for resistance to different isolates by crosses among resistant materials.

EVALUATION OF RESISTANCE TO ANTHRACNOSE IN RWANDA

The reactions of new sources of resistance to anthracnose at Rubona in 1986 and 1987 (ISAR, 1987) to local races of the pathogen are shown in Table 7.

Table 6. Reactions to *Colletotrichum lindemuthianum* of bean cultivars and lines grown in the Great Lakes.

Cultivar/ line	Isolates					
	KEN CL-1	TAN CL-3	ZAI CL-5	ZAI CL-2	BUR CL-4	KEN CL-6
Calima	6.7	9.0	7.4	1.1	1.0	9.0
BAT 1386	1.0	1.0	1.4	2.8	3.0	1.0
A 484	3.4	3.4	7.5	1.2	1.0	5.5
A 475	2.5	3.7	4.4	9.0	9.0	3.6
A 240	1.0	1.0	1.0	9.0	9.0	1.3
A 483	3.0	6.1	8.0	1.0	1.0	4.8
A 30	1.0	1.0	1.0	9.0	8.8	1.0
A 252	1.0	1.0	1.0	4.5	4.8	1.0
PVMX 1535	1.0	1.0	1.0	9.0	9.0	1.0
G 2333	1.0	1.0	1.0	1.8	1.0	1.0
Rubona 5	8.6	9.0	7.2	7.3	8.7	8.8
Mutiki 2	8.0	8.3	6.3	1.0	1.0	9.0
Tostado	9.0	9.0	9.0	1.8	1.0	9.0
Kibungo 2	9.0	9.0	9.0	1.8	1.0	9.0
Karama	9.0	9.0	9.0	8.8	8.8	9.0
Habyalimana	1.0	1.0	1.0	1.9	1.6	1.5
Carolina	1.0	1.0	1.0	8.0	1.0/9.0	1.1
Cyunyu	1.0	1.2	1.0	1.9	1.1	1.1
Urunyumba 3	4.8	8.8	7.6	1.2	1.0	3.4
Gisengi 6	6.1	7.3	7.5	3.2	3.9	5.3
Urubonobono	1.3	1.0	1.0	7.7	9.0	1.6
C 10	1.1	1.0	1.0	2.2	3.6	1.5

Table 7. Reactions of new sources of resistance to *Colletotrichum lindemuthianum* at Rubona in 1986 and 1987.

1986		1987			
Line	Reaction	Line	Reaction	Line	Reaction
G 2816	1	G 11521	1	EMP 143	1
P.I. 165426	1	PVA 46	1	PVA 1258	1
BAT 1275	1	RWR 65	1	BAN 27	1
G 12727 (AB 136)	1	PVA 784	1	ZAA 84099	1
Cornell 49242	1	Kanyamanza 2	1	MCD 255	1
G 2333	1	PVA 784	1	DOR 308	1
G 3991	1	PVA 1377	1	RAB 214	1
G 7199	1	PVA 3004	1	RAB 211	1
A 411 (res)	1			Rubona 5 (sus)	7.3
A 336 (res)	1				
Rubona 5 (sus)	6.8				

CONCLUSIONS

Work to determine the distribution of races of the anthracnose pathogen predominating in each country is important and should continue. This would permit the introduction of genotypes which are resistant to the races present in a specific region. Wider adaptation would be achieved by the identification or development of genotypes with resistance to several races of anthracnose. The anthracnose resistance of existing materials should be incorporated into cultivars which are highly productive but susceptible to the pathogen to improve their resistance and productivity.

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SECTION 3: RUST (*Uromyces appendiculatus* (Pers.) Unger)

DISTRIBUTION, PREVALENCE AND ECONOMIC IMPORTANCE

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Bean rust is caused by *Uromyces appendiculatus* (Pers.) Unger. The disease is found worldwide, wherever beans are grown. It causes considerable production problems in some parts of Africa. Bean rust is reported from Burundi (Devos and van Durme, 1982), Ethiopia (Stewart and Dagnatchew Yirgue, 1967), Malawi (Edje *et al.*, 1973.), Kenya (Acland, 1971; Hubbeling, 1973; Mukunya, 1974), Uganda (Acland, 1971; MOA, 1983), Tanzania (Acland, 1971; Brockman, 1975), Rwanda (ISAR, 1982), Zaire, Zimbabwe and Zambia (personal communications).

Acland (1971) reported bean rust to be the major cause of crop losses and yield fluctuations in bean in eastern Africa. Moreover, in the absence of quantified figures, Mukunya (1974) and Hubbeling (1973) have indicated bean rust as the major fungal disease of bean in Kenya. Brockman (1975) attributed large losses to bean rust in the high elevation production areas of Tanzania. Edje *et al.* (1973) suggested that bean rust continued to be a major disease of bean in Malawi. There is thus, ample information to indicate the widespread occurrence of bean rust in eastern and southern Africa. However, the economic importance of bean rust has not been estimated sufficiently to establish its significance. Despite such limited information Padwick (1956) has estimated a 10% yield loss due to bean rust. He, however, did not mention the infection level and cultivars. Subsequently, Singh and Musyimi (1981) provided a fair assessment of the economic significance of bean rust, indicating a small range, between 3 and 38% at 3 and 79% infection levels, respectively. A report from Morogoro, Tanzania revealed that protection of bean by fungicides increased yields by 10.3% (Howland and Macartney, 1966). In national variety trials at Awassa in Ethiopia (Stewart and Dagnatchew Yirgue, 1967), three varieties that were extremely susceptible (100% infected) produced no seed at all.

Though limited disease loss estimates are available in some countries, they are obtained either from selected research centres or on selected variety which may or may not be extrapolated to other similar regions in Africa. This needs to be investigated further in any future bean rust research program.

SURVIVAL AND SPREAD

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U. appendiculatus is widespread throughout the tropics and subtropics, extending also into temperate regions. Its host range is tribe-specific, being effectively confined to the genera *Phaseolus*, *Vigna* and *Lablab* of the Phaseoleae. Between them, these genera contain species native to the Americas (bean and lima bean), Africa (cowpea and bambarra groundnut) and Asia (mung bean and hyacinth bean); and no single ancestral host, nor region of origin, is readily discernible (Allen, 1983).

The international dissemination of bean rust is presumed to have occurred principally by means of windborne urediospores. Seed transmission probably plays a negligible role in the long distance dissemination of bean rust, although there is indirect evidence on at least one occasion that *U. appendiculatus* may be seedborne (Deighton, 1945).

U. appendiculatus is an autoecious, macrocyclic rust which has a full complement of spore forms. Secondary dispersal occurs as urediospores and is favoured by cloudy, humid weather with heavy dew and temperatures in the range of 21-27°C (Schein, 1961). Urediospores are disseminated principally by wind and to a lesser extent through contact with animals, including man and his implements (Laundon and Waterson, 1965), and probably also by insects (Zaumeyer and Thomas, 1957). Urediospore production and release are also influenced by environmental factors. It has been estimated that *U. appendiculatus* can produce 10^6 urediospores/cm on leaves bearing 2-100 pustules/cm (Yarwood, 1961); such a sporulation capacity is about ten-fold that of the soybean rust pathogen, *Phakopsora pachyrhizi* (Melching *et al.*, 1979) suggesting that bean rust has a relatively greater capacity for secondary spread.

The relative importance of the different spore forms in the seasonal carry-over of bean rust is controversial and is likely to vary with locality. The production of teliospores, which require a rest period before germinating (Zaumeyer and Thomas, 1957), is generally considered to be in part under environmental control. Waters (1928) found that factors such as light, temperature and moisture so influenced the host that the rust fungus reacted by changing from the uredial to the telial stage or, under proper manipulation, in the reverse. Dundas and Scott (1939) concluded that the proportion of teliospores of bean rust increased not only with increasingly unfavourable conditions such as advanced leaf age, but also with host plant resistance. This is supported by Rothman (1974). Hyperparasitism may also induce teliospore production in some rust fungi (Biali *et al.*, 1972).

Harter *et al* (1935) found that bean rust collections from climatically distinct areas had different abilities to produce teliospores and that these differences could not be ascribed to environment alone but also to genetic variation in the fungus. Working with ten isolates of diverse origin, Allen (1975a) found that only six produced teliospores under controlled conditions. Teliospores were most commonly found in British isolates (086 and 089), in which production was occasionally abundant, especially developing as

secondary sori surrounding a primary uredium. Of the eastern African isolates, 025, 041 and 076 occasionally produced telia but, under the same environmental conditions (growth chamber) and on the same host varieties, never as abundantly or as frequently as the British isolates (Table 8).

Table 8. Production of teliospores of bean seedings by isolates of *U. appendiculatus* of diverse origin.

Isolate no.	Origin	Host	Frequency	Days from inoculation with urediospores
089	England	Seaway		26
025	Uganda	Seaway		29
089	England	Seaway		15
086	England	Seaway		21
025	Uganda	USDA No.814		35
		Seaway	Abundant	18
		Seafarer	Abundant	18
089	England	Bunda 1101/1	Abundant	18
		Bunda 240/2	Abundant	18
		Mutike 4	Abundant	18
		V 3210	Abundant	18
		Pinto 5	Abundant	18
		Mexico 142	Abundant	18
		US No. 3	Abundant	18
		USDA No.780	Abundant	18
		USDA No. 181	Abundant	18
		USDA No. 650	Abundant	18
		Golden Gate Wax	Abundant	18
086	England	Bunda 1101/1	Common	18
		USDA No. 780	Common	18
		USDA No. 181	Common	18
110	Colombia	Mutike 4	Rare	18
089	England	Seaway	Abundant	22
086	England	Seaway	Common	22
041	Uganda	Seaway	Rare	22
076	Kenya	Seaway	Rare	22
110	Colombia	Seaway	Common	28

These data confirm that there is variation in the ability to produce telia between isolates from climatically distinct regions. For instance, teliospores are the major source of overwintering inoculum in Oregon where it has been shown that the spores survive on stakes used for supporting climbing varieties of bean (Milbrath, 1944). The fungus probably also overwinters as teliospores in Brazil (Netto *et al.*, 1967) but telia are apparently rarely seen in eastern Africa (Atkins, 1973). In areas where telia are rare, rust epidemics presumably depend either on the transport of urediospores from elsewhere (Townsend, 1939) or on the direct overwintering of urediospores (Marcus, 1952; Fromme and Wingard, 1921).

With respect to the other spore forms of this macrocyclic rust, little is known of the significance of aecia; the extent to which aecia contribute to the evolution of novel pathogenic variants seems unknown and, in any case, aecial development appears to be rare in nature (Jones, 1960; Groth and Mogen, 1978).

Furthermore, little is known about the many species of *Aecidium* described from legumes in the tropics, and it seems possible that some may represent hitherto unknown aecial stages of otherwise well-known species. *Aecidium caulicola* P. Henn. and *A. vignae* Cke from cowpea in Africa (Snowdon, 1921; Allen, 1979) are recognized synonyms of *U. appendiculatus* (Laundon and Waterson, 1965).

Like the well-known heteroecious wheat stem rust fungus, *Puccinia graminis*, in which there is an alternation of generations (and spore forms) with season between the herbaceous wheat and the perennial woody barberry, there are also heteroecious species of *Uromyces* which alternate predominantly between a legume and *Euphorbia* (Olive, 1911; Butler, 1958) : but all seem to be temperate not tropical species. *U. appendiculatus* is well known to be autoecious. Nor is there any evidence that other legume species serve significantly as reservoirs of infection.

Carryover is likely to be confined to volunteer bean seedlings and to the growing of successive crops of bean in areas of evenly distributed rainfall. So what are the implications of what is known about bean rust epidemiology as regards potentials for cultural control? Bean rust is widely distributed despite not being seedborne. No alternate hosts are present in the life cycle, nor are alternative uredial hosts known to be epidemiologically significant. As a pulverulent (dry spore) rust, with a relatively high productive capacity and dependence upon windborne inoculum, there are opportunities for impeding effective dispersal and deposition by protecting susceptible bean crops with non-host barriers (e.g. a maize:bean intercrop), and by interspersing susceptible plants within genetically diverse varietal mixtures, each of which has shown potential in decreasing the severity of rust (van Rheenen *et al.*, 1981; Msuku and Edje, 1982; Lyimo and Teri, 1984).

PATHOGENIC VARIATION

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Many workers have observed that bean cultivars vary in their reaction to infection by *U. appendiculatus* and that the pathogen possesses much pathogenic variability.

Though many cultivars possessing resistance to one or more races have been identified, genotype immune to all existing physiologic races has been found. This has prompted scientists to investigate the various physiologic races of bean rust prevalent in their region.

Variation in natural populations of the pathogen has been noted in South and Central America, Australia, Europe, U.S.A. and eastern and southern

Africa. Physiologic races of bean rust were identified in Brazil in the 1960s. Augustin and da Costa (1971) and Netto *et al.* (1969) have listed more than 40 physiologic races. Augustin and da Costa's report suggests the prevalence of 16 races, designated B1-B16, of which five were found the most frequent. In an earlier investigation Netto *et al.* (1969) identified 26 races (FM1-FM26), which they suggested were rather different than the ones reported elsewhere.

New races of bean rust have also been reported in other Latin American countries. They include: four races in Peru (Concepcion and Laroya, 1972); eight in Costa Rica (Christen and Echandi, 1967; Vargas, 1967); 31 in Mexico (Crispin and Dongo, 1962; Dongo and Crispin, 1962); 12 in Puerto Rico (Lopez, 1976); and 11 in Colombia (Zuniga de Rodriguez, 1974) In eastern Australia, Ballantyne (1975) selected 11 races of bean rust and surveys made from 1954 to 1970 in Queensland, Australia identified eight more races (Ogle and Johnson, 1974), indicating the diversity of the pathogen under Australian conditions.

From laboratory screening of bean rust isolates, Howland and Macartney (1966) identified eight races in eastern Africa and Macartney (1966) described six new races in Tanzania, where he also found cv. Tengeru 8 resistant to all the races identified. Allen (1975b) identified six races in Malawi. Unfortunately, these races have not been compared for their similarity. In general, information on bean rust races in Africa is limited. Comparisons are difficult to make because standard differentials and assessment methods were not used. There is a need to standardize these procedures and techniques and establish at least the predominant races existing in various bean growing regions in Africa.

SOURCES OF RESISTANCE FOR AFRICA

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Disease resistance is an important component of an overall bean rust control strategy. For the African small farmer, varietal resistance is both the safest and most economic. Bean varieties grown in many parts of the world are highly vulnerable to bean rust. This may be due to the presence of a narrow genetic base in certain localities.

Considering the importance of bean rust, several bean growing countries have embarked upon extensive and well-devised disease screening programmes and breeding for resistance. Sources of materials include local collections and those obtained through international seed exchange. In Africa, some attempts have been made to develop varieties resistant to bean rust. This review focuses on research results available in some parts of Africa and suggests strategies for the future.

In the early 1960s, Macartney and his colleagues in Tanzania screened several varieties of bean and established some lines resistant against bean rust (Macartney, 1966). The first of these series of trials was reported in 1962, when several varieties were planted in a series of rust nurseries for

field testing. In the Arusha-Tengeru area, 29 varieties were found completely resistant to rust while in the Olmolong-Moshi area, 12 varieties were established as rust resistant. Race 2 was the most virulent race.

Macartney further evaluated 26 varieties of Tengeru origin against six of the eight prevalent races. His results suggested Tengeru 8, 9, 14 and 19 to be highly resistant to all races; Tengeru 16 was susceptible to races D and F, while Tengeru 4 was susceptible to E and F.

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SECTION 4: ASCOCHYTA BLIGHT (*Phoma exigua* var. *diversispora*)

CLASSIFICATION, DISTRIBUTION, PREVALENCE, ECONOMIC IMPORTANCE, SURVIVAL, SPREAD, PREVENTION AND CURE

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INTRODUCTION

Ascochyta leaf spot of bean has long been known as a minor disease (Zaumeyer and Thomas, 1957). The disease was ascribed to two pathogens, *Ascochyta phaseolorum* Sacc. and *A. boltshauseri* Sacc. For about a decade, problems with ascochyta blight have been regularly described, especially by workers at CIAT in Colombia and *A. phaseolorum* has been held responsible. In Europe, work by Boerema *et al.* (1981) identified the causal organism of a serious blight, not unlike the South American one, as *Phoma exigua* Desm. var. *diversispora* (Bub.) Boerema. Information from this research, together with additional information on occurrence and spread of the pathogen concerned, will be discussed in the present paper.

SYMPTOMS AND MICROSCOPIC STRUCTURES

Boerema (1972) had already concluded that *A. phaseolorum* was synonymous with *P. exigua* Desm. The principal distinction between *Phoma*, with a phialidic ontogeny of the conidia and *Ascochyta*, with annellidic ontogeny, has been described in detail in Boerema and Bollen (1975). According to Boerema and Verhoeven (1979), the fungus is still more specifically described as *Phoma exigua* var. *exigua*. This fungus is a weak pathogen, causing leaf spots on various species in different families. It is responsible for leaf and pod spots of bean at the end of the growing season.

A second pathogen, more or less similar in symptoms and occurrence, is *Stegonosporopsis hortensis* (Sacc. & Malbr.) Petr., formerly known as *A. boltshauseri*. (Boerema and Verhoeven, 1979). These fungi are compared to the typically pathogenic *P exigua* var. *diversispora* in Table 9 (based on Table 3 of Boerema *et al.*, 1981).

PREVALENCE AND DAMAGE

Exact estimates of the prevalence of the three different pathogens are difficult to give. Though some authors have made a distinction between the pathogens already at an early stage (Bubak and Kabat, 1905; Anon., 1972), others most probably have mistaken *P. exigua* var. *diversispora* for *P. exigua* var. *exigua* (*A. phaseolorum*) (Anon., 1980; Price and Cishahayo, 1986). It can be stated that *P. exigua* var. *exigua* is scarcely virulent, *S. hortensis* is moderately virulent (Sneep, 1945) and *P. exigua* var. *diversispora* is highly virulent. All three need humid conditions to develop epidemics. They therefore occur in all zones where bean is grown without irrigation. They

prefer moderate temperatures. This explains their importance in bean production in summer in temperate regions and in high altitudes in the tropics. In temperate regions *P. exigua* var. *exigua* and *S. hortensis* may cause severe disease at the end of the growing season, when the end of the summer also means long nights, accompanied by strong cooling and high relative humidity. In Europe, *P. exigua* var. *diversispora* has caused severe problems only in extremely wet and cold summers. At high altitudes in the tropics, humidity tends to be rather high for long periods, thus explaining the regular occurrence of ascochyta blight.

