BREEDING FOR DISEASE RESISTANCE TO THE MAJOR FOLIAR PATHOGENS OF DRY BEANS (*Phaseolus vulgaris*) IN SOUTH AFRICA

by

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ABSTRACT

Resistances to bean common mosaic virus, halo, common and Ascochyta blight, angular leaf spot, anthracnose and rust pathogens of beans in South Africa were combined by reverse dichotomous crossing. Full resistance to Uromyces appendiculatus from Carioca 80 was conditioned by a single dominant gene. Partially dominant resistance to Phaeoisariopsis griseola was conditioned by a single gene in Carioca 80 and two genes in PAI 127. Differences in aggressiveness of isolates of Phoma exigua var. exigua were found. Different levels of Ascochyta blight resistance were found in the glasshouse, but field testing showed little difference after flowering.

Inoculations of differential cultivars indicated the presence of at least eight races of U.appendiculatus and the α-Brazil race of Colletotrichum lindemuthianum. Inoculations of the old set of halo blight differential cultivars identified races 1 and 2.

Forty-five lines with partial resistance to rust were obtained by recurrent selection. Very highly significant differences were noted between ratings of percentage leaf area affected by rust and yield of 23 cultivars planted in field trials. Significant genotype x environment interaction was noted for rust ratings. Ratings at different dates within a trial were correlated with one another, showing few ratings are required per trial, and a correlation of -0.678 between yield and rust rating was found.

Inheritance of partial resistance and improved yield of eight cultivars crossed in a full diallel was mostly due to additive effects but non-additive effects were also very highly significant. Reciprocal effects were not significant for yield and rust ratings. Genotype x environment interactions were significant for rust ratings and yield. High estimates of narrow-sense heritability for rust resistance were obtained.

No relationship between resistance and time to flowering, pustule size, leaf hairs and stomata was found. Latent periods in unifoliate leaves did not correlate with resistance but a closer match was found in the fourth

trifoliate leaves. Inoculations with three additional single-pustule isolates of the 23 parent cultivars indicated the cultivars had similar levels of resistance.

Ring necrosis was found in nine cultivars or crosses with them. The ring reaction was conditioned by a single dominant gene and possibly by the epistatic interaction of two dominant genes in Carioca 80. Differences in symptom severity in plants derived from Epicure indicated the possibility of additional gene interaction.

DECLARATION

I hereby declare that the information presented in this thesis is the result of my own work unless otherwise indicated.

B.R.Edington

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LIST OF ABBREVIATIONS

The following is a list of abbreviations used in this thesis:

BCMV	Bean common mosaic virus
BH	Brown haricot
BlCMV	Blackeye cowpea mosaic virus
CIAT	Centro Internacional de Agricultura Tropical
CV	Co-efficient of variation
cv(s)	Cultivar(s)
dia	Diameter
DC	Diallel cross
DF	Degrees of freedom
DL	Dwarf lethal
DMRT	Duncan's multiple range test
dpi	Days post inoculation
GCA	General combining ability
GN	Great northern
HBR	Halo blight resistant
IBRN	International bean rust nursery
LSD	Least significant difference
LWK	Large white kidney
MS	Mean sum of squares
NC	Natal cross
NS	No significance at the 5 % level of confidence
OMA	Oatmeal agar
OPSC	Oil and Protein Seed Centre
p	Probability level
PC	Potchefstroom cross
PI	United States Department of Agriculture plant introduction number
PLS	Painted lady sugar
RC	Rust cross
r.h.	Relative humidity
RSS	Red speckled sugar
SC	Second cross
SCA	Specific combining ability
SPI	Single pustule isolate

SS	Sum of squares
SWC	Small white canning
TC	Third cross
TMV	Tobacco mosaic virus
UI	University of Idaho
UNP	University of Natal, Pietermaritzburg
VR	Variance ratio
YH	Yellow haricot
*, **, ***	Significant at the 5 %, 1 % and 0.1 % levels of confidence, respectively.

The first mention of a disease is followed by its causal organism in brackets.

CHAPTER ONE

INTRODUCTION

Beans are an important crop in temperate to tropical regions. In developed countries they are more important as a vegetable whereas in developing countries they are grown more for use as a dry pulse for human protein consumption. In 1990, production of dry beans (all types) in developed and developing countries was 2 427 000 and 13 867 000 t and for green beans it was 1 524 000 and 1 612 000 t, respectively (Anon., 1991a).

In 1991, 89 539 t of common dry beans (Phaseolus vulgaris L.), 9 488 t of large white kidney beans (Phaseolus coccineus L.) and 93 t of tepary beans (Phaseolus acutifolius A.Gray) were produced in South Africa for marketing, with a total value of ca. R120 million (Anon., 1992). Several yields in past years have been severely affected by drought and accompanying high temperatures. In 1983, total South African production was only 26 879 t and in 1984, 47 332 t (Anon., 1988a). The large quantity produced in 1990, 108 355 t, led to lower relative prices for beans which in turn led to a higher local consumption of 80 366 t (Anon., 1992). In spite of a slightly lower yield in 1991, consumption reached yet another record high. This was partly due to more aggressive promotion of dry beans by the Dry Bean Board. Consumption formerly oscillated about 70 000 t. There is some international trading in beans. In 1990, 30 139 t were exported but in 1991 only 13 139 t were exported due to an adequate supply of beans on the world markets. Of the 1991 exports, 6201 t were supplied to "foreign" countries and 7695 t were supplied to "neighbouring" countries (Anon., 1992).

According to Anon. (1991b) production during 1990/91 season was hampered by unfavourable climatic conditions during planting time and drought prevailing in much of the country. Of all the dry bean types, more than 42 % of the production was from the Eastern Transvaal Highveld, especially in the Delmas area, and 62 % was produced in the Transvaal. The Orange Free State produced 34 %, mainly in the eastern regions, and the Cape Province produced 3 %. Production in Natal has increased in the past from 0.3 % in 1982 to 4.5 % in 1985, but in 1991 the figure was only 1.5 %. An increase in the planting of beans in 1990 compared to 1989 (yield of 85 122 t) was primarily

due to farmers looking for alternate crops to maize. In 1991 there was a slight reduction of 2 % in plantings compared to 1990.

Dry Bean Board production figures are only for beans which pass through accredited marketing channels and hence actual production in South Africa will be higher. Lea and Stanford (1982) found maize and beans to be the most important arable crops in peri-urban areas after surveying black farmers in KwaZulu. These beans do not usually pass through normal marketing channels. Perhaps the recent reduction of the percentage of beans being produced in Natal according to Dry Bean Board figures is only a record of the reduction of beans passing through accredited dealers.