Table 9. Diagnostic characters of *Phoma exigua* var. *diversispora*, *P. exigua* var. *exigua* and *Stagonosporopsis hortensis* on phaseolus beans.

Species	*Phoma exigua* var. *diversispora*	*Phoma exigua* var. *exigua*	*Stagonosporopsis hortensis*
Synonyms	*P. diversispora*	*A. phaseolorum*	*A. boltshauseri*
Diseases	black node	blotch or leaf spot speckle	leaf spot
Symptoms	leaf blight in all growth stages; blackening at nodes and ends of pods; numerous pycnidia, especially on stems and pods; plants may be killed	small specks on older leaves and pods; sometimes pycnidia	reddish brown, small spots on leaves pods and stems; some pycnidia; plants may be somewhat stunted
Microscopic structures			
in vivo	pycnidia mu conidia, 1-2 celled, 6.8 x 2.7 mu	pycnidia, 150 mu; conidia 1-2 celled, 6 x 3 mu	pycnidia, 150 mu; conidia 1-3 celled, 10-27 x 3-6 mu
in vitro	conidia mostly 1 celled, 6.8 x 2.7 mu	conidia mostly 1 celled, 6 x 3 mu	conidia mostly 1 celled, 7 x 2.5 mu
Discoloration of culture medium with NaOH			
	none (no E)	green (production of E)	none (no E)

The damage caused by the pathogen depends largely on the humidity of the season, the starting dose of inoculum and the exact timing of conditions for the development of the disease. Munyemana (1987) reported reductions in yield of the order of 50% with inoculation at flowering time. In Europe, attack of snap bean has resulted in economic losses of 100% in infected crops, since even a small percentage of pod attack renders the crop valueless for canning.

Little is known about pathogenic variation. Munyemana (1987) found greater virulence in isolates from beans at higher altitudes in Rwanda. But I did not observe such a relationship when studying samples of ascochyta blight received from South and Meso America and eastern and central Africa. Neither was there any clear difference according to the host from which the pathogen had been isolated (Table 10). It should be noted, that *P. exigua* var. *diversispora* is a pathogen of many leguminous grain crops. *P. vulgaris* and *Vigna unguiculata* are very susceptible. According to Boerema (1982) *V. unguiculata* is most probably the primary host of the fungus.

Table 10. Positive identification of *Phoma exigua* var. *diversispora* or a very near relative in material of different origin.

Collector	Source	Crop	*Phoma exigua* var.
D.J. Allen, 1985-86	Burundi	*P. vulgaris*	*diversispora*
	Rwanda	*P. vulgaris*	*diversispora*
	Uganda	*P. vulgaris*	*diversispora*
	Tanzania	*P. vulgaris*	*diversispora*
	Zambia	*P. vulgaris*	*diversispora*
	Rwanda	*P. coccineus*	*diversispora*
	Kenya	*P. lunatus*	*diversispora*
	Zimbabwe	*P. lunatus*	*diversispora*
	Zambia	*P. lunatus*	*diversispora*
	Zambia	*V. radiata*	*diversispora*
	Kenya	*V. angularis*	*diversispora*
P. Trutmann 1986-87	GLR (Rwanda?)	*P. vulgaris*	*diversispora*
	Guatemala	*P. vulgaris*	*diversispora*-like
J. Kornegay 1987	Colombia	*P. vulgaris*	*diversispora*-like
	Peru	*P. vulgaris*	*diversispora*-like
IPO	Netherlands (Germany, France)	*P. vulgaris*	*diversispora*

Examination of the isolates from the material in Table 10 by Miss M.M.J. Dorenbosch at the Plant Protection Service, Wageningen, the Netherlands, has revealed consistent differences in cultural characteristics between the isolates from the western hemisphere and those from other origins. The same observation had already been made in 1979 with material from Colombia compared to isolates from Western Europe. It is still open to further research whether the isolates from South and Meso America are also var. *diversispora* but, considering symptoms on the plant, spore form and size and lack of coloration of the culture medium with NaOH, this appears likely.

SURVIVAL AND SPREAD

The fungus can survive on bean straw. After incorporation of infected straw into soil, next summer's crop showed some black node disease on an

experimental field in the Netherlands. Dry conservation of straw above soil will certainly permit the pathogen to survive for longer periods. The best conservation, however, is in contaminated seeds. Seeds from diseased plots can be contaminated to a high degree (Table 11) and are not safe even at some meters from a focus.

Table 11. The relationship between field attack and seed infestation; *Phoma exigua* var. *diversispora*/*P. vulgaris* (Ester, 1981).

	Position in field	No. of germinated seeds	No. of plants with black node	Per cent disease
5m	A	240	8	3.4
A B C	B	932	17	1.8
focus	C	599	8	1.4

Young plants developing from contaminated seeds may produce pycnidia from which spores may spread by rain splash. If the relative humidity is sufficiently high for a long period, this may lead to heavy attack of the crop. The necessity of high humidity for successful infection cannot be overstressed. In the greenhouse, with heat in winter or spring, artificial inoculation should be followed by coverage of the inoculated plants with a plastic lid for even five or six days, until appearance of symptoms (Boerema *et al.*, 1981). Interruption of 100% relative humidity after four days inhibited development of clear symptoms.

PREVENTION AND CURE

Priority should be given to prevention of attack of the crop by *P. exigua* var. *diversispora*. Clean seed ranks first as a practical method to prevent epidemics. Seeds should preferably be taken from healthy crops. If seed is suspected to be contaminated, treatment is necessary. Experiments have shown that seed treatment with classical fungicides like thiram gives reasonable protection. Since benomyl increased effectiveness, it was concluded that thiram only kills superficial contamination, whereas the systemic fungicide kills the pathogen within the seed (Table 12).

Research on resistant germplasm has started. Many accessions of *P. coccineus* are claimed to have good resistance (Anon., 1987). Munyemana (1987) mentioned resistance to occur in climbing bean. Both might at least partially be due to better aeration of a supported, climbing crop. To the best of my knowledge no absolute resistance has yet been detected.

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I gratefully acknowledge isolation of numerous cultures and execution of inoculation experiments by Miss I. Vos.

Table 12. Effect of treatment of seed infested with *Phoma exigua* var. *diversispora*. (Ester, 1981).

Fungicides and rates	Active ingredient	Per cent diseased plants
Aatifon, 4g/kg	thiram, 1g	0.7
Aatifon, 4g/kg + Benlate, 1g/kg	thiram, 1g + benomyl, 0.5g	0
Aatifon, 4g/kg + Bavistin, 1g/kg	thiram, 1g + carbendazim, 0.5g	0
Aatifon, 4g/kg + Topsin M, 1g/kg	thiram, 1 g + thiophanate-methyl, 0.5g	0.1 0.1
Control		10.5

PATHOGENIC VARIATION AND SOURCES OF RESISTANCE FOR AFRICA

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Ascochyta blight (*Phoma exigua* var. *diversispora*) of common bean (*Phaseolus vulgaris*) occurs in many areas of Latin America, the United States, Europe and Africa. Although the disease has been reported as being economically important, there are very few references in the literature on resistance in bean to ascochyta blight. Indeed, the majority of reports classify *P. vulgaris* generally as being susceptible with little genotypic variation in disease reaction.

The International Center for Tropical Agriculture (CIAT) in Colombia has identified sources of resistance in common bean to ascochyta blight in germplasm accessions as well as breeding lines. The level of resistance encountered, however, is low to moderate, with higher levels of resistance generally found among climbing bean than among bush bean types (Table 13).

Table 13. Percentages of bush and climbing bean types resistant, intermediate and susceptible to ascochyta blight at Popayan.

Growth habit	No. of lines evaluated	Resistant		Intermediate		Susceptible	
		55	96	55	96	55	96
Bush	62	3.3	0	88.7	14.5	8.0	85.5
Climbing	51	19.6	53.0	68.7	31.3	9.7	15.7

Resistance is expressed as reduced levels of disease (or percentage area infected) on bean leaves, stems and pods. Because disease incidence and severity generally increase over time several disease ratings are needed during the growing season. At CIAT, the first rating (on a 1-9 scale, where 1 is no disease and 9 is severe disease) is at late flowering (V4), then at pod fill (R6) and, finally at pod maturation (R8), when pod infection is also scored.

In 1986, an International Ascochyta Blight Nursery (IBABN) was distributed from CIAT to national programs for evaluation. The nursery consists of 12 bush bean lines and 14 climbing bean lines, representing the best sources of resistance available at that time. Susceptible checks are also included. The purpose of this nursery is to provide resistant germplasm to national programs and to monitor pathogen variation in different bean growing areas of Latin America and Africa. Results of nurseries grown in Rwanda and Colombia indicate that pathogen variability may be minimal in these two areas (Tables 14 and 15) and that screening for resistance to ascochyta blight in Popayan, Colombia may identify bean genotypes suitable for Rwanda.

Table 14. Ascochyta blight reactions of bush bean entries in International Bean Ascochyta Blight Nursery (IBABN) at Popayan and in Rwanda.

Identification	Popayan 86B	Rwanda 86B	Rating
Local check	7.0	5.0	S
BAT 477	6.5	5.2	S
CATU	5.3	3.8	I
BAN 6	5.3	3.7	I
EMP 117	5.0	4.7	I
BAT 1225	3.5	4.2	R
BAT 1569	3.3	3.0	R
G 4603	3.3	2.7	R
PAI 119	4.3	2.9	R
G 17098	3.3	3.2	R
BAT 795	4.2	3.0	R
BAT 1416	3.3	3.8	R
A 182	4.2	3.4	R

Average of three rating periods on a 1-9 scale, where 1 = immune and 9 = very susceptible

High levels of resistance to ascochyta blight have been identified in *P. coccineus* subsp. *polyanthus* and *coccineus* (Table 16). A collaborative project between CIAT and the University of Gembloux, Belgium is in progress to transfer the resistance in *P. coccineus* to *P. vulgaris* through hybridization.

This project is of a long term nature due to the difficulties involved in working with interspecific hybrids, although several advanced line materials with high levels of ascochyta resistance have entered CIAT's VEF testing scheme. These materials, however, still have problems with long maturation times and many continue segregating even past the F_8 generation.

Table 15. Ascochyta blight reactions of climbing bean entries in International Bean Ascochyta Blight Nursery (IBABN) at Popayan and in Rwanda.

Identification	Popayan 86B	Rwanda 86B	Rating
AND 244	8.0	4.7	S
VRA 81058	5.3	4.1	I
ZAV 91	3.8	3.4	R
G 12582	2.8	2.8	R
ASC 6	4.7	3.5	R
ASC 1	3.8	2.8	R
G 10747	3.1	3.3	R
ZAV 21	4.3	4.0	R
VRA 81051	3.5	2.4	R
AFR 223	5.5	3.5	R
VRA 81018	4.7	3.4	R
G 35182(*coccineus*)	1.6	1.8	R
ASC 4	3.6	3.3	R
G 12307	4.5	3.1	R

Average of three rating periods on a 1-9 scale, where 1 = immune and 9 = very susceptible

Table 16. *Phaseolus coccineus* accessions with high levels of resistance to ascochyta blight, 1986B.

CIAT no.	Origin	Subspecies[1]
G 35182 (Guate 1076)	Guatemala	*polyanthus*
G 35357	Colombia	*coccineus*
G 35358	Colombia	*coccineus*
G 35361	Colombia	*coccineus*
G 35421	Mexico	*coccineus*
G 35429	Mexico	*coccineus*
G 35430	Mexico	*coccineus*

[1] All 33 *P. coccineus* subsp. *polyanthus* accessions evaluated showed resistance to ascochyta blight, whereas *P. coccineus subs. coccineus* accessions were more variable and many accessions were susceptible.

Inheritance studies for resistance to ascochyta blight are being implemented in Colombia and Rwanda. Although these studies are just beginning, it appears that resistance is heritable and can be selected for in early generations (Table 17). It is not yet known whether resistance is governed by a few or many genes.

Table 17. F_2 and F_3 generations of crosses to combine sources of resistance to ascochyta blight at Popayan in 1987A.

F_2 code	Cross	No. of F_2 selections	No. of F_3 families
YG 77	VRA 81018 x G 2333	1	1
YG 79	VRA 81018 x Urubonobono	1	0
YG 80	VRA 81018 x G 11060	10	4
YG 81	VRA 81018 x Gisenyi 6	4	1
YG 83	VRA 81059 x G 13671	12	4
YG 84	VRA 81059 x G 2333	5	1
YG 85	VRA 81059 x Urubonobono	4	0
YG 87	V 80010 x G 2333	10	1
YG 89	V 80010 x Urubonobono	6	0
YG 90	V 80010 x G 11060	8	1
YG 91	V 80010 x C 10	2	0
YG 92	BAT 1222 x BAT 1486	15	6
YG 93	BAT 1222 x BAT 1251	15	8
YG 95	V 80010 x VRA 81018	5	3
YG 96	V 80010 x VRA 81059	6	1
YG 97	BAT 1486 x BAT 1225	15	4
YG 98	BAT 1486 x BAT 1251	14	11
YG 105	BAN 6 x BAT 1222	10	5
YG 106	BAN 6 x BAT 1225	14	12
YG 107	BAN 6 x BAT 1486	15	13
YG 108	PVA 1408 x Rubona 5	20	6
YG 109	PVA 1408 x Tostado	20	15
YG 110	PVA 1408 x BAT 1251	11	5
YG 117	BAT 1222 x PVA 1408	11	1
YG 113	BAN 6 x VRA 81059	9	4
YG 114	BAN 6 x VRA 81018	8	4

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SECTION 5: COMMON BACTERIAL BLIGHT
(*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye)

DISTRIBUTION, PREVALENCE AND ECONOMIC IMPORTANCE

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INTRODUCTION

Bacterial blights of common bean are caused by four different organisms (Atanasoff and Kovachevski, 1929; Njuguna *et al.*, 1981; Allen, 1983; Anon., 1983). These are:

Xanthomonas campestris pv. *phaseoli*, which causes common bacterial blight;

Xanthomonas campestris pv. *phaseoli* (var. *fuscans*), causing fuscous blight;

Xanthomonas campestris pv. *vignicola*; and

Pseudomonas syringae pv. *phaseolicola*, causing halo blight.

This paper is concerned with common and fuscous blights and, for the present purpose, they are treated as one disease (bacterial blight) since they frequently occur together and the field symptoms are similar (Yoshii *et al.*, 1976).

DISTRIBUTION

Bacterial blight is widespread, reported in almost all areas where bean is grown (Hayward and Waterston, 1965).

Table 18 summarizes the world distribution of CBB. The disease has been reported from Asia (Reinking, 1919; Ideta, 1936), Australia and New Zealand (Magee, 1930), Canada (Suit, 1934; Wallen and Jackson, 1975), Europe (Delacroix, 1901; Jorstad, 1922; Atanasoff and Kovachevski, 1929; Kern, 1929; Kovachevsky, 1929; Galachyan, 1936; Alfaro and Silvan, 1940; Tesic, 1946; Bremer *et al.*, 1947; Singh, 1984) and the Americas (Burkholder, 1917; Burkholder, 1930; Suit, 1934; Andersen, 1951; de Bertelli, 1947; Pinto de Torres, 1968; Schieber, 1970; Wallen and Jackson, 1975; Crispin and Campos, 1976).

In Africa, today the disease occurs in Algeria (Singh, 1984), Burundi (Perreaux and Minimbazi, 1986), Central African Republic (Anon. 1975), Egypt, Ethiopia (Anon. 1982, 1983; Habtu Assefa, 1983), Kenya (McDonald, 1936; Riley, 1960; Robinson, 1960; Hubbeling, 1973; Limo and Schumann, 1973; Kaiser, 1976; Anon. 1980; Mukunya *et al.*, 1981; Njuguna *et al.*, 1981; van Rheenen *et al.*, 1981;), Madagascar (Bouriquet, 1934), Malawi (Edje *et al.*, 1973; Msuku and Edje 1982; Peregrine, 1972), Morocco (Singh, 1984), Mozambique (Anon. 1975), Nigeria (Anon. 1975), Somali (Anon. 1975), South

African Republic (Doidge, 1919; Boelema, 1967; Melis, 1985), Sudan (Sabet, 1959; Sabet, 1960), Swaziland (Lim, 1984), Rwanda (Durnez, 1983), Tanzania (Walker, 1952; Clinton, 1961; Patel, 1975; Ebbels and Allen, 1979), Uganda (Leakey, 1963; Leakey and Mukasa, 1963a, 1963b; Leakey, 1973; Rubaihayo *et al.*, 1981), Zambia (Naik *et al.*, 1981; Roose, 1984) and Zimbabwe (Whiteside, 1955; Mariga, 1984; Whingwiri, 1984; Venge, 1986).

Table 18. World distribution of *X. campestris* pv. *phaseoli*.

AFRICA	**EUROPE**
Central African Republic	Bulgaria
Egypt	Czechoslovakia
Ethiopia	France
Kenya	Germany
Madagascar	Hungary
Malawi	Norway
Mozambique	Poland
Nigeria	Rumania
Zimbabwe	Spain
Somalia	Sweden
South Africa	Switzerland
Sudan	U.S.S.R.
Tanzania	Yugoslavia
Uganda	
Zambia	**NORTH AMERICA**
ASIA	Bermuda
	Canada
Cambodia	Mexico
Ceylon	U.S.A
China	
India	**CENTRAL AMERICA AND WEST INDIES**
Indonesia	
Israel	Costa Rica
Japan	Dominican Republic
Lebanon	Guatemala
Phillipines	Honduras
Taiwan	Jamaica
Turkey	Nicaragua
	Panama
SOUTH AMERICA	Salvador
Argentina	**AUSTRALIA & OCEANIA**
Brazil	
Chile	Australia
Colombia	Hawaii
Venezuela	New Zealand

PREVALENCE AND ECONOMIC IMPORTANCE

While bacterial blight may occur wherever bean is grown, its prevalence varies within countries and between seasons depending somewhat on weather conditions (Zaumeyer and Thomas, 1957). In most countries, it is most prevalent in areas with moderate to high day temperatures (Allen, 1983; Mariga, 1984). In Kenya, for example, bacterial blight is more prevalent in Central and Eastern Provinces than in Nyanza, Rift Valley and Western Provinces (Mukunya *et al.*, 1981). In Uganda, the disease is prevalent in all bean growing areas (Sengooba, 1985). Prevalence in Uganda, however, varies from season to season. In the United States, bacterial blight is prevalent in states east of the Rocky mountains (Zaumeyer and Thomas, 1957), compared to California, Idaho, Nevada, Washington and Oregon, where it rarely causes enough loss to be of economic importance.

Bacterial blight is regarded as a major disease or a major production problem in most African countries where it occurs (Riley, 1960; Leakey, 1963; Mukasa and Leakey, 1965; Anon. 1970; Edje *et al.*, 1973; Mulindwa, 1974; Patel, 1975; Kaiser, 1976; Muthangya, 1980; Naik *et al.*, 1981; Mariga, 1984;). In most African countries, however, the actual yield losses caused by the disease are not given. In some cases, only incidence and severity are reported (Mukunya *et al.*, 1981; Habtu Assefa, 1982; Sengooba, 1985). In Kenya, the incidence of bacterial blight is high in the Rift Valley and Eastern Provinces and of low incidence in Western Nyanza and Rift Valley Provinces (Mukunya *et al.*, 1981). In Uganda, although the exact losses caused by bacterial blight have not been estimated, in 1984, the disease was so serious on bean that the crop which had been grown for seed (for farmers) by the seed project (which is the main seed multiplication scheme in the country) could not be used for seed during that year. Since then, the project has not been able to multiply bean seed for farmers because of the disease. They are still waiting for breeders to provide a cultivar which is resistant/tolerant to bacterial blight.

Outside of Africa, yield losses caused by this disease were estimated as far back as the early 20th century. In the United States, losses ranged from 40 to 60% of the crop in Colorado and 50% in Oklahoma in 1919. Calculated on the basis of weight, the losses were more than 3.4 million lb of snap beans and 34.7 million lb of dry beans (Zaumeyer and Thomas, 1957).

Andersen (1951) estimated that bacterial blights caused a 3.5 million US dollar loss to growers in three Michigan counties in 1951. In 1953, the disease was widespread in western Nebraska and the loss caused was estimated at a little more than one million US dollars.

In Canada, Wallen and Jackson (1975) reported a 38% yield loss in Ontario due to common and fuscous blights in two years field trials. Aerial infra-red photographic surveys suggested that losses for the bean crop grown in Ontario ranged from 1252 t in 1970 to 218 t in 1972 (Jackson and Wallen, 1975; Wallen and Jackson, 1975). In Colombia, yield losses estimated at 22% and 45% have been obtained by natural and artificial infections respectively (Yoshii *et al.*, 1976). Field surveys estimated losses of 13% due to common and fuscous blight while, in the Cauca Valley alone, common bacterial blight is responsible for 17% crop loss (Pinstrup-Andersen *et al.*, 1976).

SURVIVAL AND SPREAD

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The discontinuity of crops does not generally provide suitable conditions for phytopathogenic agents to survive between seasons. Their survival depends on their capacity to escape from or endure less favourable environmental conditions. In this paper, we will discuss the means of survival of common bacterial blight of bean, *Xanthomonas campestris* pv. *phaseoli* (E.F. Smith) Dowson, which is frequently found associated with *X. c.* pv. *phaseoli* var. *fuscans* (Burk.) Starr and Burk. In Burundi, common bacterial blight is found in all bean growing areas but assumes damaging proportions only in low altitude areas (e.g. the plain of Imbo, about 800 masl), where average temperatures (25-28°C) are more favourable for its development. This bibliographic review is inspired mainly by the work of Schwartz and Galvez (1980) and the article by Schuster and Coyne (1974).

SURVIVAL

Phytopathogenic bacteria do not form spores or other special survival structures as do fungi and nematodes. During unfavourable periods they are in association with animate or inanimate agents, such as seeds, crop residues (straw), plants (including epiphytic survival associations), insects, soil and other non-host material (Schuster and Coyne, 1974). The same authors suggest that survival in the tropics is longer than in temperate climates due to the continual increase in the bacterial population at temperatures which are never limiting and epiphytic survival on perennial plant hosts.

Seeds

Seed is the most effective means of survival of phytopathogenic bacteria. The transmission of *X. c.* pv. *phaseoli* by seed has been known since 1872 (Yoshii, 1980). Viable and virulent bacteria have been isolated from bean seeds 3, 10 and 15 years old. The pathogen is short-lived in the superficial layer of seeds, but survives longer if it has invaded the seed deeply as far as the embryo. Penetration sites are natural openings such as the hilum, the micropyle or fresh wounds. Survival depends on the temperature at which the harvest is stored. Between 18 and 32°C, bacteria can survive for up to two years and up to seven years at less than 18°C (Wallen and Galway, 1979).

Plant residues (straw)

Even if the seed used is free from bacteria, common bacterial blight occurs in places where crops are not rotated (Schuster, 1970). Several authors point out that crop residues constitute a primary source of disease inoculum (Zaumeyer and Thomas, 1957; Schuster and Harris, 1957; Kranz *et al.*, 1977). It seems, however, that this depends on the climatic conditions of the period between seasons. Research carried out in Michigan (USA) has shown that

bean *Xanthomonas* cannot survive the winter (Saettler *et al.*, 1986). Finding that even in badly attacked bean plots the pathogen does not survive from one season to another, Wimalajeewa and Nancarrow (1980) conclude that infected residues do not constitute a primary source of inoculum in East Gippsland in Australia. The same authors show that length of survival is inversely proportional to the depth to which the residues are incorporated, being longer if the residues are on the surface or in the upper layers of the soil (less than 20 cm depth). On infected leaves or pods, the bacterium can survive 11 weeks on the surface and three weeks if incorporated in the soil.

There is no precise information available on the importance of crop residues as a primary source of inoculum in Central Africa. We can, however, speculate that the short dry season (January-February) separating the two cultural seasons is *a priori* favourable to the survival of the bacterium, as long as it is sufficiently pronounced, which is generally the case in warmer regions where the disease is most rife. If this season is less pronounced, rapid decomposition of the residues markedly reduces the inoculum potential of *X. c.* pv. *phaseoli*. It should also be noted that the harvesting of whole plants, as commonly practised by most farmers, probably constitutes an effective crop sanitation measure.

Epiphytic survival

Epiphytic flora are organisms living on healthy, aerial parts of the plant and not affecting its life (Leben, 1965). A relatively short survival period of pv. *phaseoli* has been observed on apparently healthy bean plants (Thomas and Graham, 1952), but the number of bacteria can increase on leaves not showing disease symptoms (Weller and Saettler, 1987). Haas (1972) found that, in conditions of artificial infection, *X. c.* pv. *phaseoli* var. *fuscans* survived on primary bean leaves but disappeared rapidly from trifoliolate leaves. It can also colonize roots but disappears after two weeks (Stanek and Lasik, 1965). Root exudates inhibit the pathogen whilst those of the seeds stimulate its growth.

Insects

X. c. pv. *phaseoli* can survive inside the bodies of insects (digestive tract and faeces) (Kaiser and Vakili, 1978) belonging to various species. These include:

- *Cerotoma ruficornis* Oliv., Chrysomelidae, Coleoptera
- *Diaprepes abbreviata* L., Curculionidae, Coleoptera
- *Chalcodermus ebeninus* Boheman., Curculionidae, Coleoptera
- *Nezara viridula* L., Pentatomidae, Hemiptera
- *Empoasca* spp., Cicadellidae, Hemiptera

Some isolates of *X. c.* pv. *phaseoli* have been able to survive up to 19 days in the bodies of living insects and others up to 14 days in dead insects.

Secretion of polysaccharides

The resistance to desiccation of *X. c.* pv. *phaseoli* is assured by the secretion of an extracellular polysaccharide in culture and in the host plant, which enables prolonged survival of 1,325 days in very varied conditions (Wilson *et al.*, 1965).

Soil

X. c. pv. *phaseoli* is short-lived when free in the soil because of its poor saprophytic competitivity. Sutton and Wallen (1970) were unable to isolate it from soil where infected beans were growing.

Other host plants

X. c. pv. *phaseoli* var. *fuscans* can survive on various host plants such as other *Phaseolus* spp., *Pisum sativum*, *Glycine max*, *Vigna unguiculata*, *Lycopersicum esculentum*, *Lupinus polyphyllus* and *Dolichos lablab* (Kranz et al., 1977). The importance of these secondary hosts in the epidemiology of the common bacterial blight in central Africa is not known.

DISPERSAL

The different modes of survival of bacteria constitute the primary inoculum and the source of contamination of other plants. With movement, they ensure the dispersal and spread of the disease.

The seed constitutes the principal element in disease propagation. It allows the spread of the disease among regions and countries and even continents through trade, exchange or national or international aid. Epidemiological studies carried out in Canada showed that 0.5% of infected seeds suffice to give rise to devastating epidemics (Wallen and Sutton, 1965).

Rain ensures the dispersal of the disease among plants and fields through splash and water-flow. The same is true of irrigation water (Steadman *et al.*, 1975).

Wind, carrying infected residues or contaminated soil (Claflin *et al.*, 1973), is also a dispersing factor for the disease. Aerial dispersal seems theoretically possible but has not yet been mentioned for *X. c.* pv. *phaseoli* (Yoshii, 1980).

Insects feeding on contaminated plants could constitute a secondary dispersal factor (Kaiser and Vakili, 1978).

CONCLUSIONS

It will be noted firstly, that knowledge of the epidemiology of common bacterial blight in Central Africa and, particularly of sources of inoculum, is minimal. The scarcity of means and the diversity of problems make it

necessary, however, to define certain priority directions for research or action, which this bibliographical review helps to indicate.

The fundamental importance of the seed as a source of primary inoculum reinforces the relevance of trials aiming for the production of healthy seed on farm. This factor also challenges institutes or organizations involved in the production, multiplication or diffusion of improved cultivars to be aware of the necessity of conforming to the strictest sanitary standards for the seeds produced.

In the context of the development of leguminous crops as feed, green manure or cover plants as envisaged at present, the possible rôle of infection relay or of increasing the inoculum potential deserves further investigation.

PATHOGENIC VARIATION

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Yoshii (1980) and Allen (1983) have reviewed the literature concerning the variability in pathogenicity of *Xanthomonas campestris* pv. *phaseoli* and the sources of resistance to common bacterial blight. They draw the following conclusions:

- variation in pathogenicity occurs among isolates of *X. c.* pv. *phaseoli*. This variation is mainly quantitative and differential interactions between host and isolate are not clearly exhibited. Yoshii (1980) notes that such variation can be complicated by differences in infection method, isolate age and other factors;

- variants of the *fuscans* type are generally more pathogenic, but do not have special taxonomic status (Bradbury, 1986);

- various sources of resistance have been identified, generally partial (often called tolerance). The highest level of resistance has been found in *Phaseolus acutifolius* and partially transferred to *P. vulgaris*. This resistance is quantitative. The level of resistance of a cultivar can vary according to its age, frequently diminishing at flowering. The leaves and pods do not necessarily show the same level of resistance. The adaptation of a cultivar to environmental conditions also affects the expression of resistance: certain cultivars which are resistant in temperate climates are susceptible in tropical regions, unrelated to differences in pathogenicity of the bacterial isolates involved.

Even if it is clear that differences in pathogenicity exist among isolates of *X. c.* pv. *phaseoli*, as do different levels of resistance among bean cultivars, these characteristics are poorly defined in the literature and it is surprising how little effort is made to characterize host x parasite interactions. Table 19 illustrates our thesis by presenting evaluation scales used by various authors to characterize the results of

infection, most often artificial, in fields or in controlled conditions.

Table 19. Evaluation scales used to characterize the reactions to *Xanthomonas campestris* on bean leaves or the pathogenicity of the bacterium.

A. Coyne and Schuster (1970)

T: high tolerance, occasional small lesions on leaves
T-: moderately high tolerance, some light lesions developed on some leaves.
S: numerous lesions spread over most of the leaves.

B. Coyne and Schuster (1974)

Classification (without grades): highly, moderately or slightly susceptible, tolerant.

C. Ekpo and Saettler (1976)

1: tolerant, some necrotic spots
2: slightly susceptible, less than 20% necrosis on infected leaves
3: moderately susceptible, 20 to 50 % necrosis on infected leaves
4: very susceptible, more than 50% necrosis on infected leaves

D. Yoshii *et al.* (1978)

1: very tolerant; no symptoms, the bacteria can be reisolated from leaves
2: tolerant; small, light lesions on 1 to 5% of leaves
3: moderately susceptible; moderate number of lesions of variable size, some chlorotic leaves
4: susceptible; numerous severe lesions, spread over most of the leaves, pronounced chloroses and necroses
5: very susceptible, very severe infection, plants chlorotic, necrotic and extensively defoliated.

E. Schuster *et al.* (1983)

1: no symptoms in infected zone
2: 1-25% necrosis in infected zone
3: 26-50% necrosis in infected zone
4: 51-75% necrosis in infected zone
5: 76-100% necrosis in infected zone

F. Schuster (1983)

1: no infection
2-4: not defined
5: very severe infection or death

G. Zapata *et al.* (1985)

1: resistant
2: slightly susceptible
3: moderately susceptible
4: susceptible
5: very susceptible

Some of these scales are essentially qualitative (B, G), fixing different classes of resistance without defining the symptoms they represent. Others are quantitative, based on the extent of lesions (C, E). However, the authors interpret their results on the basis of indices calculated to the

first decimal place, whose biological significance is questionable as indices do not necessarily correspond with areas of necrosis. The scale better defines possible different types of reaction but how does one quantify small lesions on 90% of the leaves or large lesions on a few leaves?

The scales presented certainly allow a classification of the cultivars or the isolates analysed, but their non-standardization complicates the comparison of results obtained in different places or times. They do not allow a precise approach to the diversity of reaction of cultivars, nor a better comprehension of the mechanisms of resistance (e.g. type, size and number of lesions). Little information is available on the repeatability of results obtained in different experimental conditions or on the homogeneity of reactions within a particular cultivar and hence the biological significance of the indices used in classification.

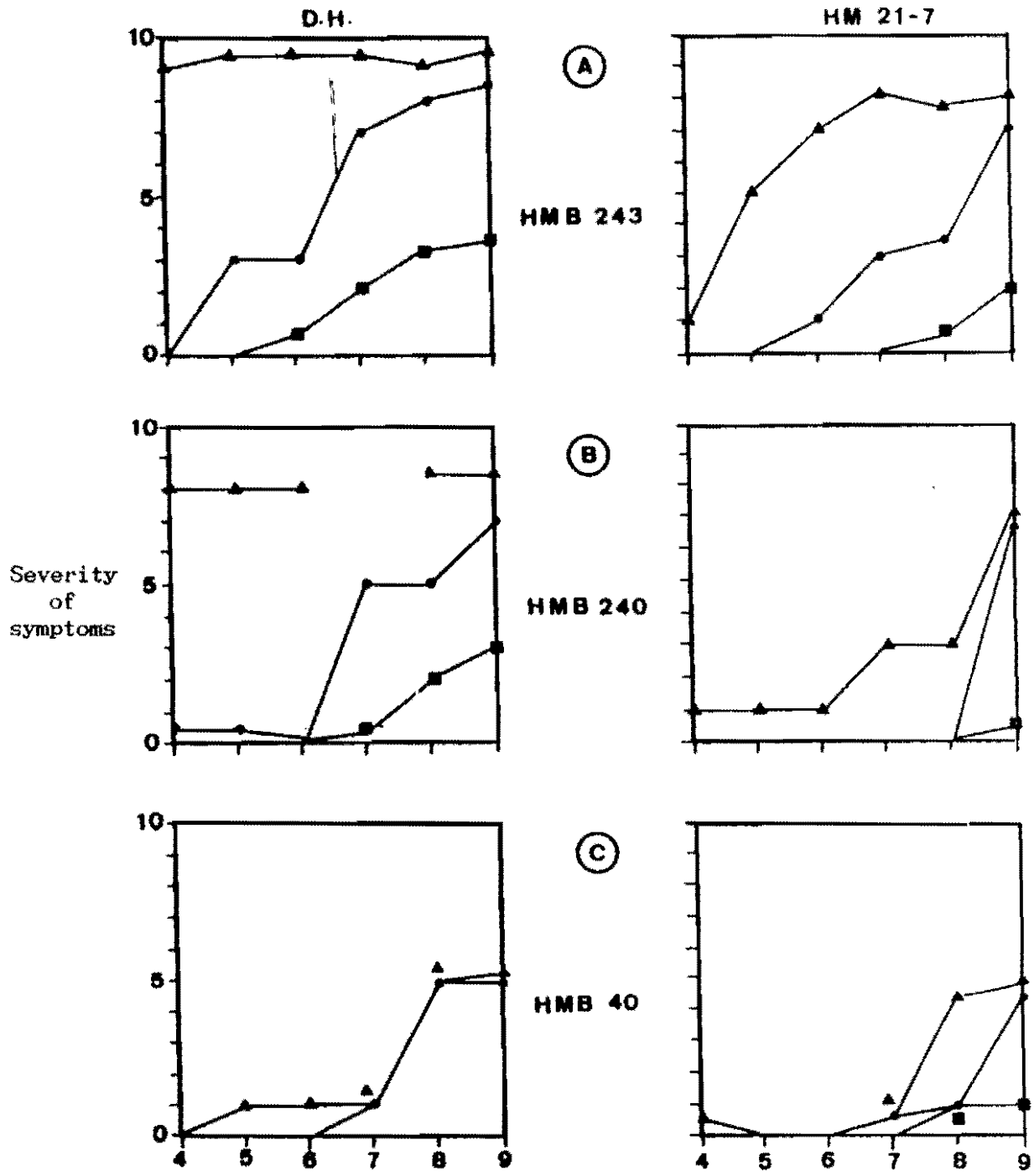
The Phytopathology Laboratories of the Catholic University of Louvain (Belgium) and the Institut des Sciences Agronomiques du Burundi have begun a series of parallel studies to develop tools to better characterize the resistance of bean cultivars or the pathogenicity of the bacteria. The results presented below are still very preliminary and fragmentary but can be considered as a basis of reflection and discussion of the orientation of future work. The methodology followed was inspired particularly by the principles of titration of phytopathogenic bacteria (Ercolani, 1984).