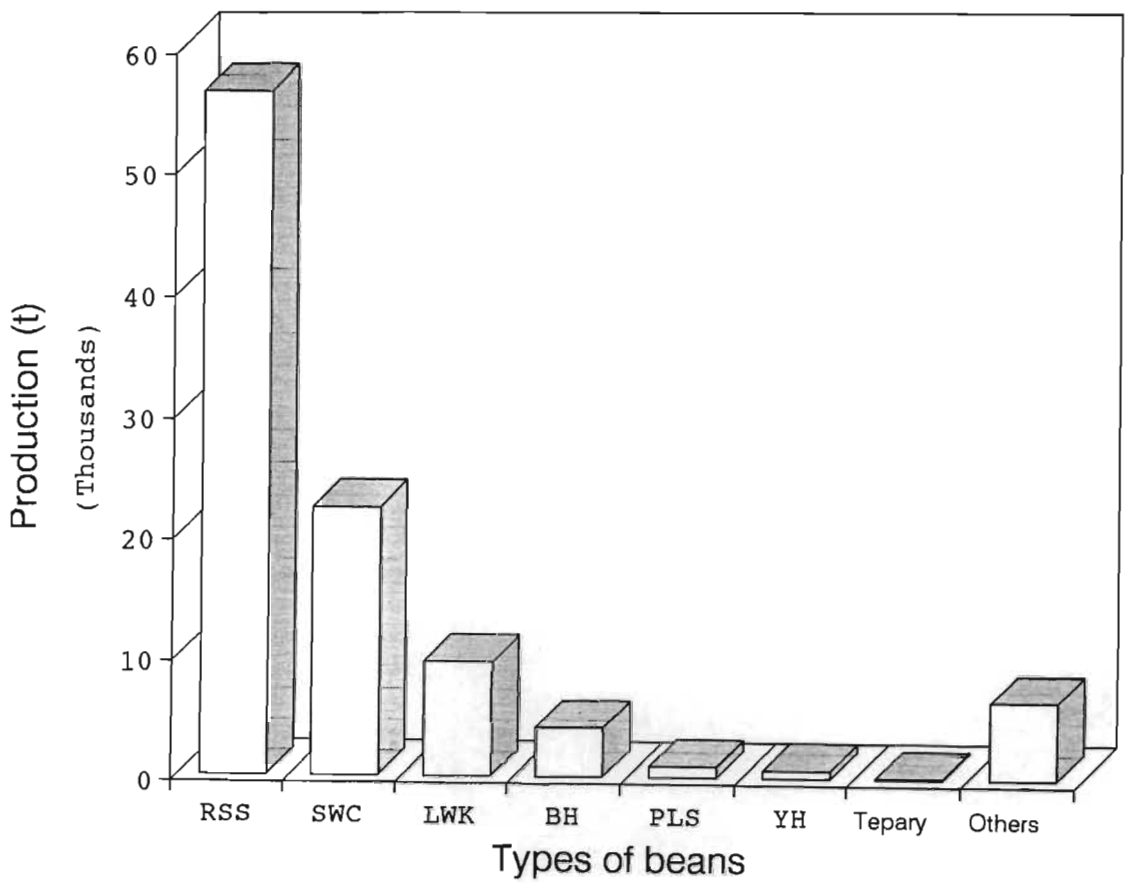


Figure 1. The types of beans produced in South Africa in 1991.

RSS = Red speckled sugar
SWC = Small white canning
LWK = Large white kidney
BH = Brown haricot
PLS = Painted lady sugar
YH = Yellow haricot

Production of the various types of dry beans (Figure 1.) depends largely on prices paid for them and ease of cultivation. Small white beans (22 % of the 1991 total, 22 224 t, with an average price of R979 t^{-1}) are mostly used in the canning industry (Anon., 1992). Other types of beans are usually sold loose in the packaging trade. Production of large white kidney beans dropped from 46.8 % (35 761 t) of the total in 1981 (a favourable year) to 14 % (3757 t) in 1983 because of drought (Anon., 1988a). In 1990, a favourable year, production of large white kidneys was only 14 % (14 828 t) and in 1991, 10 % (9488 t) of the total with an average price of R1038 t^{-1} showing a drop in consumer preference (Anon., 1992). Production of red speckled sugar beans reached yet another record high in 1990, with 57 110 t (53 % of the total) being produced and an average price of R1346 t^{-1}, showing the high consumer preference for this type (Anon., 1991b). In 1991 red speckled sugar bean production was slightly down to 56 369 t and the price was also slightly down to R1327 t^{-1} compared to 1990 (Anon., 1992). The relative area under speckled sugar bean cultivation was even greater as the small white canning beans tend to yield 200 to 300 kg ha^{-1} more.

Early bean breeding work in South Africa has resulted in the release of several improved small white canning cultivars. Unfortunately, it is only recently that a possible export market for this type of bean has developed due to the low value of the rand and lifting of sanctions. The class of bean most favoured by the South African consumer as evinced by the high price is the red speckled sugar bean. Hence the increased input by seed companies into producing improved red speckled sugar bean cultivars.

Bean production in South Africa is hindered by several economically important diseases. Of the foliar pathogens, bean common mosaic virus (BCMV), common blight (*Xanthomonas campestris* pv. *phaseoli* (Smith) Dye), halo blight (*Pseudomonas syringae* pv. *phaseolicola* (Burkholder) Young *et al.*) and rust (*Uromyces appendiculatus* (Pers.) Unger) are of national importance and angular leaf spot (*Phaeoisariopsis griseola* (Sacc.) Ferrais), anthracnose (*Colletotrichum lindemuthianum* (Sacc., *et* Magn.) Scrib) and Ascochyta blight (*Phoma exigua* var. *exigua* Desmazieres) are only, at present, of local importance in Natal, Republic of South Africa (Melis, 1987a). The more humid conditions of Natal favour disease-spread. Disease losses depend on initial inoculum levels and climatic conditions favouring

disease spread.

The importance of these diseases, except rust, is mostly due to the fact they can be seed-borne. Problems with contaminated seed, particularly with the bacterial blights, has led to the development of a certified disease-free seed scheme in South Africa (and also in other countries). It has been very successful barring that not all growers use certified disease-free seed and only seed of the major cultivars is available under this scheme. With the exception of BCMV and rust, the diseases can also be transmitted from one season to the next in plant debris and therefore failure to rotate will allow disease build-up. Other agronomic practices such as fungicide use will also hinder disease development.

The lack of disease control by agronomic practices is most prevalent amongst subsistence growers and they will benefit greatly from increased stability of yield from resistant cultivars. Increases in yield and reduced input costs will also improve profitability of larger-scale growers.

The purpose of this study was firstly to produce a line with resistance to all the major foliar pathogens of beans in South Africa. The line was to be produced in the glasshouse with the aim of producing material for field selection by a commercial seed company. The second aim was to produce a cultivar or breeding line with horizontal resistance (sensu Van der Plank, 1963) to rust, the most important pathogen of beans in South Africa. In both cases, improvement of a large-seeded, red speckled sugar bean type for use by a commercial seed company either as a cultivar or as source of resistance in crossing programmes was the goal. In addition, the slightly incompatible reaction was investigated. Initially it was thought to be a possible source of horizontal resistance and was later studied more as an academic exercise as it became obvious that it was a form of vertical resistance.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

The purpose of commercial plant breeding is the genetic improvement of a crop in order to increase its economic return for the grower. This can be done by increasing yield or quality and/or reducing input costs. In this respect, breeding for disease resistance is no different.