In one experiment, the cultivars Double de Hollande and HM21-7 were infected with increasing concentrations (10^4 to 10^9 cells/ml) of bacterial strains HMB 240 (*X. c.* pv. *phaseoli* var. *fuscans*), HMB 243 (*X. c.* pv. *phaseoli* and HMB 40 (*X. c.* pv. *vignicola*). The infection was carried out by infiltration of the bacterial suspension into the first trifoliolate leaf, one infiltration site (1 cm x 0.5 cm) per half foliole, on plants 4 weeks old. The scale used is intended to describe precisely the development of symptoms at the point of infection:

0: necrosis only of the injection point
1: yellowing of the infiltration site (i.s.)
1.5: yellowing and beginning of aqueous appearance
2: aqueous appearance of the i.s.
3: necrosis of area less than 50% of the i.s.
4.5: complete necrosis of the i.s.
5: beginning of yellowing around the necrosis (max. 2 mm)
6: extension of yellowing
7: oily appearance at the border of the necrosis
7.5: extension of symptoms by about 1 mm around the necrosis
8: extension with diameter less than 1 x that of i.s.
9: extension with diameter more than 1 x that of i.s.
10: tendency towards generalized necrosis, yellowing

Although the two cultivars were barely distinguishable 20 days after infection with the strain HMB 243 at concentrations above 10^7 cells/ml, they were clearly differentiated at lower concentrations of inoculum (Figure 1A) and by the development of symptoms over time, suggesting a host x parasite interaction. The results of infection by the strain HMB 240, which is clearly less pathogenic, confirm this difference (Figure 1B). Only the high concentrations of pv. *vignicola* induce limited necrosis of the infiltration site, more rapidly with the cultivar, Double de Hollande.

Figure 1. Severity of symptoms developed on the cultivars Double de Holland (DH) and HM-21-7 inoculated by foliar infiltration of different concentrations of *Xanthomonas campestris* pv. *phaseoli* (HMB 243), pv. *phaseoli* var. fuscans (HMB 240) and pv. *vignicola* (HMB 40), 6, 12 and 20 days after inoculation.



In addition, the scale described gives good discrimination among a set of cultivars (Figure 2). These results, involving certain sources of resistance provided by CIAT - G 40016, G 40034 (*P. acutifolius*), X112 (*P. vulgaris*), X159 (*P. vulgaris* x *P. acutifolius*) - were obtained 20 days after infiltration with a bacterial suspension of 10^7 cells/ml. More precise information could probably be obtained with the use of a wider range of concentrations.

Figure 3 illustrates the development of symptoms on the cultivar Karama 1/2 (considered susceptible) 7 days after infection by infiltration of a range of bacterial concentrations ranging from 10^2 to 10^9 cells/ml. The symptoms were evaluated according to the following scale, very similar in concept to the preceding one:

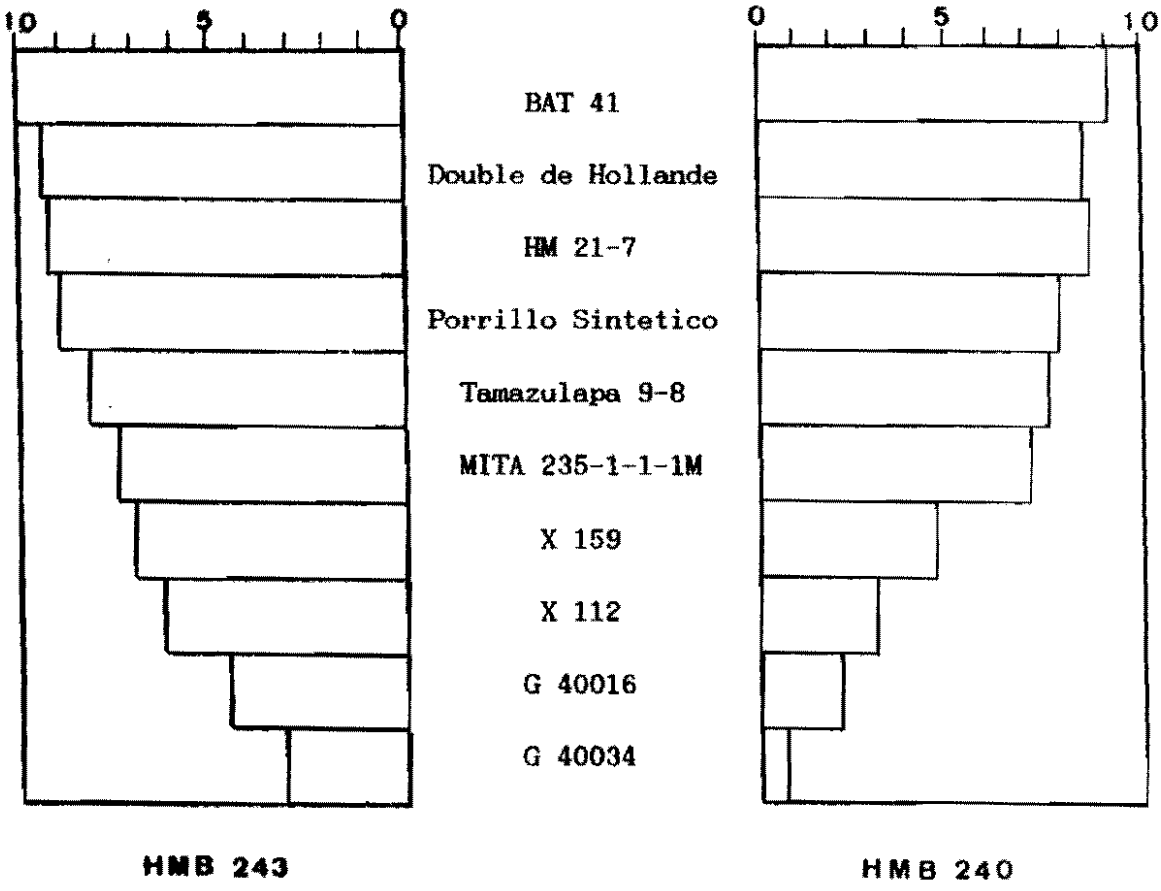
0: no symptoms
1: slightly necrotic zone, diffuse in the infiltration site (i.s.), without apparent necrosis
2: marked chlorotic zone in the i.s., with indefinite border and necrotic point in centre
3: marked chlorosis of all the i.s., clear border and necrotic centre
4: aqueous necrotic lesion, smaller than the i.s., without chlorotic border
5: like 4, but with narrow yellowish border less than 2 mm wide
6: small aqueous necrotic lesions, spread over all the i.s., with yellowish border wider than 2 mm
7: necrotic lesion equal to or bigger than i.s., limited yellowish border
8: like 7, but with wide border which may extend to the edge of the foliole
9: extensive necrosis/infected leaf falls

There is a distinct linear relationship between the log of the bacterial concentration and the severity of the symptoms, indicating some biological significance of the evaluation scale. This type of relation could serve as an "internal standard" in screening trials of cultivars, where the slopes and intercepts of the regression lines would allow a relative characterization of the reactions of the cultivars tested. A standard cultivar could be used to characterize the test conditions and permit comparisons over time and space.

Although the indices used in Figure 3 are statistically well defined, the dispersion of values for concentrations 10^5 and to some extent 10^7 (Table 20) still poses the problem of their biological significance. The origin of this dispersion (experimental or biological variability) requires further study.

Figure 4 illustrates a first application, still very qualitative, of the method. Three cultivars evaluated as resistant in the field in the Pepinière Régionale d'Evaluation des Lignées Avancées en Afrique Centrale (PRELAAC) (Ntahimpera and Perreaux, 1987) were compared with four cultivars classed intermediate to susceptible, with three concentrations of inoculum. The development of symptoms in the two extreme cultivars (Araona and RWR 96) are very distinct. The others are less clearly distinguishable, except at the inoculum concentration of 10^3 c.b./ml after 13 days. The reasons for the poor development of symptoms on the cultivars Kiburu Moshi and Karama 1/2 at 10^5 c.b./ml are not known.

Figure 2. Severity of symptoms developed by ten cultivars of *Phaseolus* spp. inoculated by foliar infiltration of isolates HMB 243 (*Xanthomonas campestris* pv. *phaseoli* and HMB 240 (pv. *phaseoli* var. *fuscans*) at a concentration of 10^7 cells/ml (20 days after inoculation).



MITA 235-1-1-1M: *P. vulgaris* x *P. acutifolius*
X 159: *P. vulgaris* x *P. coccineus*
G 40034, G 40016: *P. acutifolius*
Others: *P. vulgaris*

Figure 3. Mean severity of symptoms developed after seven days on the cultivar Karama 1/2 inoculated by foliar infiltration of different concentrations of isolate E162 of *Xanthomonas campestris* pv. *phaseoli* var. *fuscans* (means ± confidence interval, P = 0.05).

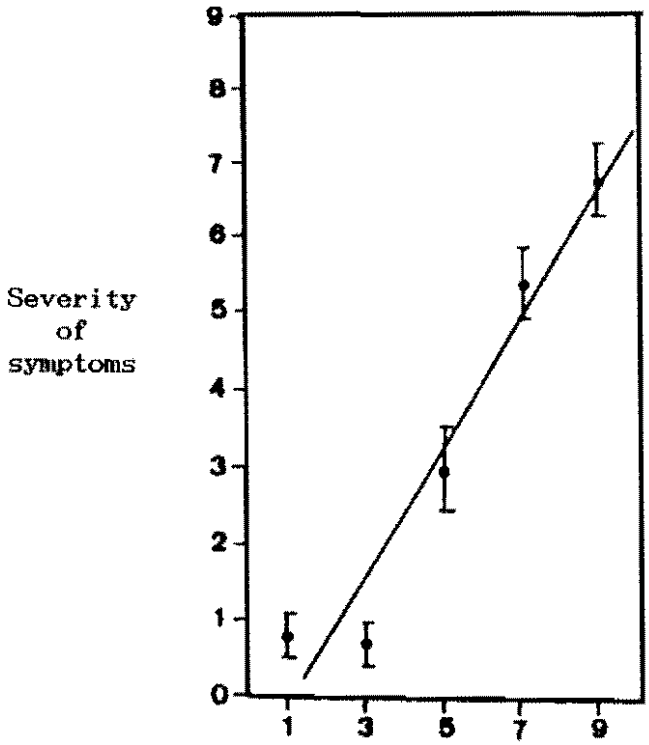
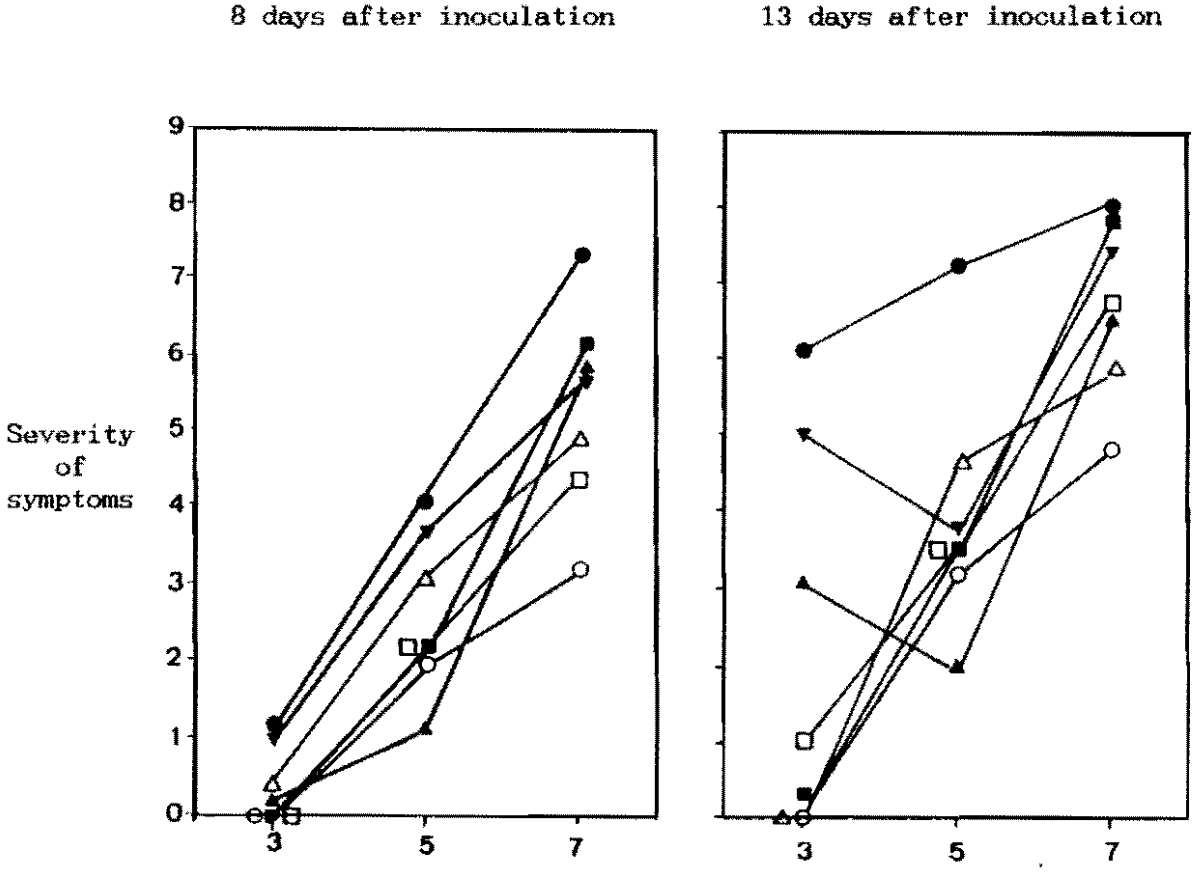


Figure 4. Severity of symptoms developed 8 and 13 days after inoculation on seven bean cultivars inoculated by foliar infiltration of different concentrations of *Xanthomonas campestris* pv. *phaseoli*.



Aroana (6); Baseka (6); Karama 1/2 (6); Kiburu moshi (5); RWR 96 (3); PVA 1435 (3); PVA 880 (4)

Table 20. Relative frequency (%) of symptom severity values on the cultivar Karama 1/2, 7 days after infection by foliar infiltration of different concentrations of strain E 162 of *Xanthomonas campestris* pv. *phaseoli*.

	Concentration of inoculum (c.b./ml)					
Score	0	10	10^3	10^5	10^7	10^9
0	100	42	67	2		
1		42	5	24		
2		17	24	19		
3			5	29	21	3
4				2	2	
5				2	29	
6				21	21	28
7					19	44
8					7	25
9						
No. of i.s.	30	36	42	42	42	32

Although the methods can be further refined, the trials reported here offer interesting perspectives for the development of methodologies capable of better characterizing the reactions of bean cultivars to common bacterial blight. They aim to define a more standardized methodology, with which the influence of the experimental conditions can also be taken into account.

For more rapid and certain progress, it is essential to base this type of research on more fundamental knowledge of the pathogenesis of the disease and to more accurately link macroscopic symptoms with host x pathogen interactions at cellular or tissue level.

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ESTABLISHMENT OF FIELD AND SCREENHOUSE EPIDEMICS AND RATING FOR RESISTANCE

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Disease is the interaction of the pathogen, host and environment in a given period of time and all components must be considered in establishing field and screenhouse epidemics.

FIELD NURSERIES

Factors to keep in mind.

1. Locate target areas or hot spots that are representative of bean producing areas.

2. Sowing dates should correspond to those of farmers' planting to evaluate predominant diseases.

3. Proper nursery designs should be used to avoid field variation. Soil fertility should be adequate but not excessive.

4. Use appropriate checks - importance cannot be underestimated in disease nurseries. Uses: measure uniformity and level of disease throughout the field.

5. Adequate disease pressure can be achieved through spreaders (mixtures of susceptible varieties), which can be inoculated with mixtures of isolates or races. They are usually sown earlier than the test lines. Nurseries can also be inoculated by spreading infected leaves within trial rows or with spore suspensions from cultured isolates.

6. Evaluations of disease should be conducted 2-3 times during the growing season.

SCREENHOUSE EVALUATION

When appropriate field nurseries are available, it is preferable to use them. However, in some cases, greenhouse evaluations are needed, especially in some breeding programs, such as backcross breeding.

1. Like field studies, checks are essential.

2. For genetic studies, individual isolates are normally used. At CIAT, mixtures of isolates are used in general screenings to help identify broad sources of resistance.

3. Seedlings are usually inoculated but different resistance mechanisms may be distinguished by using plant age at time of inoculation.

EXPERIMENTAL DESIGNS FOR DISEASE NURSERIES

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INTRODUCTION

In all biological research, effective experimentation depends to some degree on the proper management (control and estimation) of the variability arising from environmental features other than the treatments imposed. This is particularly true where yield and other agronomic features are of interest as their expression is usually much affected by environmental variation. In disease nurseries, the treatments are the genotypes being evaluated and the crucial environmental feature is disease pressure and other environmental factors are important only insofar as they affect the level or variability of disease pressure.

Pastor-Corrales and Kornegay (above) list various measures for inducing uniform disease pressure. Here we consider some experimental designs which can be used to control and estimate the level and variability of disease pressure in field and screenhouse tests.

EXPERIMENTAL DESIGNS

If resistance is such that genotypes are either immune or fully susceptible, a uniform, optimum level of infection and a simple unreplicated and unrandomised design should be sufficient to distinguish resistant and susceptible genotypes. However, all too often, the resistance being evaluated is quantitatively inherited (this is true of ALS, AB and CBB - three out of five of the diseases considered here), so is incomplete and results in a continuous spectrum of disease reactions. Even where resistance is qualitative, there is frequently a measurable background of quantitative resistance which is worth exploitation. Furthermore, even though all the measures to produce infection are applied, uniform epidemics are never guaranteed and disease expression may be continuous and irregular. In such situations, the use of designs which help to control and measure variability may rescue nurseries which would be otherwise of little value. Although not considered, the nature of the score is important - the analyses described assume normal distribution of actual or transformed values.

Conventional designs

Where the number of test entries is not large (say not more than 100-200), replication and randomisation will provide an estimate of the random variability and tests of significance of observed differences. With smaller numbers, randomised complete block designs are adequate. In the absence of information on disease development, square blocks are likely to be most

efficient. If gradients are suspected, long narrow blocks running across the direction of the gradient will be more appropriate.

With larger numbers of test entries, blocks become too large to produce uniform disease pressure and lattice designs, which group test entries into sub-blocks within blocks are more appropriate. There are many types of lattice, from simple lattices with two replicates to balanced lattices and lattice squares, with k+1 and k+1/2 replications where k is the number of entries in each block. Cochrane and Cox (1957) give plans for lattices up to 12x12 (i.e. 144 entries) but plans for larger numbers of entries are easily constructed. Cubic lattices accomodate even larger numbers of entries, for example an 8x8x8 cubic lattice accomodates 512 entries in three blocks with sub-blocks of 8.

Nearest neighbour analysis

First proposed by Papadakis (1937) and described by Pearce (1983), adjustment according to neighbouring plots is especially useful where disease expresion is irregular. The analysis may be applied to any replicated design and may be used to adjust the disease scores of each plot according to the mean disease score of its neighbours.

The analysis proceeds as follows:

1. Compute: (a) the deviations of each plot from the mean of all plots of that treatment; (b) the mean deviations (X) of the neighbours of each plot; treatment totals and means of the Xs; analysis of covariance of actual values (Y) on X; regression (b) of Y on X.

2. Adjust Y for each plot by subtracting b(X-x).

3. Iterate steps 1 and 2 until the adjustments are the same.

4. Compute the analysis of variance of the adjusted values.

Since replication is necessary, the area required is enlarged but the method allows adjustment for patchy variation, which is not possible in an orthodox analysis of variance, and provides an estimate of error.

Regular checks

Where the number of test entries is much greater than 500, replication is obviously impractical because of the land area that would be required and the difficulty of ensuring uniform disease pressure over so large an area. The simplest means of obtaining an indication of environmental variability in an unreplicated set of test entries is the inclusion of regular checks. This is common practice in disease nurseries, in the form of two or three susceptibles and resistants to every 10-20 test entries. Such checks may be used to reduce field variation by adjusting the disease scores of the test entries according to those of the checks.

The simplest form of adjustment is to express the disease score of each test entry as a proportion of the disease score of its nearest susceptible check or checks. Spreader rows could also be used for this purpose.

Alternatively, the disease score of each test entry may be adjusted by subtracting the deviation of the mean disease score of the nearest set of susceptible checks (and spreaders) from the mean disease score of all susceptible checks and spreaders. For example, if the mean disease score for the nearest susceptible checks and spreaders is 8.5 and the overall mean score is 7.5, the score for each test entry in that sector of the nursery is adjusted downwards by 8.5-7.5=1.0 units.

The latter adjustment is preferable as the actual and adjusted data are of the same units. Both have the disadvantage of providing no estimate of error for comparison of the differences among test entries.

Augmented designs

In augmented designs (Federer, 1956), test entries are again unreplicated but are randomized and grouped in blocks of a size (say 20-50) appropriate to the number of test entries and checks and size of field. A number of different check entries (say 2-5 or about 10% of the number of test entries per block) are randomized within each block. The disease scores of the checks can then be analysed in the manner of a randomised complete block design to derive an estimate of error to compare the disease scores of the test entries.

The disease scores of the test entries can also be adjusted by the deviation of the mean score of the checks in the same block from the mean score of all checks, so that $B_j - y..$ is the adjustment for the score of each test entry in the jth block, where B_j is the mean of the checks in the jth block and $y..$ is the overall mean check score. Note that the variance of the difference of two test entries in the same block will be twice the error mean square, while that of two test entries indifferent blocks will contain an additional quantity for block differences.

The method assumes that the random components associated with the scores of the checks and test entries are similar. This is most likely to be so if the checks represent the same range of variation as the test entries. If this assumption is correct, the method provides a measure of the random variation and a means of adjusting the scores of the checks according to this variation.

One further point. It is common practice to include test entries in order of origin or some other systematic arrangement, so that similar materials are compared more precisely. But in evaluating a set of materials, we are as well interested in the relative performances of dissimilar materials, so in the absence of compelling reasons for grouping, less biased comparisons are obtained by randomizing the test entries.

SUMMARY

Various experimental designs to manage variation in disease expression in disease nurseries are described. If the number of test entries is not large, replication is desirable. Randomized complete blocks are appropriate for small numbers of test entries: where test entries are more, lattice designs are necessary. Neighbouring plots analysis is appropriate where disease expression is irregular. Augmented designs are advocated where the number of test entries is very large.

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OVERVIEW

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INTRODUCTION

In many regions of Africa, bean production systems are characterized by the use of varietal mixtures by small farmers. In order to provide these systems with appropriate disease control technologies, it is essential to understand better the nature of disease development in varietal mixtures compared to pure variety stands and to evaluate the effectiveness of various strategies.

Consequently, this review will attempt to cover general literature on:

1) disease development in mixtures and pure culture;

2) the effect on disease of adding resistant components to susceptible mixtures; and

3) the potential of non genetic methods to control disease in mixtures.

DISEASE DEVELOPMENT IN VARIETAL MIXTURES AND IN PURE STANDS

Are mixtures worthwhile?

A survey of the literature shows that mixtures yield at least as well as the mean of their components, often more so, and on some occasions outyield even the highest component (Wolfe, 1985; Wolfe and Barrett, 1980; Trenbath, 1974). It is indeed rare to find a mixture yielding less than the mean of its components. Mixtures increase the stability of yield and the security of farmers. In this context the contribution of mixtures in reducing disease development is an important factor to consider in areas where diseases limit the yield of beans.

Do varietal mixtures reduce disease incidence and, if so, by what mechanism?

In his recent review, Wolfe (1985) notes that, "host mixtures may restrict the spread of diseases considerably relative to the mean of their components, providing that the components differ in their susceptibility". This point is illustrated by Wolfe and Barrett (1981) where barley mixtures, including components with different resistances to powdery mildew, were evaluated in pure stand and in all possible 2-way and 3-way combinations. As the complexity of the mixture increased so did the size of the reduction of disease. A reduction in disease was also observed by Teri (1986) using *Phaseolus vulgaris* pure and as a varietal mixture.

Jeger (1985) considers Wolfe's principle to be the rule for most specialized and non-specialized pathogens. Compared to the mean of the pure stands, when non-specialized pathogens infect mixtures, if the spread of diseases in the susceptible component of the mixture is hindered more than the spread in the resistant component is increased, then the overall infection tends towards the resistant component. Furthermore, in situations where component varieties are susceptible to different races of a pathogen, disease restriction is likely to be more effective since each race is restrained by its non-host (Jeger, 1985).

What is the mechanism of disease restriction in varietal mixtures?

Apart from the direct effect of the resistant component on the overall level of disease, the resistant components can have an effect on the level of disease attacking the susceptible component. The degree is dependent on a number of factors. Mixtures affect outside and inside-generated inoculum differently (Wolfe, 1985).

Foreign inoculum. When the predominant form of infection arises from sources outside the field, then the best effect a mixture can have is to provide diversification. More precisely, the infection caused by an exogenous spore shower landing on a mixture equals the mean infection of the components (Wolfe, 1985). Thus, if 25% of the mixture is resistant then 75% of the crop will become infected and there will be no protective effect visible.

Internally produced inoculum. When the predominant part of the inoculum is produced within the field, then mixtures have a unique effect on reducing pathogen spread and hence, provide a degree of protection to the susceptible component. Four mechanisms are usually stated (Burdon, 1981; Burton and Chilvers, 1982):

a) Decrease in spatial density of susceptible plants

The presence of a resistant component within a mixture effectively decreases the density of the susceptible component. The reduction of the amount of susceptible tissue available reduces the maximum extent of pathogen spread. In addition, the chances of spore survival are reduced. The ideal spacing arrangement would be one in which susceptible plants do not occur as neighbours. The extent to which spatial density affects spread of disease depends on whether the pathogen dispersal has a steep or a shallow gradient, i.e. spore concentration does or does not drop off rapidly with distance away from the inoculum source (McCartney and Fitt, 1985). Intercropping different species of crops would simulate a similar effect as that provided by resistant varieties in mixtures.

b) Barrier provided by resistant plants.

Resistant varieties interfere with the passage of spores from one susceptible plant to another.

c) Replacement

The replacement of susceptible plants by resistant components effectively increases the surface area occupied by resistant plants. As a

result, the chances of a spore landing on susceptible tissue are reduced (Burdon, 1981).

d) Induced resistance

Resistance in normally susceptible plants is induced by non-pathogenic spores which ward off infection by pathogenic spores landing on the same area. The effect may be cumulative and could account for a considerable amount of disease restriction provided by mixtures (Wolfe, 1985).

In any one pathogen generation the effect of these three factors may be small. However, over several generations the cumulative effect on pathogen multiplication may be significant.

THE EFFECT ON DISEASE OF ADDING RESISTANT COMPONENTS TO SUSCEPTIBLE VARIETIES

What proportion of resistants in mixtures is required in order to reduce significantly the level of disease in the susceptible component?

From the section above it is evident that, in mixtures, resistant components reduce the general level of disease. However, disease development in mixtures depends on many factors. In particular, it depends on the composition of the mixture; the quality and amount of exogenous inoculum; and the number of pathogen generations during the active development of the epidemic (Wolfe, 1985). For example, cereal rust in the USA is almost directly related to the exogenous inoculum at the beginning of the epidemic. The number of pathogen generations are as few as three to four. In this case, the mixture offers little more than diversification. However, cereal mildew has 20 to 30 generations during an epidemic. Disease increases to carrying capacity in pure culture but not in mixtures (Wolfe, 1985). Thus, it is evident that the effect of introducing resistant components will vary according to the type of disease and presumably the proportion of resistants in the mixture.

Browning and Fry (1981) were able to estimate that to restrict crown rust of oats adequately, the proportion of the resistant component in the mixture needed to be between 40% and 50%. In contrast, mixtures containing 50% of a single resistant and 50% of a single susceptible variety developed severe rust epidemics. Jensen and Kent (1963), reported that as little as 40% of even partial protection in a population may provide full protection. Burdon and Chilvers (1982) found that a 50% resistant mixture substantially reduced the rate of spread of *Pythium irregular* and resulted in a lower level of disease compared to monoculture at the same density. These results imply that relatively high proportions of resistance are required in order to reduce significantly the general level of disease in mixtures.

One of the few studies in which good control was obtained with low percentage of resistance in mixtures was in experiments using the wheat:*Septoria nodorum* and barley:*Rhynchosporium secalis* disease systems (Jeger *et al.*, 1981). In this unspecialized host-parasite relationship, 25% resistance in a mixture resulted in large decreases in disease *vis a vis* that which was expected. If these results can be obtained for diseases of beans in Africa, then there is good reason to believe that the addition of resistant varieties to farmers' mixtures can have a large impact in the short term. If,

however, greater proportions of resistance are required to obtain substantial reductions of disease, then impact from resistant varieties will be slower and less measurable.

What other strategies can have impact in the control of disease in mixtures?

Variety non specific methods are likely to be of greater importance in systems where mixtures dominate than in those which use single varieties. Little literature was found on this topic. Nevertheless, it would be worth exploring further the relative merit of adapted use of improved cultural, biological and chemical techniques.

VARIETAL MIXTURES FOR RUST CONTROL

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INTRODUCTION

Given the predominance of varietal mixtures in a large part of Africa, the "improvement of a characteristic" approach to bean breeding seems particularly appropriate. Thus, multilines could be developed by selecting for optimal levels of resistance to individual diseases and then mixing components in appropriate proportions. For pathogens with several strains, like rust and anthracnose, elements with specific resistance genes to different races could be mixed and this should give stable resistance of the population. However, the means of achieving stable, high-yielding mixtures with suitable disease resistance have not yet been sufficiently studied for bean. The population dynamics of mixtures made up of components with different genes for disease resistance needs to be studied and this could be done with mathematical models once knowledge of the rate of spread of diseases is obtained.

Local mixtures should also be studied to determine what resistance already exists in the population and thus to predict the kind of resistance components that should be introduced to improve the level of disease resistance and the yield of the population.

An alternative solution to mixing pure lines would be a bulk selection approach, comprising the inter-crossing of lines carrying disease resistance genes with germplasm adapted to local conditions. These populations would be grown in plots managed by farmers, perhaps starting with the F_4 generation and the farmer would be encouraged to select appropriate components. This would have the advantage of involving the farmer in the selection process from the beginning and could eliminate the risk of the farmer selecting against introduced resistant components because of other undesired characteristics.

MATERIALS AND METHODS

The characteristics of genotypes selected for this study are listed in Table 21. They were selected to combine different growth habits and levels of rust resistance. The trials were carried out at CIAT, Cali, Colombia (1000masl, 3°N latitude) during three seasons from 1985 to 1986. The individual plots consisted of 6 rows 6 m long, surrounded by cowpea to reduce the spread of diseases between plots.

Table 21. Characteristics of the bean genotypes.

Genotype	Growth habit	Rust reaction	CBB reaction
ICA 15438	I	R	I
ICA L-23	I	R	R
BAT 1769	I	R	I
BAT 1297	II	S	S
XAN 43	II	R	R
PAI 49	II	I	S
PVMX 1531	III	S	S
XAN 33	III	I	S
G 12491	III	R	I

Growth habit: I = determinate bush; II = indeterminate bush; III = indeterminate semi-climbing
Disease reaction: S = susceptible; I = intermediate; R = resistant

The mixtures were composed by mixing genotypes with the same growth habit in equal proportions (e.g., M1 = mixture of type 1). Second order mixtures were made up by mixing the three combinations of types together (e.g. M12 = mixture of types 1 and 2). A third order mixture consists of the three genotypes mixed together (M123).

RESULTS

The yields of the mixtures are shown in Table 22. The coefficient of variation was 7.8% and the differences were highly significant. The expected yield for each mixture was calculated using the yield of each component in pure stand. The only mixture which did not yield significantly more than expected was the M1, in which all the components were rust resistant. The mixtures with the best yield increase were M2 and M23. The second and third order mixtures did not appear superior to first order mixtures, and their yields were predictable from the latter.

Table 22. Seed yields (kg/ha) of mixtures.

Mixture	Harvested yield	Expected yield	Mixture effect
M 1	1819	1845	-1.4
M 2	1871	1698	+10.2
M 3	2051	1949	+5.2
M 12	1844	1771	+4.1
M 13	1984	1880	+5.5
M 23	1955	1823	+7.2
M 123	1914	1831	+4.6
C.V. (%)	7.8		

In Table 23, the mixing coefficient is defined as the yield of single plants of a component in a mixture divided by the yield of single plants of the same component in pure stand. In order to measure this, it is important to be able to distinguish the components on the basis of grain type. It is clear from this table that BAT 1297 and PVMX 1531 contribute to the positive effect of mixing, since their mixing coefficient is always greater than one. These components were susceptible to rust. For example, in M2, PAI 49 loses 30% of its yield in the mixture, but BAT 1297 gains 60%. Factors other than protection against rust which could contribute to these yield gains are: large leaf area index, which contributes to competitive ability; and susceptibility to lodging. It is not likely that the former leads to more yield in mixtures, but the latter may because components susceptible to lodging could be supported by those resistant to lodging.

Table 23. Mixing coefficients[1] of genotypes in mixtures trials.

Genotype	M1	M2	M3	M12	M13	M23	M123	Mean	Rust
ICA 15438	0.9	-	-	0.8	0.9	-	0.8	0.8	R
ICA L-23	1.0	-	-	0.8	1.0	-	1.0	0.9	R
BAT 1769	1.1	-	-	1.1	0.9	-	1.0	1.0	R
BAT 1297	-	1.6	-	1.9	-	1.8	1.7	1.7	S
XAN 43	-	1.0	1.1	-	1.1	1.0	1.0	1.1	R
PAI 49	-	0.7	-	0.8	-	0.7	0.7	0.7	I
PVMX 1531	-	-	1.4	-	1.3	1.2	1.4	1.3	S
XAN 33	-	-	0.9	-	1.0	0.8	1.0	1.0	I
G 12491	-	-	1.0	-	1.2	1.1	1.2	1.1	R

[1] Mixing coeficient = single plant yield in mixed populations/single plant yield in pure stand

In Table 24, the yield losses obtained in the plots inoculated with rust, compared to the protected plots, are shown for the components in pure stand. On average, the resistant components lost 7% of their yield, compared to a 46% loss for the two susceptible components (BAT 1297 and PVMX 1531). The intermediate varieties lost on average 25%.

Table 24. Comparison of yields (kg/ha) in protected (P) and rust inoculated (I) plots of genotypes in pure stand.

Genotype	Rust reaction	Seed yield P	Seed yield I	I/P %
ICA 15438	R	1933	1984	103
ICA L-23	R	1857	1701	92
BAT 1769	R	1658	1502	91
XAN 43	R	1934	1849	88
G 12491	R	1986	1849	93
Mean				93
PAI 49	I	1755	1333	76
XAN 33	I	2313	1692	73
Mean				75
BAT 1297	S	2400	1057	44
PVMX 1531	S	2521	1615	64
Mean				54

In Table 25, we can see the mixing coefficient for individual varieties in the protected and inoculated plots. In general, there was no difference between the two coefficients for the resistant components, whilst there was a difference of 60% for the susceptible component, BAT 1297, which indicates that this component gained competitive capacity in the inoculated plots, because of protection against rust afforded by the neighbouring resistant plants. It was observed that PVMX 1531 was also much less affected by rust in the inoculated plots when in mixtures, but this did not result in an increased mixing coefficient because the component had a greater tendency to lodge when it was not affected by rust.

Table 25. Mixing coefficients of genotypes in protected (P) and rust inoculated plots.

Genotype	Rust reaction	P	I
ICA 15438	R	0.70	0.71
ICA L-23	R	1.03	1.00
BAT 1769	R	0.86	1.00
XAN 43	R	1.13	1.07
G 12491	R	0.73	0.84
PAI 49	I	0.68	0.84
XAN 33	I	1.05	1.10
BAT 1297	S	1.42	2.03
PVMX 1531	S	1.33	1.34

CONCLUSIONS

It was concluded that the yield of a variety in pure stand was not a good parameter for estimating its competitivity, especially in the presence of rust. Leaf area at flowering provided a good estimate of competitive ability, in protected or inoculated conditions. This was because the plants infected with rust tended to attain a smaller maximum leaf area index and leaf area duration was shorter. The difference between leaf area indices at flowering in inoculated or protected plots of BAT 1297 explained the difference in competitive abilities observed when individual plants were protected against rust infection by resistant neighbouring plants in the mixture.