Disease can often be controlled by other means such as pesticide use, but plant resistance to a disease has the advantage in that it requires no contribution by the grower and is "enviro-friendly". The only involvement of the grower is in the initial selection of a cultivar. If a cultivar has failed due to poor disease resistance he is unlikely to use it again if there is a better option. But many subsistence growers do not have the resources to withstand a crop failure, death or relocation may result as a consequence. What they require above all else is yield stability. All the diseases considered in this study have a large destructive potential and can cause yield instability.

The type of disease resistance used in a cultivar depends to a large extent on what is available and how easily it can be incorporated into an economically viable cultivar. With dry beans there is a high consumer preference for certain seed characteristics and this must be borne in mind when producing a new cultivar. For example, with the speckled sugar beans a larger seed size that can be spherical to cylindrical, but not kidney-shaped, is preferred; the background colour must be white or cream and the speckle a dark pink to maroon colour. The most commonly grown speckled sugar bean cultivar at present is Bonus and it is susceptible to all the diseases below. Sources of resistance used in this multiple-disease resistance study are from cultivars with small- or medium-sized seed of various combinations of colours and speckles.

Various labels have been ascribed to types of resistance depending on what aspect is examined (Buddenhagen and de Ponti, 1983). From a genetical

point of view it is often termed monogenic, digenic, etc. or oligogenic, i.e. one or few genes involved or polygenic when an unknown number of genes are involved. Each gene may have a major or minor role in the resistance. From a pathological point of view, the degree of resistance is often termed immunity (no apparent symptoms), full resistance or partial resistance depending on amount of pathogen reproduction. A plant may be tolerant if it supports a high level of pathogen reproduction with little apparent damage or yield loss. From an epidemiological point of view, resistance is often termed vertical or horizontal whilst others favour the terms transient and durable (the two sets of terms do not necessarily overlap). A plant breeder must have a holistic approach to resistance as the pathosystem does not impose classification systems upon itself.

A breakdown of resistance could lead to loss of income and hence durable resistance would be favoured by growers. Gene for gene interaction of vertical resistance can be very transient against highly variable and mobile pathogens such as the rusts. But vertical resistance is far easier to manipulate especially when one considers it will be only one of several desirable factors in a new cultivar. Horizontal resistance that is polygenically inherited may require several cycles of recurrent selection to arrive at a desired level of resistance in association with acceptable agronomic and seed size, shape and colour characteristics. Hence, in this study where a large number of disease resistances were being combined, vertical resistance (if available) was used but where resistance to rust alone was considered, horizontal resistance was the goal.

2.2 The diseases and sources of resistance

2.2.1 Bean common mosaic virus

Bean common mosaic virus (BCMV) has been reported in nearly every country of the world (Gálvez and Morales, 1989) and has a great potential to reduce yields. Hampton (1975) indicated that yield losses of up to 70 % from plants with severe symptoms were possible. Lockhart and Fisher (1974) reported growers' estimates of losses of about 50 % in Morocco. It is readily aphid-transmitted and from season to season in infected seed (Bos,

1971). Rates of seed transmission up to 86 % have been found (Medina and Grogan, 1961). A 100 % infection rate of plants was obtained with a high aphid infestation from a seed source initially only 2 to 6 % infected (Gálvez and Morales, 1989).

Resistance to BCMV is available as a series of recessive genes or a dominant gene. The recessive genes require a strain non-specific gene (bc-u) to operate and two genes (bc-1^1 & bc-1^2 and bc-2^1 & bc-2^2) confer strain-specific resistance whilst bc-3 confers strain non-specific resistance (Drijfhout, 1978). The dominant gene (I-gene) was discovered by R.D. Corbett in a single plant in a stand of a susceptible green bean cv. Refugee (Pierce, 1935). After extensive use of the I-gene, systemic necrosis was noted by Jenkins (1939) and reported as a separate disease based on symptomatology (Jenkins, 1940). The systemic necrosis is known as "black root" and its occurrence depends upon environmental/host/strain interactions. If infection occurs with a strain that can induce "black root" in a plant containing the I-gene, without recessive gene resistance, the initial veinal necrosis can quickly spread to the growing point and thereafter kill the whole plant. The I-gene inhibits viral multiplication and, as a result, there is no plant to plant transmission within the field or season to season transmission in the seed.

The strain-nonspecific gene bc-3 has only recently become available and, in breeding for resistance to BCMV in South Africa, use has been made of the I-gene (except for the cv. Teebus, which has recessive gene resistance). Incorporation of a single dominant gene into a new cultivar is easier than the incorporation of two recessive genes (bc-u and bc-3). The prevailing isolate of BCMV in the Transvaal and Natal is of virus pathogenicity group V (Edington & Whitlock, 1988) and can cause temperature-dependent necrosis when temperatures rise above 27°C after infection of I-gene containing plants. Van Rheenen and Muigai (1984a) suggested using the I-gene for resistance breeding in Kenya where a temperature non-dependent necrosis inducing strain is present. At present, most of the production of large-seeded types is from susceptible cultivars, but new cultivars with I-gene resistance are becoming increasingly available. All major green bean cultivars have the I-gene and if all future cultivar releases have resistance to BCMV, it will no longer be an economically important disease.

A problem that may occur in the use of cultivars containing the I-gene is that necrosis ("black root") can be induced by four other potyviruses, namely blackeye cowpea mosaic virus (BlCMV), cowpea aphid-borne mosaic virus, soya bean mosaic virus and watermelon mosaic virus (Kyle and Dickson, 1988) and also by the bean severe mosaic virus complex (Kornegay, 1992). Another problem that may occur in breeding for disease resistance is the linkage of resistance to one pathogen to susceptibility to another. Morales and Castaño (1992) found increased symptom severity of cultivars containing the I-gene after infection with certain comoviruses. Fortunately, comoviruses are not known to cause a major disease of beans in South Africa at this stage.

McKern et al. (1992) have recently proposed that BCMV consists of two distinct potyviruses based on serology and composition of protein coat. Strains that cause necrosis in I-gene containing plants independent of temperature were given the suggested name of bean necrotic mosaic virus. Remaining isolates of BCMV, presumably including the South African isolate as other members of pathogenicity group V are in this section, retain the name BCMV. The new BCMV group also includes BlCMV, azuki bean mosaic virus, peanut stripe virus and three isolates of potyviruses from soya beans.

2.2.2 Common Blight (Xanthomonas campestris pv. phaseoli)

The two bacterial diseases, halo and common blight, are similar in many respects. Their epidemiologies are very similar except for effect of temperature. Common blight (Xanthomonas campestris pv. phaseoli (Smith) Dye) is a warmer weather disease and halo blight cooler weather. Both are seed-borne and carried over in plant debris. Once an infection locus is established, the pathogen can spread rapidly by raindrop dispersal or mechanical transfer (Saettler, 1989; Schwartz 1989b). Hail storms and movement by machines, etc. within a wet canopy are effective means of spread.

The economic value of loss caused by the separate bacterial diseases can be difficult to assess as there is often a dual infection (Zaumeyer and Thomas, 1957). Wallen and Jackson (1975) reported a yield loss of 38 % after inoculating beans with the common blight pathogen at the 3 to 4 leaf stage.

They also reported a loss of 1252 t due to common blight in the state of Ontario alone in 1970. Yield losses estimated at 22 % and 45 % from natural and artificial infections, respectively, were reported by Yoshii et al. (1976) in Colombia. Economic loss due to both blights is exacerbated by discolouration of remaining seed and resultant reduction in quality. The two blights also have no effective economic means of chemical control; copper fungicides prevent further spread rather than eliminating the pathogens (Coyne and Schuster, 1983; Schwartz, 1989b; Nevill and Parsons, 1991). This makes breeding for resistance a very favourable option. Doidge reported the presence of common blight in South Africa in 1918.

The distinguishing symptom of common blight is that after inducing the initial water-soaked lesion, the bacterium causes necrosis of the leaf tissue with a small chlorotic margin (Saettler, 1989). Halo blight also causes the same symptom in older leaves, particularly in warmer, drier conditions, when the halo region becomes necrotic, but typical halo symptoms should still be visible in younger leaves.

The common blight pathogen does not have pathogenic specialization in common beans per se, although there are differences in levels of pathogenicity. The cv. Great Northern Nebraska No.1 Selection 27 is resistant to North American isolates but more susceptible to Colombian isolates of the bacterium (Coyne et al., 1963; Schuster and Coyne, 1971; Yoshii et al., 1978). Zapata and Vidaver (1987) reported pathogenic specialization in tepary beans (P.acutifolius) based on qualitative (hypersensitivity) and quantitative reactions in the pods and cotyledons of five cultivars. Zaiter et al. (1989b), however, indicated a situation in tepary beans similar to that of common beans. Isolates also differ in levels of virulence (Saettler, 1989). A variant of common blight, fuscous blight, was previously separated on the bacterium's ability to produce melanin on media containing tyrosine, but the variant is no longer considered important. Boelema (1967) in noting the presence of fuscous blight in South Africa suggested it would be of great importance as the cv. Seminole was resistant to halo and common blight but not fuscous blight. Burkholder and Bullard (1946) found the cv. Great Northern Nebraska No. 1, from which Great Northern Nebraska No. 1 Selection 27 is derived, to be one of the more resistant cultivars of the 40 they tested with fuscous blight.

Resistance to common blight has been reported in tepary beans (Rands and Brotherton, 1925; Schuster, 1955; Coyne et al., 1963; Coyne and Schuster, 1973) and recently reported as due to a single dominant gene after crosses between tepary lines (Drijfhout and Blok, 1987). Honma (1956) made crosses between tepary beans and the common bean cv. Great Northern, culturing the embryo to ensure survival, and reported common blight resistance of the F_3 generation as quantitatively inherited. The cv. Great Northern Nebraska No. 1 was selected and further improvement was found in several late-maturing plants and the cv. Great Northern Nebraska No 1 Selection 27 was released (Scharen, 1959; Coyne et al., 1963; Coyne and Schuster, 1970). This cultivar has been used in several crosses and many great northern (GN) cultivars such as GN Tara and GN Jules have their resistances derived from GN Nebraska No. 1 Selection 27 (Coyne and Schuster, 1969; Coyne and Schuster, 1970). The bacterium has been isolated from seed of some of the more resistant great northern cultivars even when the external appearance of the seed was normal (Schuster et al., 1979). The resistance of most common bean cultivars is incomplete and often called tolerance. Pathogenic isolates of the bacterium have also been isolated from epiphytic populations of bacteria on symptomless bean leaves (Thomas and Graham, 1952).

The vegetative stage of the plant is more resistant than pod-filling stage and resistance is linked to long photoperiod and high-temperature-delayed flowering (Coyne et al., 1973; Coyne and Schuster, 1974a). Hence under tropical conditions, where flowering is induced more rapidly, the great northern cultivars were poorly adapted and more susceptible (Webster et al., 1980; Webster et al., 1983). Schuster et al. (1973), however, found that the cv. GN Nebraska No. 1 Selection 27 was resistant to an isolate of the bacterium from Nebraska but slightly susceptible to isolates from Colombia and Uganda when tested under the same conditions. The cv. ICA Guali was released as common blight resistant in Colombia (Leakey, 1973) and has been reported as resistant to the Nebraska isolate but moderately susceptible to Colombian and Ugandan isolates of the bacterium (Schuster et al., 1973).

United States Department of Agriculture Plant Introduction (PI) 207262 has been reported as having different genes for resistance to a Nebraska isolate of the bacterium when compared to GN Nebraska No. 1 Selection 27 and having resistance to Colombian and Ugandan isolates (Schuster et al., 1973; Ekpo

and Saettler, 1976). Velich et al. (1986) reported the cvs GN Nebraska No.1 Selection 27 and GN Jules as resistant and PI 207262 as only moderately resistant to a Hungarian isolate of the bacterium. In addition, Coyne and Schuster (1974b) in reporting a differential reaction of pods and foliage of several lines infected with a Nebraska isolate of the bacterium, found the pods of PI 207262 to be slightly susceptible and those of cv. GN Nebraska No.1 Selection 27 were resistant.

Sources of resistance have also been reported in P.coccineus and lines have been developed by interspecific crosses (Coyne and Schuster, 1973; Anon., 1982; Mohan and Mohan, 1983; Zapata et al., 1985; Park and Dhanvantari, 1987). Other crosses have been made with tepary beans and improved common blight resistance obtained (Thomas and Waines, 1984; Anon. 1984). Scott and Micheals (1988) reported resistance in common beans derived from tepary beans controlled by two loci requiring a dominant allele at both loci. Michaels (1992) reported that monogenic dominant resistance genes recently derived from tepary crosses in the common bean lines XAN 159 and OAC 88-1 segregated independently. Adams et al. (1988) reported resistance derived from a Cobalt-60 mutant with a single major gene with modifying genes affecting the reaction. The cv. ICA L24 has been reported as having "some type" of leaf penetration resistance to common blight as after wound inoculation the disease reaction increases considerably and, under heavy disease pressure, pods sometimes show more severe disease symptoms than might have otherwise been expected (Anon. 1984).

Quantitative leaf resistance to common blight has been reported with differing levels of dominance (Aggour, 1987) and low narrow-sense heritability estimates (Coyne and Schuster, 1974a; Aggour and Coyne, 1989; Webster et al., 1980), but a high estimate in a cross with the cv. GN Jules was found (Webster et al., 1980). The low estimates of heritability show the high effect of the environment has on the genotype expression. Pompeu and Crowder (1972) obtained a narrow-sense heritability estimate of 45 % when working in growth chambers. Transgressive segregation for resistance has also been observed in certain crosses (Valladares-Sanchez et al., 1979). Webster et al. (1980) found a reasonable correlation between disease ratings at the seedling stage and mature plants, but Aggour and Coyne (1989) reported poor correlation. Differences in reaction to common blight can

occur in a single disease situation compared to a mixed infection such as common blight and rust (Zaiter et al., 1989a). Common blight symptom expression is affected by stage of development and nutrition of the plants and leaves (Patel and Walker, 1963). Hence a possible problem for a multiple disease testing programme would be to compare plants and leaves at the same stage.