The increased mixing coefficient of a susceptible variety like BAT 1297, which is in any case relatively competitive, would lead to an increase in the proportion of the susceptible component in the mixture after several seasons, if the mixture were simply harvested and replanted without intentional selection. It is supposed that such a process would continue until the proportion of resistants in the population became too low to afford protection against rust, at which point the susceptible component would again lose its competitive capacity. This could result in a cyclical change in the proportions of resistant and susceptible components, which would imply that yield losses due to rust would occur from time to time, when there was a large proportion of susceptibles in the population. It is preferable, therefore, that for a given disease, the resistant components be at least as competitive as the susceptible components.

VARIETAL MIXTURES FOR ANGULAR LEAF SPOT CONTROL

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INTRODUCTION

The utilization of mixtures composed of different cultivars of common bean (*Phaseolus vulgaris*) is common practice for farmers in the Great Lakes region of central Africa. The genetic diversity of mixtures can have a protective effect against various agents, among them pathogenic micro-organisms. But angular leaf spot caused by *Phaeoisariopsis griseola* can be severe in some circumstances, despite the heterogeneity of the mixture. In addition the yields of the mixtures are often inferior to those of improved cultivars. Studies conducted elsewhere on varietal mixtures of common bean (Ishabairu and Teri, 1984) and cereals (Jeger *et al.*, 1981; Leonard, 1969; Wolfe *et al.*, 1981) have shown that disease severity is reduced in mixtures compared to that in the same varieties grown in pure stand and that this can lead to an increase in yield of the mixtures. This phenomenon is attributed to the interception of air-borne spores of the pathogen by resistant plants, which thus create a barrier effect.

MATERIALS AND METHODS

Two experiments were conducted during the A and B seasons of 1987 on the the experimental station at Mulungu-Tshirumbi in a field naturally infected with *Phaeoisariopsis griseola*.

The first experiment, sown in late November of 1986 (A season), suffered severe damage due to bean stem maggot and ascochyta blight. The second experiment was sown on 2 April 1987 (B season). The treatments were the local cultivar (itself a mixture), purchased in the market, and the resistant line BAT 76 combined in the proportions (local cultivar:BAT 76) 100:0, 90:10, 80:20 and 0:100 (A season) or 100:0, 75:25, 25:75 and 0:100.

Both experiments were randomized complete blocks with plots of 4 m x 4 m separated by 8 m wide strips of soyabean to reduce contamination with spores from neighbouring plots. Sowing was in rows 20 cm apart with 20 cm between plants within rows, to simulate the planting system of farmers which is broadcasting. BAT 76, was sown at random within the rows of the mixtures and plants were marked to distinguish them from the local cultivar.

Angular leaf spot was rated at full flowering (R6), pod formation (R7) and maturity (R9) on the 3rd, 5th and 7th trifoliolate leaves of eight plants chosen at random from each of rows 3, 7, 10, 13 and 16 of each plot (i.e. 40 plants per plot) and expressed in terms of percentage leaf/pod area infected. In addition, on each evaluation date, the angular leaf spot reactions of whole plots were rated on a scale of 1-9, where 1 = no infection and 9 = 50% or more of leaf area infected. Grain yields were recorded in season B. Analyses of variance of disease ratings and grain yields were conducted using MSTAT.

RESULTS AND DISCUSSION

In both seasons, angular leaf spot severity was significantly less on BAT 76 than on the local cultivar (Tables 26 and 27). In season A, the inclusion of 10 and 20% BAT 76 in mixtures with the local cultivar had no effect on angular leaf spot severity (Table 26).

Table 26. Effect of adding a resistant line to a local cultivar (LC) on percent leaf area infected by angular leaf spot in season 1987A.

Combinations LC:BAT 76	3rd leaf	5th leaf			7th leaf		General	
	R6	R6	R7	R8	R7	R8	R7	R8
100:0	16.6a	0.8a	19.0a	20.4a	9.6a	12.1a	32.2	57.5a
90:10	16.9a	0.8a	19.0a	19.0a	8.1a	10.9a	27.2	33.3a
80:20	18.6a	0.9a	19.1a	20.8a	8.3a	11.8a	20.ab	29.2a
0:100	3.1b	0.5b	5.1b	5.9b	0.3b	0.4b	11.3b	11.3b

Means in the same column followed by the same letter are not significantly different (P = 0.05) according to the L.S.D.

In contrast, in season B, the severity of *P. griseola* was significantly reduced on the 5th leaf at the pod filling stage (R8) in the 75:25 and 25:75 local cultivar:BAT 76 mixtures, which did not differ significantly from eachother (Table 27). The general disease score at the same stage was also less for the mixtures (3 and 1.5%) than the local cultivar (14%).

BAT 76 and the mixture containing 75% BAT 76 produced significantly greater yields than the local cultivar (Table 27). The two mixtures did not differ significantly in yield. The increases in yield of the mixtures over the local cultivar were 8 (25% BAT 76) and 28% (75% BAT 76), proportional to the content of BAT 76 in the mixtures. Similar results were obtained in Tanzania by Ishabairu and Teri (1984), though they observed the farmer's mixture to be least infected.

Table 27. Effect of adding a resistant line to a local cultivar (LC) on angular leaf spot and grain yield in season 1987B.

Combinations LC:BAT 76	Per cent leaf area infected					General score	Grain yield (kg/ha)
	3rd leaf	5th leaf		7th leaf			
	R6	R6	R8	R7	R8	R8	
100:0	5.9a	2.1a	4.2a	1.4a	2.6a	14.0a	609b
75:25	4.5ab	2.0a	2.4b	1.3a	1.6ab	3.0b	658ab
25:75	5.1ab	1.7a	2.5b	1.0a	1.5ab	1.5b	777a
0:100	1.2b	1.4a	0.7c	0.4b	0.3b	1.5b	753a

Means in the same column followed by the same letter are not significantly different (P = 0.05) according to the L.S.D.

Finally, though it is premature to draw conclusions from this study, these preliminary results indicate that the mixing of a resistant component with a susceptible variety may provide an effective strategy for the control of angular leaf spot in bean.

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CHEMICAL CONTROL

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INTRODUCTION

Chemical control is a term which is unknown in agricultural circles in Rwanda, especially on small-scale farms. Notwithstanding, given its demographic, geographic and economic situation, Rwanda must intensify its agriculture to meet its nutritional and economic needs. The intensification of agriculture necessarily implies the use of agricultural inputs such as fertilizers (chemical or organic), plant protection products and all the tools and apparatus for their application. Countries with high agricultural production are those which use large quantities of agricultural inputs. On the other hand, countries where people die of hunger and suffer from malnutrition are those where the use of agricultural inputs is rare or unknown.

Biological methods (cultural practices and resistant cultivars) can reduce damage from certain diseases, though only in rare cases do these confer complete protection. Nevertheless, chemicals are expensive, so the use of biological methods to control the diseases of cultivated plants must be considered as a means of reducing the expenses involved in buying plant protection products. For example, one or two chemical treatments per week for a susceptible cultivar, may be reduced to one treatment every two or three weeks for a resistant or tolerant cultivar. This reduces the amount of chemical and labour used and wear-and-tear on the equipment to apply the treatments.

In my paper I will briefly describe the chemicals generally recommended for the control of certain bean diseases; then we will look at the research carried out in Africa on chemical plant protection, particularly in Rwanda. We will end by outlining the problems posed by the use of chemicals in Rwanda and the importance of the research carried out.

CHEMICALS RECOMMENDED FOR THE CONTROL OF BEAN DISEASES

The chemicals, doses and treatment frequencies which I present below are from Schwartz and Galvez (1980). Table 28 lists the different diseases and the products used to control them.

Table 28. Chemical control of important diseases of common bean.

Disease/ pathogen	Chemical: Common name	Rate/ha	Remarks
Rust	Sulphur	25-30 kg	every 7-10 days after appearance of pustules
	Chlorothalonil	225 g	in 100 l water
	Maneb (Dithane M22)	4-5 kg	
	Maneb (Manzate D80W)	4 kg	in 1,000 l water
	Mancozeb	3-4 kg	
	Oxycarboxin	1.5-2.5 kg	20 & 40 days after sowing/ every 2 wks to flowering
curative - 3 days after infection, preventive - 7 days before infection			
Anthracnose	Ferbam, Ziram, Thiram, Ceresan	5 g/kg	seed dressings
	Maneb, Zineb	3.5 g/l	preventive treatments
	Benomyl	0.55 g/l	" "
	Captafol	3.5 kg/ha	" "
	Carbendazim	0.5 kg/ha	" "
	Fentin hydroxide	1.5 g/l	" "
Angular leaf spot	Sulfur, Ferbam		adherent
	Zineb	0.5 g/l	
	Benomyl		seed treatment
Rhizoctonia	PCNB	5.8 kg	in 378 l water
	Benomyl, Carboxin Busan, Thiram, Zineb	1-3 g/kg	seeds
	Chloroneb, Captan	5.8 kg	in 378 l water
	PCNB + Captan or Pyroxychlor		for *Pythium*
Fusarium solani	Mabam, Formaldehyde, Thiram, PCNB		
	Benomyl	0.56 kg	just after sowing
	Captafol	4.7 l	
	Busan	2.4 l	just after sowing
F. oxysporum Schlecht. f. sp. *phaseoli*	Ceresan Semesan		to seeds, as for *F. solani*
Sclerotinia sclerotiorum	Benomyl, Dicloran, Dichlone, PCNB (thiabendazole)		

Table 28 (continued).

Disease/ pathogen	Chemical: Common name	Rate/ha	Remarks
Alternaria leaf spot	Chlorothalonil	1200 g/l	
	Thiophanate	2 g/l	
	Zineb	2.4 g/l	
Ascochyta blight	Sulphur		seed treatment
	Benomyl	0.55 g/l	
	Zineb	2.4 g/l	
	Chlorothalonil	2.24 kg	
Macrophomina	Ceresan		seed treatment
	Benomyl	1 kg	
Mycovellosiella phaseoli	Benomyl	0.55 g/l	
	Thiophanate	2 g/l	
Erysiphe polygoni	Sulphur		
	Dinocap	1.2 g/l	
BACTERIA			
Common blight	Cu sulphate		
Halo blight	Bordeaux mixture	0.2-0.4 kg	weekly or twice weekly treatment - impractical/costly
	Cu oxychloride		
	Cu sulphate		
	Cu oxide		
	Streptomycin sulphate		
	Dihydrostreptomycin sulphate		
	Captan	1.2 g/kg	seed treatments - superficial - no chemical control for seed transmission
	Ceresan		
	Thiram		
	Benomyl		

[1] other names of chemicals: Chlorothalonil = Daconil; Maneb = Dithane M22; Maneb = Manzate D80W; Mancozeb = Dithane M45; Oxycarboxin = Plantvax; Ferbam = Carbamate, Ferbame; Ziram = Antene, Carbazine; Thiram = Arasan; Zineb = Aspor, Dithane Z-78; Benomyl = Benlate; Captafol = Difolatan; Carbendazim = Bavistin; Fentin hydroxide = Du-Ter; PCNB = Brassical, Avical; Carboxin = Vitavax; Chloroneb = Demosan; Captan = Merpan, Orthocide; Pyroxyfur = Grandstand, DOWCO 44; Captafol = Difolatan; Dicloran = Botran, Allisan; Dichlone = Phygon, Quintar, Mertect; Thiophanate = Topsin; Dinocap = Karathane, Mildex; Cu sulphate = Bluestone; Bordeaux mixture = Cu sulphate + Ca hydroxide; Cu oxychloride = Blitox; Cu oxide = Cuprous oxide.

RESEARCH ON PLANT PROTECTION IN AFRICA

Some publications can be found, here and there for certain countries, on research concerning the chemical control of bean diseases. In my case, I have limited myself to abstracts as published in CIAT (1983, 1984, 1986 and 1988). Thanks to these abstracts, we can obtain an idea of the different fungicides used in certain African countries. We do not presume to present a complete list, as we hope it can be complemented by some of those present.

Research carried out by Maramba (1983a, 1983b) in Zimbabwe showed that Benomyl, Manzozeb, Fentin Acetate, Manebe, Metirame, Thiram and Zineb were very effective in the control of *C. lindemuthianum*. Benomyl and Zineb are recommended against *Ascochyta phaseolorum*.

Fadl (1984) in Egypt effectively controlled bean diseases with oxycarboxine.

In Tanzania the work of Bujulu and Lotasarwaki (1983) demonstrated the efficacy of oxycarboxin, Cu hydroxide, Mancozeb, benomyl, chlorothalonil and triforin against bean rust, in a trial with 13 fungicides. Jaffer (1971) doubled yields by treating bean rust with either oxycarboxin (2.5 kg m.a/ha), or triforin (0.1% in aqueous solution) or butrizol (0.675 kg/ha).

In Uganda, Simbwa-Bunnya (1973) effectively controlled *Uromyces phaseoli, Sclerotium sclerotiorum and Isariopsis griseola* with Benomyl, Thiabendazole, Mancozeb and Captafol. In the same country, Sengooba (1985) showed that treatment with Mancozeb, Benomyl or Triphenyltin acetate effectively controlled *Phaeoisariopsis griseola* and *C. lindemuthianum*.

RESEARCH ON PLANT PROTECTION IN RWANDA

At the moment we have very few results concerning plant protection treatments in Rwanda. Experiments have been carried out by CIAT staff (Trutmann and Kayitare, personal communications). The only results we have are those of experiments carried out in 1966, 1967 and 1969. At ISAR-Rubona in 1966, treatment against rust with Propeneb or copper oxychloride almost doubled bean yields (ISAR, 1982).

CULTURAL CONTROL WITH EMPHASIS ON AFRICA

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INTRODUCTION

Cultural disease control includes all those measures designed to reduce disease problems through the manipulation of the crop or the cropping environment without the use of chemicals or active breeding. Cultural control measures are in many cases old practices based as much on the art of the farmer as on agricultural science. There are three basic groups of

cultural control measures: 1) those aimed at eliminating the pathogen from the plant or from the area in which the plants are growing; 2) those directed at the production of pathogen-free propagative materials; and 3) those intended to increase the resistance of the host to the pathogen or create conditions unfavourable to the pathogen (Agrios, 1978).

CULTURAL CONTROL MEASURES

In the case of bean diseases many cultural control measures have been studied, though mainly outside of Africa. The measures recommended for different diseases are summarized in Table 29 (Zaumeyer and Thomas, 1957; Schwartz and Galvez, 1980).

Table 29. Cultural control measures recommended for important diseases of phaseolus bean in Africa.

Control measure	Diseases								
	Rust	Anth	BB	ALS	RR	WB	WM	AB	BCMV
Crop rotation	+	+	+	+	+	+	+	+	+
Removal of plant debris	+	+	+	+		+	+	+	
Deep ploughing			+	+	+				
Planting disease free seeds		+	+	+		+	+	+	+
Adjusting planting dates	+					+			+
No movement in crop during wet weather		+	+						
Shallow planting					+				
Reduced plant density	+				+		+	+	

Anth = anthracnose; BB = halo and common bacterial blights; ALS = angular leaf spot; RR = root rots; WB = web blight; WM = white mould; AB = ascochyta blight; BCMV = bean common mosaic virus

The cultural control measures mentioned include crop rotation, an old practice used mainly to maintain soil fertility but also for disease control. A 2-3 year crop rotation is often mentioned as a useful practice to minimize bean diseases levels (Table 29) (Schwartz and Galvez, 1980). Available literature revealed no active research on crop rotation as a bean disease control measure anywhere in Africa. However, many crop production handbooks and reports contain recommendations of crop rotation as a control for a range of bean diseases.

Karel *et al.* (1981), in his report on bean production in Tanzania mentioned crop rotation as a recommended control measure for halo blight (*Pseudomonas syringae* pv. *phaseolicola*) and anthracnose (*Colletotrichum lindemuthianum*). Edje *et al.* (1981) reported crop rotation as one of the recommended control measures for *Phaeoisariopsis griseola* in Malawi. Sengooba (1980) studied the survival of *P. griseola* in bean straw kept under various conditions and deduced that crop rotation can control this pathogen. Mostade (1977) working in Tanzania recommended crop rotation as a control for *Rhizoctonia solani* and mentioned that grasses should be included in the

rotation and Westhuizen *et al.* (1979), in South Africa, made a similar recommendation for the control of *Sclerotium* rot. The value of crop rotation will however, vary with the cropping system and will largely depend on whether there are sources of inoculum other than soil.

The removal of bean plant debris from the field is widely practised in Africa as, in most cases, whole plants are harvested. This harvesting method, however, leaves parts of plants in the field, mainly leaves, which fall on the ground as they senesce and defoliate through the life of the crop. Proper field sanitation can be achieved possibly in a few cases where deep ploughing is practised in addition to removing the plant debris. Under Kawanda conditions, Sengooba (1980), found the survival of *P. griseola* under soil to be around two months while it was up to nine months under laboratory conditions. This observation supports deep ploughing as beneficial in controlling *P. griseola* and work carried out outside of Africa indicates that it is an effective control for bacterial blight and the root rots.

Use of disease free-seed is desirable. However, in many African countries, disease-free seeds are not obtainable. There are a few seed companies but the needs of the farmers are in most cases not satisfied in terms of quantity and diversity. In the majority of cases therefore, farmers grow, store and sort their own seeds.

Studies have been carried out in Rwanda on the effect of improved seed selection methods and the removal of diseased leaves and seedlings, particularly those infected by important seedborne diseases such as anthracnose, angular leaf spot, ascochyta blight and bean common mosaic virus. The results indicated significant reduction in disease levels and up to 12% increase in yield was recorded where seed was selected and diseased leaves and seedlings were removed in the early stage of the crop.

In another experiment, comparing disease free seeds with seed produced at the same location, again in Rwanda, a 22% increase in yield was recorded (CIAT, 1985 and 1986). Omunyin (1983), in his report on viral diseases of beans, pointed out that in Kenya, the practice of rogueing to control BCMV has been limited mainly to seed multiplication trials but considering the need for bean seeds free of BCMV and the tendency of farmers to use seed from previous harvests for planting, rogueing could be very useful in eliminating the initial source of virus inoculum in farmers' fields.