2.2.3 Halo blight (Pseudomonas syringae pv. phaseolicola)

Halo blight (Pseudomonas syringae pv. phaseolicola (Burkholder) Young et al.) was first described by Burkholder in 1926 after isolation from diseased beans in New York State. After only five years, Burkholder and Zaleski (1932) reported it as the most serious bacterial "trouble" of beans. Hayward and Waterson (1965) suggested it was the most widespread of the bean bacterial diseases. Wager (1936) reported "considerable" losses due to halo blight in eastern and central Transvaal. He also suggested that most of the loss due to the bacterial blights was from halo blight with common blight occurring only sporadically. Doidge et al. (1953) reported halo blight being more prevalent than common blight in South Africa. Halo blight is more common on the Highveld, where the greatest production of dry beans occurs, and hence is more important than common blight (Melis, 1987a).

Leaf infection by the bacterium results in a small water-soaked lesion after four days. During this time the bacterium multiplies very rapidly and thereafter, the number of bacterial cells remains static or declines. In susceptible plants a halo develops around the water-soaked lesion and systemic chlorosis can occur because of toxin (phaseolotoxin) production, but incidence and severity depends on environmental conditions (Jensen and Livingston, 1944; Hoitnik et al., 1960; Patil et al., 1974; Schwartz, 1989b). Halo symptoms have been obtained from the application of the toxin alone and a selection method has been suggested by noting the response of calli to bacterial culture filtrates (Mitchell and Bieleski, 1977; Hartman et al., 1985). Phaseolotoxin has also been shown to induce chlorosis in unrelated plants such as cucumber (Cucumis sativus L.), maize (Zea mays L.) and tomato (Lycopersicon esculentum Mill.) (Ferguson and Johnston, 1980). Systemic infection can occur and infection of vascular tissue causes a red discolouration (Zaumeyer and Thomas, 1957). Yarwood (1969) reported halo

blight lesion size increased with prior infection of rust.

Pod symptoms of water-soaked lesions, with or without a reddish brown border depending on age, are identical to those caused by common blight. The two diseases can usually be separated by the bacterial exudate at the centre; for halo blight it is white to cream coloured and for common blight, yellow. Pod infection at an early stage can cause the whole pod to rot. Seed infection by the bacterium usually occurs by surface contamination either from within an infected pod or from contact during or after harvest with infected material (Zaumeyer and Thomas, 1957).

Classification of the bacterium into races began after the discovery of the resistance of the cv. Red Mexican University of Idaho (UI) 3 (Jensen and Goss, 1942; Walker and Patel, 1964). After inoculation of Red Mexican UI 3, the initial rapid multiplication of the bacterium occurs whether race 1 (avirulent) or race 2 (virulent) is used, although race 1 multiplication is not as extensive (Omer and Wood, 1968). Thereafter, a hypersensitive reaction is initiated by race 1 and the water-soaked lesion becomes a small discrete necrotic lesion with little or no chlorosis (Jensen and Goss, 1942). The resistance of Red Mexican UI 3 was found to be dominant and monogenic (Schuster, 1950). A third race (race 3) was also identified based on a hypersensitive reaction to another single dominant gene that is not linked to that found in Red Mexican UI 3 (Anon., 1985; Davies et al., 1986). Inoculations of a set of halo blight differential cultivars have identified the presence of race 1 in South Africa (Edington, 1989b). Use of race-specific resistance to race 1 would be unwise as Boelema (1984) has reported the presence of race 2 (i.e. virulent in Red Mexican UI 3) in South Africa.

According to Dr A.J. Liebenberg (Pers. Comm., 1992), Dr D. Teverson of the Institute of Horticultural Research, Wellesbourne, England has expanded the number of races to nine by increasing the number of differential cultivars to six of common beans and one of tepary beans. The number of races will probably increase as yet more resistance genes specific for different biotypes of the bacterium are discovered. As the new differential cultivars were not available at the beginning of this study, races referred to in this thesis are only 1, 2 and 3.

A few cultivars have been identified as having race non-specific resistance to halo blight. Resistance to races 1 and 2 was identified in the PI 150414 and found to be governed by a single recessive gene (Patel and Walker, 1965 and 1966). Hill et al. (1972) reported separate genes controlling resistance to the water-soaking in the leaf and in the pod and thirdly to systemic chlorosis. Linkage between the genes governing resistance to systemic chlorosis and leaf infection has been noted (Coyne et al., 1971; Hill et al., 1972). Leaf resistance gene of the cv. GN Nebraska No.1 Selection 27, reported by Coyne et al. (1966), was the same as that of PI 150414 (Hill et al., 1972), but in contrast to the work of Patel and Walker (1965), dominant. Taylor et al. (1978) reported that the resistance gene of PI 150414 was inherited as a recessive in two crosses and as partially dominant in a third cross, indicating that genetic background can greatly affect symptom development.

Anonymous (1985) reported the cvs GN Jules and Wisconsin Halo Blight Resistant (HBR) 72 were resistant to the three races classified at that time. GN Jules was derived from a cross between GN Nebraska No.1 Selection 27 and GN 1140 and Wisconsin HBR 72 from crosses to obtain resistance from PI 150414 (Coyne and Schuster, 1970; Hagedorn et al., 1974). Hence it is most probable that the three cultivars, GN Jules, GN Nebraska No.1 Selection 27 and Wisconsin HBR 72, have the same resistance gene. GN Nebraska No.1 Selection 27 was, however, reported by Anon. (1985) as only having intermediate resistance to race 3. The three cultivars were found to have leaf resistance to an isolate of race 1 collected at Potchefstroom, Transvaal, by the present author (unpublished data).

Crosses were made by the present author between speckled sugar bean lines and Wisconsin HBR 72. After discarding susceptible F_2 plants, F_3 progeny were planted in a field trial with every twelfth row being Wisconsin HBR 72. Warm conditions did not favour halo blight development and there was poor leaf symptom development but the pods were severely blighted (Edington, 1989a). Zaiter and Coyne (1984) reported Wisconsin HBR 72 as having only a moderate pod resistance to a Nebraska isolate of the bacterium. In addition, Velich and Szarka (1978) reported Wisconsin HBR 72 as moderately susceptible to a Hungarian isolate. Both groups, however, reported the pods of GN Nebraska No.1 Selection 27 as moderately resistant. Stoetzer et al.

(1984) indicated the reaction of GN Nebraska No. 1 Selection 27 as highly resistant, GN Tara as resistant, PI 150414 as resistant but also susceptible in one season and GN Jules as susceptible under field conditions in Kenya (races 1 and 2 were probably present).

2.2.4 Angular leaf spot (Phaeoisariopsis griseola)

Angular leaf spot (Phaeoisariopsis griseola (Sacc.) Ferrais) is favoured by the same climatic conditions (moderate temperatures and high rainfall) as rust, but, unlike rust, is at present only important in Natal in South Africa (Melis, 1987a). It was first reported in Italy in 1878 (cited in Inglis and Hagedorn, 1986) and is now found in many major bean-growing countries of the world (Anon., 1986b). It can be seed-transmitted but Dhingra and Kushalappa (1980) found no correlation between pod disease severity and rate of seed infection. Seed infection only occurred when the seed was directly under a lesion at the pod suture. The main mode of survival is in plant debris (Cardona-Alvarez and Walker, 1956). Two reports of loss due to angular leaf spot of up to 80 % have been made (Anon., 1980; Schwartz et al., 1981).

The first report of resistance to angular leaf spot was by Gardner and Mains in 1929 (cited in Schwartz et al., 1982) and there have been several since. Breeding for resistance has been complicated by pathogenic variability. Alvarez-Ayala and Schwartz (1979) found that the cv. Caraota 260 reported to be resistant in Brazil, was susceptible to three out of four Colombian isolates. Anonymous (1984) reported the cv. BAT 332 to be resistant in Colombia, but susceptible in parts of Brazil and Argentina. Anonymous (1985) suggested that 17 isolates from central and southern America could be categorized into five pathogenicity groups based on reactions in eight bean cultivars. Buruchara (1983) suggested the presence of seven races in Colombia based on the differential reaction of six bean cultivars. Anonymous (1990) reported the presence of different races in Malawi.

Barros et al. (1958) reported resistance as usually recessive and controlled by two or three independent "factors", but in a few crosses, dominant. The resistance of cv. Line 258 was governed by a single dominant gene (Cardona-Alvarez, 1958), whilst that of cv. Caraota 260 is governed by a single

recessive gene (Santos-Filho et al., 1976). Singh and Saini (1980) reported a recessive resistance gene derived from an interspecific cross with a P.coccineus cultivar. Hagedorn and Rand (1986) reported disease-rate-reducing resistance in PI 209488 on which symptoms were only noted 20 days post inoculation. Buruchara et al. (1988) indicated differences in incidence and/or severity amongst 14 cultivars after inoculation with a single isolate under field conditions in Kenya.

2.2.5 Anthracnose (Colletotrichum lindemuthianum)

Anthracnose (Colletotrichum lindemuthianum (Sacc. et Magn.) Scrib) could be considered as one of the classical plant diseases. Zaumeyer and Thomas (1957) cited the "earliest satisfactory evidence of its collection dates back to 1843." Barrus (1911 and 1918) was the first to report physiological specialization of a plant pathogen and the first report of inheritance of disease resistance in beans was made by Burkholder (1918), both with anthracnose. A third race, gamma, was reported by Burkholder (1923) to differ from the alpha and beta races in its ability to infect several bean cultivars. Further races have been reported by Andrus and Wade (1942), Charrier and Bannerot (1970), Fouilloux (1975), Hubbeling (1976), Schnoch et al. (1975), Krüger et al. (1977) and Hubbeling (1977). There are other reports of new races and others that eventually turned out to be additional isolates of previously reported races indicating the high degree of pathogenic variability of the fungus (Pastor-Corrales and Tu, 1989). Hence the need for a standard set of differential cultivars such as those suggested by Drijfhout and Davis (1989).

Zaumeyer and Thomas (1957) reported that from 1912 to 1920, anthracnose was the most important disease in the USA east of the Mississippi. Its importance declined thereafter with the introduction of resistant cultivars and certified disease-free seed schemes. Pastor-Corrales and Tu (1989), however, suggested it is still probably the most important disease of beans worldwide. Mmbaga and Stavely (1988) reported anthracnose as the most important disease of beans in Tanzania. Shao and Teri (1985) indicated net gains of \$1010 ha^{-1} and \$653 ha^{-1} by controlling anthracnose with benomyl and by growing resistant cultivars, respectively, in Tanzania. The impact of anthracnose in South Africa in the recent past has been reduced by a

certified disease-free seed scheme (Melis, 1987a) and drought conditions unfavourable for disease development. It is presently only important in Natal where growers have tended to replant their own seed.

Separate dominant genes have been reported as conferring resistance to the alpha, beta and gamma races (McRostie, 1919; McRostie, 1921; Burkholder, 1923). Andrus and Wade (1942) and Cardenas et al. (1964), however, found a more complicated system of inheritance of ten genes with epistatic interactions at three loci. Resistance to anthracnose was believed to have been simplified with the discovery of the Are-gene in the cv. Cornell 49242 (Mastenbroek, 1960). The Are-gene conferred resistance to all the then known races, but others such the kappa and jota races identified later can overcome it. Anonymous (1986a) found that none of their six African isolates, from Kenya, Burundi, Tanzania and Zaire, could overcome the Are-gene. In addition, use has been made of the Are-gene in Malawi (A.J. Liebenberg, pers. comm., 1988).

2.2.6 Ascochyta blight (Phoma exigua var. exigua and P. e. diverspora)

Ascochyta blight is also a cool-weather disease, but until 1987 it was only reported in Natal with the causal organism identified as Phoma exigua var. exigua (Desmazieres) (Trench et al., 1986; Melis, 1987a). Schwartz (1989a) suggested the most common causal organism of Ascochyta blight is P.exigua var. diverspora ((Bub.) Boerema) and most of the literature refers to this variant. It is a weak pathogen, often requiring damage, such as from a hail storm, to infect (Sutton and Waterston, 1966; Melis, 1987a). Ascochyta blight can be seed-borne, but is more important where an inadequate rotation is practised. Schwartz et al. (1981) reported loss due to Ascochyta blight in Colombia of ca. 40 % and suggested this was a conservative estimate as there was only a moderate infection. Anonymous (1980) reported losses in Colombia of 41 and 52 %, respectively, from two cultivars. Ascochyta blight is common at higher altitudes in East African countries such as Burundi, Rwanda, Zaire, Kenya and Zambia (Schwartz, 1989a) and was reported in the Belgian Congo in 1938 (Hendrickx, 1939). Hansford (1932) suggested that Ascochyta blight was jeopardizing bean production in certain parts of Uganda in 1931, a wet year. Doidge (cited in Trench et al., 1986) reported an Ascochyta blight of beans in Natal in 1922-23.

There appears poor resistance to Ascochyta blight within P.vulgaris germplasm but resistance has been found in P.coccineus and interspecific crosses have been made (Anon., 1982; Anon., 1986a; Schmit and Baudoin, 1987; Anon., 1990).

2.2.7 Rust (Uromyces appendiculatus)

Melis (1987a) suggested that rust (Uromyces appendiculatus (Pers.) Unger) was "probably the most important constraint to bean production in South Africa" and obtained a significant negative correlation between a rust infection index and seed yield in a cultivar trial (1987b). Rust is found in virtually every country where beans are grown (Zaumeyer and Thomas, 1957). Infection can cause premature senescence of leaves, leading to the death of the plant in susceptible cultivars under favourable conditions (Laudon and Waterson, 1965; Melis, 1987a). Hence there is a potential for 100 % yield loss. Melis (1987b) found chemical control of rust gave a 44 % improvement in yield for the cv. Bonus at Ukulinga (near Pietermaritzburg), Natal. Zaumeyer and Thomas (1957) reported losses up to 80 % in the USA. Lindgren et al. (1992) suggested yield loss began when rust infection exceeded 5 to 20 % of leaf area, depending on other environmental conditions. J. Medley Wood was reported as finding Uromyces phaseolorum (synonym for U.appendiculatus) on Rhynchosia sp. or Eriosema sp. at Inanda, Natal in 1881 (Bottomley, 1916). Doidge (1932) reported the presence of U.appendiculatus on beans in the Transvaal in 1909.