Adjusting sowing dates is a desirable control measure for most diseases, so that movement of inoculum from early to late sown crops is avoided. According to Schwartz and Galvez (1980), bean planting dates for specific production zones are recommended in order to avoid the incidence of rust (*Uromyces appendiculatus*) infection during preflowering to flowering stages of plant development; for web-blight (*Thanatephorus cucumeris*), early sowing is required to ensure that the crop will mature before the following rainy season and for BCMV, to avoid the vector build up which occurs as the season progresses.

However, under the smallholder, subsistence farming systems widely practised in Africa, it is difficult to adjust sowing time sufficiently to eliminate the transfer of inoculum from older to younger fields because sowing time is governed by a range of factors, including rainfall season and pattern and other farm activities, so it is difficult to change in a farming

community for the sake of a partial solution to a disease problem.

Avoidance of cultivation of row crops while dew is present on plants reduces spread of water borne spores. This has long been a sound procedure in the control of anthracnose and the bacterial blights - *P. phaseolicola* and *Xanthomonas phaseoli* (Walker, 1969). However, when Habtu Assefa (1981) studied the effect of weeding practices on the control of common bacterial blight at Melkassa in Ethiopia, neither early nor late morning weeding influenced the level of the diseases.

Reduced plant density was reported as a possible means of reducing bean rust, web blight and the root rots (Schwartz and Galvez, 1980). Habtu (1981) found that intra-row spacing influenced bacterial blight more than inter-row spacing.

Walker (1969) stated that "to be sure the grower of crops has little control over the weather but it is well for a student of plant pathology to consider how much the art of agriculture is influenced by the relation of climate to disease". The shifting of growing bean seeds in the USA to the irrigated Rocky Mountains and the Pacific coast as a means of controlling anthracnose and bacterial blight was quoted as an example. In the last decade or so plant pathologists have realized that it is not only climate but also the micro-environment created in intercropping and mixed cultivation systems, as largely practised in Africa, that influences disease levels differently from sole cropping and often contains an in-built cultural control to diseases. All available evidence suggests that the biotic, structural and micro-climatic complexity of multi-crop systems works synergistically to produce an association resistance (Altieri and Liebman, 1986). Several studies have been carried out on this subject in Africa.

Van Rheenen *et al.* (1981) studied disease levels in bean grown in pure stand or in association with maize in breeding and agronomy trials in Kenya. Bean grown in association with maize showed generally less incidence of common and halo blight, bean common mosaic, anthracnose, scab (*Elsinoe phaseoli*), black node disease (*Phoma exigua* var. *diversispora*), mildew (*Erysiphe polygoni*) and, to a lesser extent, angular leaf spot.

Mukiibi (1980) carried out a trial where bean and groundnut were grown in pure stand and in mixtures of two thirds bean:one third groundnut, one third bean:two thirds groundnut. The severity of groundnut rosette and of the leaf spot disease caused by *Cercospora arachidicola* was less in the intercrops. The diseases that developed on the bean crops were rust, angular leaf spot and white mould and their levels in pure stand did not differ from those in association.

Msuku and Edje (1982) studied the effect of mixed cropping of maize and bean on the bean diseases and reported that the damage by bacterial blight, rust, anthracnose and ascochyta blight was significantly greater in pure stands of bean than in bean grown in association with maize. Angular leaf spot incidence was greater in the intercrops at the two sites used. Dwarf bean had more web blight in pure stand than in association with maize but in climbing bean, it was observed that web blight incidence at one site was higher in the maize:bean association than in pure stands of beans.

The literature reviewed indicates that intercropping will have a variable effect on disease level depending on the disease, the associated crop, the location and the growth habit of the bean cultivar. This observation is supported by results from research carried out in Latin America (Moreno, 1977). Van Rheenen *et al* (1981) attributed the differences in disease levels in intercrops from those in sole crops to differences in relative humidity and temperature in the two systems. In the case of rust, Allen (1976) suggested that cross protection may occur between the rusts of maize and beans when the two are intercropped.

Therefore, the influence of cropping system on epidemics of diseases depends on many variables and it cannot easily be predicted. Experiments with different crop associations must be carried out and appropriate cropping patterns have to be developed for different ecological zones. Much work is needed before a general theory can be developed on the effects of cropping patterns on diseases.

CONCLUSIONS

Disease cultural control being an art, is indeed practised in Africa. The contribution of such measures to disease reduction in bean and how they can be further improved for the African farmer is an area which deserves more attention than it has received in the past. Some of the cultural control techniques have a small effect when considered alone but when integrated with other measures they should contribute significantly to efficient disease management.

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SECTION 9: SUPPLEMENTARY PRESENTATIONS

BEAN IMPROVEMENT RESEARCH IN ZAMBIA - PROGRESS AND PROSPECTS

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ABSTRACT

Although research on bean has been in progress for about three decades in Zambia, concerted and multidisciplinary team effort commenced only with the creation of the Grain Legume Commodity Research Team under the Eastern Province Agricultural Development Project at Msekera Regional Research Station in 1982. Evaluation of germplasm and breeding materials, mostly from CIAT, has resulted in the identification of several high yielding and/well adapted lines; one of these has been released for cultivation and another approved for national level pre-release testing.

Diseases are a major constraint and good progress has been made for screening for resistance to major diseases, besides identifying the existence of NL 3 BCMV strain in Zambia. Several genotypes with multiple disease resistance/tolerance have been identified and some of them have good yield potential. Among pests, research on beanfly resistance is in progress in collaboration with CIAT and seed treatment with endosulfan has been found to give control of this pest. Resistance/tolerance to flower/pod damaging insects and economics of controlling them with insecticides are being investigated.

Appropriate agronomic studies, especially with climbing bean as an intercrop with maize and suitable on-farm trials through ARPT are also in progress. The scope for introducing high yielding varieties which possess disease-resistance, coupled with simple and economic means of pest management has been adequately demonstrated. Future research, in collaboration with CIAT and other countries in the region, is expected to lead to clear monetary benefit to the small farmers who mostly cultivate bean in Zambia.

SCREENING COMMON BEAN FOR RESISTANCE TO MAJOR DISEASES IN ZAMBIA

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ABSTRACT

Screening of a large number of bean genotypes for resistance/tolerance to the five major diseases - bean common mosaic virus, scab, angular leaf spot, ascochyta blight and anthracnose - has been carried out for three cropping seasons during 1983-86. Besides resistance/tolerance for individual diseases, several genotypes showing multiple disease resistance/tolerance were identified. Of these, Carioca, ZPv-292, PAT 10, PAT 12, PAT 16, PAT 26, PAT 78, PAT 106, A 429, A 439 and A 442 have shown promising grain yield potential in national yield trials.

INTRODUCTION

Common bean (*Phaseolus vulgaris*) is the most important food grain legume crop of Zambia, where it is grown mainly in higher altitude, cooler and wetter areas. These include North-Western, Copperbelt, Central, Luapula, Northern and Eastern Provinces, occupying approximately the northern half of the country. A large number of diseases have been recorded (Angus, 1962-66) in Zambia and constitute major constraints to bean production. Observed at moderate to serious levels (more than 15% incidence or 25% plant surface affected) are: bean common mosaic virus (BCMV); angular leaf spot (*Phaeoisariopsis griseola*); scab (*Elsinoe phaseoli*); ascochyta blight (*Phoma exigua* var. *diversispora*); anthracnose (*Colletotrichum lindemuthianum*); rust (*Uromyces appendiculatus*); ashy stem blight (*Macrophomina phaseolina*); web blight (*Thanatephorus cucumeris*); and the bacterial blights (*Xanthomonas campestris* pv. *phaseoli* and *Pseudomonas syringae* pv. *phaseolicola*). BCMV causes serious damage in warmer, drier areas; most fungal diseases tend to be severe in wetter, cooler areas; and bacterial blights have widespread distribution.

Bean is grown mainly as a subsistence or local market crop by small-scale farmers who can not afford to buy chemicals to control diseases. Yield losses of between 25 and 50% due to diseases are common (Greenberg *et al.*, 1986). The only practicable means of controlling diseases in Zambia is the development of resistant or tolerant cultivars. From 1982 to 1986, a large number of local and introduced (mainly from CIAT) bean genotypes were evaluated for yield potential and disease resistance in the major bean growing areas of Zambia. This paper reports the results of this evaluation.

MATERIALS AND METHODS

Disease reactions were rated in a large number of breeding trials and disease nurseries at Chipata (warm, medium rainfall) and Mbala (cool, heavier rainfall) from 1982 to 1986. Entries identified by the prefix ZPv are from

the Zambian germplasm (Zambian *P. vulgaris*): A, G, BAT, PAT, PAI, PVAD and VEF entries are germplasm or breeding lines from CIAT.

Breeding trials

The breeding trials included the Zambia National and Preliminary Variety Trials, and CIAT International Bean Yield and Adaptation Nurseries (IBYANs) and unreplicated observation sets of germplasm and CIAT advanced breeding lines (VEFs and EPs).

Disease nurseries

Entries for disease nurseries were selected based on disease reactions in breeding trials and observation plots. Rows of susceptible spreader lines are sown two weeks previously to test materials to ensure uniform spread of disease and eliminate escapes. Nurseries specifically for BCMV have been conducted at Chipata and for the foliar diseases, scab, rust, angular leaf spot (ALS), anthracnose and ascochyta blight (AB) at Mbala. The latter four are CIAT international nurseries to determine pathogenicity patterns.

Methods

Natural outbreaks were usually uniformly severe allowing screening for resistance/tolerance to several diseases. Diseases were identified from their symptoms and, where necessary, microscopic examination. Severity was rated on 1-9 scales (where 1 = no symptoms and 9 = extremely severe disease) in early and late podding. Based on their disease reactions over two to four seasons, entries are classified into four resistance groupings: resistant (1); moderately resistant (2-3); tolerant (4-5); and susceptible (6-9).

Analyses of variance were conducted of data from replicated tests and disease scores are reported only if there are significant differences among entries.

RESULTS AND DISCUSSION

The severity of individual diseases varied between locations. BCMV predominated at Chipata, where the weather is usually warm and dry, but when rainfall was above average, rust, ALS, web blight and the bacterial blights (BB) also caused damage to susceptible entries. At Mbala (wetter and cooler), scab, ALS and AB were most severe and anthracnose, rust and BB moderate. The disease reactions of entries at the two locations are summarized in Table 30.

Table 30. Numbers of bean entries evaluated in Zambia from 1983 to 1986, their disease reaction groupings and the identities and disease reactions of the susceptible checks.

Disease	Years	No. of seasons	Locations	Susceptible checks		Test entries			
				Identities	Reactions	No.	R	MR	T
BCMV	1983-86	3	Chipata	MSS/ML	7.0	1583	10	31	25
Scab	1984-86	2	Mbala	MSS/A 485	8.0	706	54	26	3
ALS	1984-86	2	Mbala	ICA Linea 24	9.0	627	5	68	41
AB	1984-86	2	Mbala	ML/Carioca	7.0	1438	0	7	10
Anth	1984-86	2	Mbala	A 463/BAT 1380	8.5	488	2	10	7

R = resistant; MR = moderately resistant; T = tolerant; anth = anthracnose
MSS = Misamfu Speckled Sugar; ML = Mbala Local

Bean common mosaic virus

A very severe outbreak of BCMV occurred in the 1983-84 season at Chipata; in subsequent seasons BCMV was severe there. BCMV was not severe at Mbala but there were occasional outbreaks. A set of differential bean genotypes has established the local BCMV strain as NL3 in pathogenicity group VI (Kannaiyan and Greenberg, unpublished). Drijfhout (1978) reported Great Northern 31 and Red Mexican 35 to be resistant to NL3. Ten entries (ZPv 263, 287 and 292; V 4604, Pinto 114, Great Northern 31 and 123; Redlands Greenleaf A and B; and Red Mexican 35) were classified resistant in Zambia. Some of the entries with resistance or tolerance to BCMV (ZPv 248 and 292; BAT 1426, A 485, Pinto 114, PI 150414 sel., Mexican 142 sel. and G 13595) have also produced good yields in Zambia and have been included in national yield trials.

Scab

Mutitu (1979) reported the occurrence of scab on bean in Kenya, recording yield losses of 43-76% due to the disease. A natural outbreak of scab at Mbala in 1984-85 caused severe damage to susceptible materials and a scab resistance nursery was formulated for 1985-86, when the disease was also severe. Of 706 entries evaluated during the two seasons, 54 were classified resistant, 26 moderately resistant and 3 tolerant. Interestingly, resistance was found among entries from Latin America, where the disease is not known.

Angular leaf spot

This is probably the most damaging disease of bean in Zambia, occurring in nearly all bean producing areas. ALS was most severe at Mbala but also caused considerable yield loss in Chipata in 1985-86. Out of a total 627 bean entries evaluated, five (Carioca; BAT 477 and 1671; A 262 and Nanzinde) were classified resistant, 68 were classified moderately resistant and 41 were classified tolerant. Most of the resistance and tolerance was found among

materials from Latin America, where the disease prevails in many countries (Schwartz and Galvez, 1980).

Ascochyta blight

Though AB is common on bean in high rainfall areas of Zambia, it is not usually associated with loss in grain yield (Greenberg *et al.*, 1986). However, it can cause loss in yield when infection occurs during early stages of growth (Weber, 1973) and such losses have occurred with climbing bean lines, which have a considerably longer growing growth period than bush types. Severe outbreaks of AB occurred at Mbala in 1984-85 and 1985-86 and a AB nursery containing local and introduced lines was formulated for 1985-86. Of 1438 entries evaluated, none were classified resistant, seven were classified moderately resistant (G 3736, 3993, 6040 and 9603; A 152, Mexico 6 and Diacol Calima) and ten were classified tolerant.

Anthracnose

Anthracnose is most severe in the high rainfall areas of Zambia and has been observed mainly in Mbala. Severity varied among seasons being most severe in 1985-86. Two entries were classified resistant (A 267 and G 15971), ten (including Carioca) were classified moderately resistant and seven were classified tolerant.

Minor diseases

The authors have frequently observed rust at low to moderate levels in farmers' fields in high rainfall areas of Zambia without obvious loss in grain yields. The disease occurred in trials at Mbala in 1983-84, 1984-85 and 1985-86 - entries susceptible at Mbala showed only moderate levels of rust in Chipata where the disease is generally not severe. In evaluations in 1983-84 and 1985-86, two entries (Carioca and G 11254) were classified resistant, 16 were classified moderately resistant and most others were classified tolerant.

Ashy stem blight was observed at Chipata in 1983-84, when it caused quite severe damage in some bean entries. The outbreak was associated with a drought period of about 20 days shortly following crop emergence. Of 131 entries evaluated, one was classified resistant (BAT 1572), 21 (including Carioca, ZPv 292 and A 439) were classified moderately resistant and 75 were classified tolerant.

In Zambia, bacterial blights include common and halo blight. The two are grouped since halo blight occurs at the beginning of the season and is succeeded by common blight in all bean producing areas (Greenberg *et al.*, 1986). Of 733 entries evaluated in 1984-85 and 1985-86, ten were classified moderately resistant (Carioca, Mbala Local, BAT 85; A 369, 411, 429, 442 and 485; ZPv 132 and 292) and three were classified tolerant.

Moderate levels of web blight were observed on climbing bean types in association with maize at Chipata in 1984-85, presumably due to the warm, humid micro-climate in the dense crop canopy (Schwartz and Galvez, 1980). Of

202 entries evaluated, 108 were classified moderately resistant and 92 were classified tolerant.

Multiple disease resistance

In Zambia, several disease often occur simultaneously in bean crops, so there is need to identify sources of combined resistance. Some such sources are listed in Table 31.

Table 31. Bean entries combining resistance or tolerance to two or more major diseases in Zambia.

Disease resistances	Bean entries
Four diseases	
BCMV/scab/ALS/anthracnose	A 442[a]; BAT 477[b] and 1671
Three diseases	
BCMV/scab/ALS	BAT 85[a] and 331[bc]
BCMV/ALS/anthracnose	ZPv 292[ab], BAT 1426[c]
BCMV/AB/anthracnose	Diacol Calima
Scab/ALS/anthracnose	Carioca[abc]; A 429[ac] and 439[b]
ALS/AB/anthracnose	A 252
Two diseases	
BCMV/scab	G 6500
BCMV/ALS	ZPv 308; G 5132[b] and 5066[ab]; BAT 1386 and 1387; A 485[a], Pinto 114, Monroe, Mexican 142 sel.
Scab/ALS	A 262[b], ZPv 132[a], Mbala Local[a], BAT 1297[c], 84VEF 1002; PAI 16, 26, 29, 48, 61, 77, 78, 88, 97 and 106; PAT 10, 11 and 12; PVAD 1193 and 1368
ALS/AB	A 152 and 345; G 5971 and 6040
ALS/anthracnose	A 167[c] and 242; G 11254

Also resistant or tolerant to: [a] BB; [b] ashy stem blight; [c] rust

Carioca, a Brazilian cultivar that has been successfully released in Zambia, shows resistance or tolerance to six of the nine diseases recorded. Five other entries (A 429 and 442; BAT 331 and 477; and ZPv 292) express resistance or tolerance to five diseases; six entries (BAT 85, 1426 and 1671; A 252 and 439; and G 5066) were resistant or tolerant to four diseases; and a large number of entries were resistant or tolerant to three diseases. In 1985-86, G 2338 from CIAT exhibited combined resistance to all five major diseases (BCMV, scab, ALS, anthracnose, BB and AB).

Many of the lines with combined resistance, including Carioca, ZPv 292, PAT 10, PAI 16, 26, 78 and 106 and A 429, 439 and 442, have produced good yields in national yield trials. These resistant sources offer good prospects for direct release or for the development of disease resistant cultivars.

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IDENTIFICATION AND COLLECTION

Chairperson: David Allen;
Rapporteur: Peter Trutmann

The objective of this session was to discuss issues in the identification and collection of bean pathogens in Africa.

In particular:

1) the need to identify and record bean pathogens in Africa;

2) the need to establish a central document with checklists from all participating countries and to deposit samples in a central herbarium for future reference;

3) the effects of pathogen interactions.

As a basis to all pathology research it is important that pathogens be identified correctly. Certain pathogens are difficult to distinguish. This can lead to misidentification of pathogens. Examples are CBB and ascochyta blight; CBB and floury leaf spot, when spores of the latter are no longer visible; anthracnose and ascochyta blight, when the attack is early and heavy; ascochyta and ALS on primary leaves; BCMV and certain nutrient deficiencies; and alternaria leaf spot and *Pseudomonas syringae* pv. *syringae*, when plants are young.

In addition, interactions between pathogens on the same plant part often occur, which may complicate efforts to evaluate the importance of either and to evaluate for resistance. For example, commonly interactions exist between ALS and FLS; CBB, FLS and web blight; and CBB and *P. syringa* pv. *syringae*.

The importance of obtaining not only species identification, but also race information, was mentioned. It is particularly important for effective breeding programs that good information is available on pathogens with known vertical variability such as *C. lindemuthianum*, *P. syringae* pv. *syringae* and *U. appendiculatus*.

Africa does not host all pathogens which are present in Latin America, the centre of origin of bean. Some pathogens are found in Africa which are not known in Latin America. An example is scab. More commonly there are a number of diseases which occur in Latin America, but not in Africa. For example, grey leaf spot and viruses like bean golden mosaic.

Corynebacterium was also noted as an example of a pathogen which had not been reported officially from Africa, although unofficial reports indicate that it has been found in Kenya. However, this appears to be incorrect, as apparently the pathogen was isolated in Uganda and certified by CMI.

For pathogen identification, CMI (Ferry lane, Surrey, England) or equivalent institutes could be used to verify samples, perhaps at a reduced cost for bulk payments. For viruses, collaborative projects could be used, such as the one with Dr. Vetten in Braunschweig (see list of participants). Perreaux mentioned that there were plans to set up a virus laboratory in Burundi and Allen mentioned that CIP also had plans to set up a regional virology lab in Africa.

There followed a debate on the practicality of pathogen check lists. It was mentioned that identification of the organisms that limit production was the basis of efforts to reduce the problem. It was stressed, however, that national programs minimize the time spent on pathogen lists. It was proposed that each region should have a coordinator responsible for the central recovery of lists from individual scientists. Allen would be general coordinator. No funds would be put into place until the next workshop.

SURVIVAL AND SPREAD

Chairperson: Pyndji Mukishi
Rapporteur: Joseph Kayitare

Angular leaf spot

The survival of disease inoculum depends on the environment but, in general, the pathogen survives for six months in Colombian conditions. In Uganda, angular leaf spot has survived up to nine months on debris in the laboratory, seven months on debris kept outside and two months on debris incorporated into soil. A study carried out in the USA showed a survival of two years. In general, in tropical conditions the angular leaf spot pathogen can survive for six months.

The disease is spread by the use of infected seed and by crop residues left in the field. The transmitting agents are wind, water and rain. In the case of Uganda, where beans are grown continuously, inoculum is permanently present. The disease is more severe in association with either cassava or maize than in pure stands.

Anthracnose

The pathogen is most dangerous in temperatures of 17-22^{o}C. It survives on leaves, stems or any other plant material which fall on the ground and act as inoculum for subsequent seasons. The disease is spread by infected seed. The transmitting agents are wind, rain and running water.

The maize:bean association favours the disease because of the high humidity which is created. High temperature and humidity are the main factors favouring the disease.

Rust

Rust is the third most important disease of phaseolus in the world. Its origin has not been identified for it is found as much in America as in Africa. Rust has five development forms (spores). Most of the spore forms, especially urediospores, prefer humid and cloudy weather. These forms do not survive for more than a year. The disease is spread by wind, animals and, to a small extent, insects.

The teliospore form varies in importance from region to region and season to season. For the teliospore form, which is tropical, there are no other leguminous plants which serve as a reservoir for bean.

The spread of this fungus can be prevented by bean:maize association and by mixing varieties.

Ascochyta

The pathogen survives on crop residues. It disappears rapidly once residues are incorporated but remains for long periods on residues in the open air.

In European conditions, the disease is transmitted by contaminated seed. For an epidemic to spread from one plant to another, high humidity is necessary. To prevent the spread of the disease by the seeds, they can be treated with Thiram, Captan and Benomyl. The disease is rapidly eliminated when it is on the leaf surface.

There are no sources of resistance according to the information from Munyemana who has found that climbing varieties are more resistant. It is difficult for farmers to find fungicides.

Common bacterial blight

The pathogen survives on crop residues and host plants; occasionally insects favour its dispersal. The pathogen can survive on seeds for 15 years. In temperatures of 18-22°C, the pathogen can survive for two years; below 18°C, it can survive up to five years. The survival time of the pathogen is inversely proportional to the depth at which the residues are incorporated into the soil. For central Africa we have no information about the survival of the bacterium. Rapid decomposition of plant residues reduces the inoculum in the soil. Hass (1972) has demonstrated that the bacterium survives on primary leaves and disappears on trifoliolates. The bacterium survives for short periods only in the soil. The bacterium can survive on other *Phaseolus* spp., pea, soybean and cowpea. Transmitting agents include seeds, wind and rain. As little as 0.5% of infected seed can produce a devastating epidemic.

Miscellaneous

Implications of common bacterial blight and halo blight control.

- Treating seeds with streptomycin prevents bacterial attack.

For the Great Lakes, no one is working on survival and distribution of pathogens.

- Unburned crop residues act as reservoirs for pathogens. To resolve this problem, good composting is necessary to reach 60°C, which kills pathogens.

The overpopulation of Rwanda and Burundi, which does not allow good rotation, increases the amount of inoculum in the soil.

- The epidemiological study of diseases is purely classical and should be carried out by university students.

PATHOGENIC VARIATION

Chairperson: Wilson Msuku
Rapporteur: John Taylor

All pathogens vary in a range of characters, including cultural characteristics, sporulation rates, spore morphology and colour and so on. Crucial variation is in virulence (vertical pathogenicity), which is demonstrated by a clear interaction between groups of isolates (physiologic races) and cultivars. If there is no clear interaction, isolates vary in aggressiveness (horizontal pathogenicity), not virulence.

These differences are important, not mere semantics, for their implications for breeding are quite different. It is with pathogenic variation that this session is concerned, of the vertical type in the cases of angular leaf spot (ALS), anthracnose, rust and halo blight (HB) and the horizontal type in the cases of common bacterial blight (CBB) and ascochyta blight.

The other aim is to consider the extent to which resistance breeding influences evolution of pathogenic variants. For example, what effects, if any, have been the recent success in raising the level of resistance to CBB by interspecific hybridization? Will physiologic races emerge or merely more aggressive races?

Angular leaf spot

Assessment of ALS on farm indicates varietal variation indicative of vertical pathogenicities although so far little detailed investigation has been made. Similar isolates have been obtained throughout the Great Lakes Region (e.g. Rwanda and Zaire). The occurrence of races suggests the need to develop multiple resistant varieties.

Anthracnose

In the field, there is well-defined varietal variation. In different locations, reactions can change. Calima and BAT 93 show different reactions in Brazil and Mexico (vertical resistance). Categorization of races in Europe using differential varieties did not coincide with observations in Latin America and Africa. In Mexico, alpha, beta and gamma races were present as well as Mexico 1 and 2 and Brazil 1 and 2. Some new races will overcome the resistance of the widely used resistance source, Cornell 49242.

There is need to use standard varieties for differentiation of races. CIAT proposed that only widely used varieties be used (16 varieties separating isolates into 16 groups) to allow comparison among different localities. In Kenya, these are isolate groups 12 and 4, while in Zaire they are groups 12 and 6. This system has been adopted in Brazil. Pathogenic variants in Africa differ from those in Latin America and Europe. CIAT strategy has involved evaluation of 35 thousand bean accessions in the field at CIAT and advancement of resistant materials to other localities.

Rust

Extensive studies in many countries indicate a complex races structure. Similarly, races have been recorded in Africa (Tanzania, Uganda, Kenya and Malawi), although there has been no detailed survey throughout the African continent.

Bacterial blight

Resistance and pathogenic variation are two aspects of the same phenomenon. Variation in pathogenicity may be due to plant age, inoculation method and environment. Standardization of methodology for the evaluation of pathogenicity/plant resistance is needed. There is no evidence, as yet, for races within *Xanthomonas campestris* pv. *phaseoli*.

Ascochyta blight

Several fungi are implicated, causing confusion in taxonomy. Early descriptions of *Ascochyta phaseolorum* and *A. boltshauseri* are incorrect. They were of *Phoma exigua*, which occurs on many hosts, for example, potato. On bean, *P. exigua* var. *diversispora* is the main problem in Africa. It shows a slower growth in agar than *S. hortensis*. In South America, *Phoma* spp. also cause ascochyta blight. These fungi can be distinguished in culture as well as from their symptoms on plants. *P. exigua* var. *diversispora* causes blackening of the leaf pulvinus and at the junction of the petiole (black node). Leaf blight appears before black node, in which the growing point becomes completely blackened. *Stegonosporopsis hortensis* does not kill the growing point nor does it cause leaf blight. In South America, *Phoma* spp. cause leaf blight and black node. There is no evidence for variation in virulence with var. *diversispora*.

Halo blight

Two races were recognized formerly in Europe and North America. There have been many studies in limited geographic regions but not extensive in Africa or Latin America. Variation in the halo blight pathogen (*Pseudomonas syringae* pv. *phaseolicola*) is both qualitative (vertical pathogenicity) and quantitative (horizontal pathogenicity). Three races have now been identified in Africa. The previously known races (1 and 2) were widely present.

Race 1 in particular is widely distributed in leguminous plants of eight different genera (crops and weed species). Race 2 is also widespread, with a pigment producing variant, present in Tanzania and Kenya, being particularly aggressive. Race 3 was predominant in many areas, especially in Central Africa (Rwanda, Burundi, Zaire and Uganda). Race 3 is found only in Africa. A possible fourth race, which attacks soybean and some varieties of *Phaseolus vulgaris* was identified as *P. syringae* pv. *glycinae*. Major gene resistance is available to races 1 and 3 while a small number of varieties show race non-specific (quantitative) resistance.

Research activities, future needs and activities.

Angular leaf spot studies are required to determine variation within Africa. Agreed to proceed with nurseries for the introduction of material across regions including known resistant material (from Malawi, Zambia, Tanzania, Ethiopia and Uganda), BALSIT too big. Should be a smaller African BALSIT of 50 entries including key susceptible varieties. Pastor-Corrales to give guidance on entries to be included. There is clear evidence for races of anthracnose in Africa (David Allen). Isolates in Colombia not currently able to distinguish resistant materials specifically for Africa. Pastor-Corrales has classified Colombian isolates. Jeremy Davis will compare them with isolates from the Great Lakes region. It may be possible to use mixed South American isolates in Colombia to give a similar spectrum to African isolates. This will aid selection of germplasm at CIAT. There are some sources of broad resistance.

The survival of anthracnose resistance in Cornell 49242 in Europe is dictated by a clear seed policy (Thijis Gerlagh). Major genes from Cornell 49242 and other sources work in most parts of Africa and can be incorporated by a backcross programme using two genes at a time (for example, the Cornell gene + 1 other). Barry Smithson suggested regional sub-project to organize nursery based on best current knowledge.

Rust is the most variable of all pathogens. There is evidence of races in Africa. Margaret Mmbaga (Tanzania) is interested in race identification. Habtu Assefa (Ethiopia) believes more information is required to establish a project. It was suggested that they should develop a bean rust nursery for Africa with differentials based on IBRN. According to Pastor-Corrales, the characterization of races alone may not be the best approach. Rust is a cyclic problem in which major epidemics may occur every four or five years (for example, the major disaster in Cuba). Uganda (Sophie Musaana) has rust as a priority disease.

Common bacterial blight is a priority in Burundi (Dominique Perreaux) and Uganda (Fina Opio). There is no evidence of vertical resistance so resistance should be assumed to be horizontal and the most aggressive isolates used for screening. There is need to understand the fundamental aspects of pathogenicity.

With ascochyta, there is good correlation between African and Latin American, with materials performing the same in both areas. No evidence of pathogenic variation.

Betty Gondwe confirmed that races 1 and 2 of the halo blight pathogen are widespread in Tanzania and that race 3 is present only in the region of Great Lakes. Race 2 (brown pigmented type) is more aggressive and found in northern and southern Tanzania. There is specific resistance to races 1 and 3 and quantitative (race non-specific) resistance to all races, including race 2. The combination of race specific resistance with quantitative resistance is likely to provide the most durable disease control.

Chairman's summary of future needs

To continue research programmes on ascochyta blight at Wageningen and on halo-blight at Wellesbourne. Rust races sub-project to be set up by Margaret Mmbaga in Tanzania. Future of angular leaf spot not concluded.

SOURCES OF RESISTANCE FOR AFRICA

Chairperson: Jeremy Davis
Rapporteur: J. Kannaiyan

Angular leaf spot

There are several sources of resistance for Latin America but few for Africa. They include Tanzania (9) and Kenya (2). Twenty entries in the BALSIT have maintained resistance for five seasons in the Great Lakes. Their resistance has held up in Zaire, Rwanda and Zambia. Some varieties in mixtures show resistance in Zaire and more work is in progress.

Pastor-Corrales cautioned that resistance must be sought in local materials since they will be better adapted.

It was recommended that regional nurseries including local checks should be conducted at: Kawanda, Uganda; Mbala, northern Zambia; Bunda, Malawi; Uyole, Tanzania; Jima, Ethiopia; Mulungu, Zaire; and Karama, Rwanda.

Anthracnose

A well known pathogen with considerable pathogenic variability. There is little information on resistance in Africa. Six isolates belonged to three groups: Kenya, Zaire, Tanzania - Beta; Zaire, Burundi - Brazil I; and Kenya - Mexico II

At CIAT, three varieties from Rwanda were found resistant to all races but most were resistant to some races. Theresa Sengooba noted that several races are not known in Uganda or in Africa in general.

Suggested nursery locations are: Mbala, northern Zambia; Kachwekano, Uganda; Rubona, Rwanda; Ambo, Ethiopia; and Uyole and Lyamungu in Tanzania.

Rust

Another well known pathogen with evident pathogenic variation. Six races are known from Tanzania. Local sources of resistance have been identified in Africa. Other sources are available from CIAT. Peter Trutmann observed that rust was not much problem in mixtures. Multilines could be taken into consideration. Rust is widespread and sources of resistance to other diseases must be tested for rust.

A nursery to confirm resistance across locations should be considered and a Regional Sub-project was proposed for Tanzania, subject to Steering Committee approval. Tanzania and Ethiopia will be possible participants.

Ascochyta blight

Not well-known. Resistance is undocumented. At CIAT, genotypes with intermediate reactions are available but no immunity is reported. Good levels of resistance are evident in climbing bean but only low levels in bush bean. High resistance is reported in *Phaseolus coccineus*.

At CIAT, it is hoped to increase levels of resistance by crossing resistant sources and transfer resistance into local African cultivars. Yield nursery for *P. coccineus* is available.

There is a regional project in Uganda. Other proposed locations are: Rwerere, Rwanda; Mbala, Zambia; and Kachwekano, Uganda

Bacterial blight

Most important in lowlands. Sources of resistance known but methods used questionable. Methodology must be standardized.

EVALUATION AND SCREENING TECHNIQUES

Chairperson: Talo Pastor-Corrales
Rapporteur: Julia Kornegay

1. W. Msuku questioned the use of isolate mixtures because of competition, cross protection and inoculum dilution. MAPC said that these problems had been reported but experience at CIAT using mixtures of up to five isolates of *Colletotrichum lindemuthianum* had given good results. Cross protection can occur but usually it is due to different inoculation times with isolates of

different virulence. The susceptible cultivar is first inoculated with a non-pathogenic isolate and subsequently with pathogenic ones.

2. Margaret Mmbaga emphasized the importance of spreader rows, especially to eliminate escapes within nurseries. Artificial inoculation may not be negative and, in fact, may simulate inoculation from nearby fields.

3. David Allen stressed the need to balance inoculations to get adequate but not excessive disease pressure, which reduces differences between resistant and susceptible reactions.

4. M. Gerlagh questions whether a single virulent isolate may be preferred over mixtures in certain cases. MAPC said that mixtures are useful to identify broad resistance.

5. CIAT's publication "Standard System for the Evaluation of Bean Germplasm" provides a useful summary of rating methods.

6. Dominique Perreaux mentioned that resistant and susceptible reactions may be expressed within the same plant especially for CBB.

7. T. Sengooba sought advice on training technicians to evaluate nurseries uniformly. MAPC explained how the training is conducted at CIAT.

EPIDEMIOLOGY IN PURE STANDS AND VARIETAL MIXTURES

Chairperson: Dominique Perreaux
Rapporteur: J. Kannaiyan

The chairperson started the session with the following remarks:

Bean is frequently cultivated in mixtures, which reduces the impact of diseases. Growing resistant and susceptible genotypes in mixtures will reduce the rate of multiplication of pathogens resulting in less disease in mixtures than in pure stands.

Peter Trutmann reviewed the general literature on the subject. Small farmers uses mixtures commonly to reduce the risk of diseases and other factors. It is essential to understand the system well to derive greater advantages from it. Mixtures may reduce the overall disease of the components and increase yields. He also stated that intercropping and mixtures simulate the same mechanism. Mechanisms of disease reduction and proportions of resistant and susceptible components in mixtures were also discussed.

Jeremy Davis discussed his student's work on rust disease severity in mixtures of resistant, intermediate and susceptible genotypes. He also mentioned the possibility of improving local mixtures by introducing appropriate components. In general, mixtures of susceptible and resistant components had yield advantages over mixtures of resistant components alone. It is difficult to separate the physiological and pathological effects of mixtures on yield in bean.

Pyndji Mukishi reviewed the importance of the mixtures in reduction of diseases in the Great Lakes Region. Most farmers in the region grow mixtures

rather than pure stands to avoid a number of risk factors.

Participants from Uganda, Tanzania, Zambia, Malawi, Rwanda, Ethiopia and Burundi also discussed the subject.

Jeremy Davis pointed out the main difficulties in mixtures - seed production and export. There is no research work on mixtures in most countries.

Workshop participants agreed in principle to encourage mixtures in small farmers' fields to avoid risk factors like diseases, insects and drought.

Pastor-Corrales pointed out the importance of maintaining mixtures of the same seed colours and sizes and maturity, like multilines to retain genetic diversity. The best cultivars may be included as recurrent components of mixtures. Bulk breeding may also help to maintain mixtures.

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