Infection of beans is favoured by humid, moderate weather, when prolonged wetness of the leaf aids infection. Ten to fifteen days post-infection urediosori erupt to release new urediospores. Teliosori are produced late in the season giving rise to dark brown teliospores. Pycnia and aecia have rarely been observed in nature (Stavely and Pastor-Corrales, 1989) and Doidge (1932) reported the absence of this stage in South Africa. Aecia, however, have recently been found in Pietermaritzburg under natural conditions of infection (Pers. Comm., F.H.J.Rijkenberg, 1992), but the occurrence is very rare. The lack of a sexual stage does not appear to have hindered this pathogen unduly. It can produce one million urediospores per square centimetre on leaves with two to 100 uredia per square centimetre (Yarwood, 1961). Re-infection of South African summer-rainfall bean crops

may occur from winter crops in the Low Veld or at the coast.

Various methods of controlling rust have been proposed. Spraying can be costly and should be done before flowering to be of sufficient economic benefit. Biological control has also been suggested and Bacillus subtilis (Ehrenberg) Cohn and Verticillium lecanii (Zimm.) Viégas have been tested (Baker et al., 1985; Allen, 1982). Induced resistance by other species of rust and avirulent races of bean rust has also been examined. Yarwood (1956) reported cross-protection of bean plants from bean rust by sunflower rust (Puccinia helianthi Schw.) and vice versa. He also found prior inoculation of avirulent races of U.appendiculatus gave 0 to 76 % cross-protection from a virulent race. Johnson and Allen (1975) reported a reduction in sporulation by bean rust after prior or simultaneous inoculation with a weakly virulent bean rust race and that this may have a significant effect in multiline varieties. Allen (1975) also indicated a reduction in sporulation by bean rust caused by prior or simultaneous inoculation with maize (Puccinia polysora (Underw.) and P.sorghi (Schw.)) and stripe (Puccinia striiformis (West.)) rusts, and that this could be a disease-reducing factor in intercropping. Anonymous (1987) reported an increase of 13 % and a decrease of 32 % in yield when separate plots were sprayed with an avirulent and virulent race of bean rust, respectively, when compared to naturally infected plots. The highest yielding plot (22 % increase in yield) was one in which the avirulent race was sprayed followed by the virulent race three days later at weekly intervals. Unfortunately, glasshouse tests have shown the resistance is not systemic and only occurs in inoculated leaves.

Rust of beans is similar in many respects to the cereal rusts and many lessons learnt in cereal rust resistance breeding are relevant to bean rust resistance work. Biffen (1905) first reported Mendelian inheritance of resistance to a plant disease when he found a 3 : 1 ratio in the progeny of a cross between yellow or stripe rust (Puccinia glumarum syn. P.striiformis) susceptible and resistant cultivars. Fromme and Wingard (1921) reported differences in bean cultivar susceptibility to bean rust and this was corroborated by the work of Morse (1918). Interestingly, they pointed out that Gassner (1909) found two cultivars to be very susceptible whilst they rated them as resistant. They suggested the cultivars tested by Gassner

were not true to type.

Harter et al. (1935) found differences in isolates from different states of the USA. An isolate of rust from California failed to produce teliospores in any great number unlike isolates from Colarado and Virginia. In addition, they found two "forms" of rust, one of which could infect cultivars which were resistant to the other. Harter (1939) reported "13 distinct physiological races" and Dundas and Scott (1939) reported four "strains", based on differential reactions of cultivars. By 1941, Harter and Zaumeyer had increased the number of races to twenty based on the reaction of seven differential cultivars. Wingard (1933) reported resistance to bean rust in two crosses as conditioned by a dominant gene. Zaumeyer and Harter (1941) found resistances to two races to be inherited as a dominant gene and as an incomplete dominant gene against two other races. Other genetic factors affecting the inheritance of the latter two races and another two races tested were suggested.

Since the work of Zaumeyer and Harter (1941), numerous reports of races and inheritance of resistance have been made (Stavely and Pastor-Corrales, 1989), such as the identification of eight races in East Africa (Howland et al., 1966). Stavely and McMillan (1992) reported that 55 separate races are stored under liquid nitrogen by the USDA at Beltsville, Maryland. Stavely et al. (1989) suggested that nearly 200 races of bean rust have been identified worldwide. Coyne and Schuster (1975) reviewed the success of rust resistance breeding, including reports of race identification. With the presence of oligogenic resistance and the ability of rust races to overcome it (including a report of one dominant and two recessive virulence genes in rust with matching resistance genes in beans (Christ and Groth, 1982a and 1982b)), it would appear that the bean/rust interaction follows that of a "gene for gene" relationship proposed by Flor (1955). As early as 1912, Pole Evans suggested there had been little success in breeding for wheat rust resistance, and reported, "varieties recommended for their rust-resisting qualities holding good for several years in succession, and then they disappear from view altogether." He prophetically suggested that wheat breeding was not "likely to lead to results of much practical importance so long as the parasites themselves are ignored." A recent example of bean rust resistance failing is found with the cv. Kamberg. It was released

locally as rust-resistant (Vermeulen, 1984) and has become progressively more susceptible (Melis, 1987a). No bean cultivar has been resistant world-wide (Stavely and Pastor-Corrales, 1989).

The problems of durable resistance breeding in beans are similar to those of wheat (Triticum aestivum L. em. Thell) and Coyne and Schuster (1975) made several comparisons between the two. The options available depend on the use of horizontal or vertical resistance. Horizontal resistance allows limited infection with low levels of loss and, by definition, gives protection against all races (Van der Plank, 1963). Horizontal and vertical resistance are discussed more fully in the next section.

Horizontal resistance to bean rust has been attributed to several host characteristics. Groth and Urs (1982) found differences in receptivity to infection after inoculation of six cultivars partially correlated with the number of stomata. Shaik (1985) also found a partial correlation with the stomatal number. Wynn (1976) investigated the thigmotropic response of germ tubes and indicated the importance of the correct stimulus after working with leaf surfaces, replicas and artificial membranes. There were no differences in germ tube differentiation to an appresorium on five cultivars. Hoch et al. (1987) found that appresorium formation was caused primarily by abrupt changes in surface topography of two succeeding acute angles, i.e. a grove or ridge. Allen et al. (1991) reported forty bean rust races reacted similarly in thigmotropic sensing resulting in the formation of appressoria and suggested that altering stomatal characteristics may induce horizontal resistance. Statler and McVey (1987) reported the cvs Nodak and Upland as having a smaller infection density and number of uredia per square centimetre. Histological preparations indicated fewer successful penetrations and more early- and late-aborted colonies. There were no significant differences in latent periods. Aust et al. (1984) reported cultivars with nearly one third less sporulation than a fully susceptible cultivar.

Leaf pubescence has also been reported as contributing to horizontal resistance (Shaik, 1985). Leaf hairs (trichomes) can form a physical barrier and prevent water droplets forming on the epidermis thus preventing germ tubes access to the epidermis (Burrage, 1969; Von Alten, 1983). This

phenomenon tends to occur before full leaf expansion and with long straight (acicular) hairs, but not with short hooked (unciform) hairs (Zaiter et al., 1990). Later trifoliate leaves are also more pubescent than the lower trifoliates (Shaik and Steadman, 1989). Mmbaga and Steadman (1990) reported difficulty in demonstrating the resistance of pubescent cultivars under glasshouse inoculation conditions suggesting dew formation different to that in the field as the cause. Mmbaga and Steadman (1990 and 1991a) found reduced uredial size after infection of pubescent cultivars, which they suggested was due to a delay in infection caused by leaf hairs. In further studies on spore deposition, Mmbaga and Steadman (1992a) found pubescence reduced the number of spores reaching the epidermis with 60 to 80 % of spores trapped by leaf hairs on leaves 10 to 20 % expanded. There was no correlation between infection density and number of spores trapped, but correlations were found between uredial density and number of spores deposited on the epidermis (presumably ratios of those trapped to those deposited varied) and also between uredial density and leaf size at inoculation. Trapped spores were observed to produce germ tubes that grew "horizontal" to the epidermis without contacting it, some were also seen to grow along the leaf hairs.

2.3 Disease resistance: Vertical versus horizontal resistance

Van der Plank (1963) coined the terms vertical and horizontal resistance after graphically illustrating the two forms (Figure 2). When vertical resistance is present there tends to be a large difference between the host's reactions to virulent and avirulent races (such as between races 1 and 2 for cultivar 2 in Figure 2, ie. a large vertical difference). When horizontal resistance is present, and vertical resistance absent, the differences between levels of resistance to the races, due to differing degrees of aggressiveness, tend to be small. In Figure 2, cultivar 1 has a higher level of horizontal resistance compared to cultivar 2 for the races virulent in both cultivars (1, 3, 4, 6 and 7). Both types of resistance are not mutually exclusive but horizontal resistance will usually be masked by vertical resistance (in Figure 2, both cultivars have the same vertical resistance to races 2 and 5).

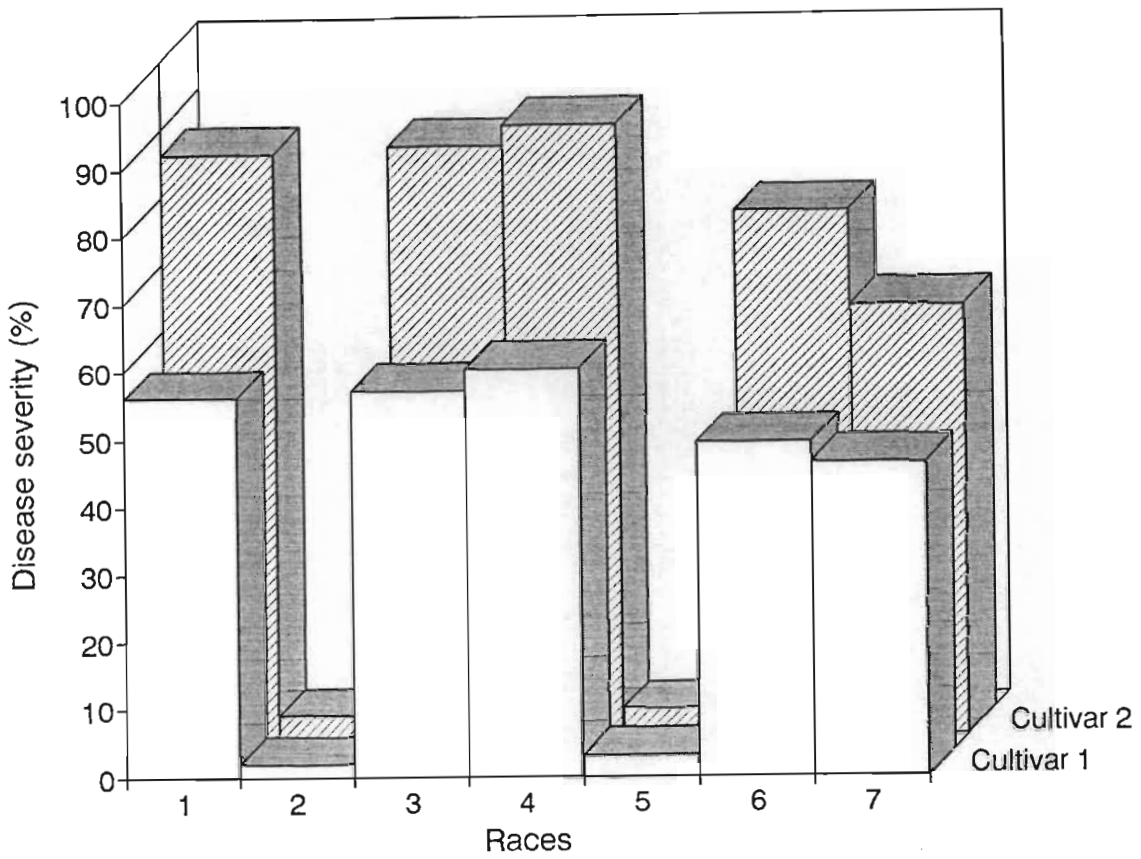


Figure 2. Vertical and horizontal resistance reactions of two cultivars to races of a pathogen (After Van der plank, 1963).

Partial resistance has often been used incorrectly as a synonym for horizontal resistance. Several vertical resistance genes have been found that do not confer full resistance but restrict the degree of pathogen reproduction (Johnson, 1984). The definition of horizontal resistance assumes that a new race of the pathogen will not arise to make the resistance redundant, it then becomes vertical resistance. In this regard, partial resistance is a preferable term to horizontal resistance. Durability of resistance can often be only assessed in hindsight; what was thought to be horizontal resistance may prove to be partial vertical resistance. The terms vertical and horizontal have the advantage over oligogenic and polygenic resistance as the boundary between oligogenic and polygenic inheritance of resistance is somewhat arbitrary (Manners, 1988).

Vertical resistance is usually conferred by one or a few genes (oligogenic inheritance). More than one vertical resistance gene may be present in a cultivar but each gene acts independently assuming no linkage (genes on the

same chromosome), epistasis (gene expression requires the expression of another gene) or allelic (each allele of the same gene conditions resistance to different race(s)) effects. One vertical resistance gene may condition resistance to more than one race, but, by definition, there is at least one race which may overcome it.

In using vertical resistance against highly variable and mobile pathogens it is assumed there will be a breakdown of resistance if only a few genes are involved. To increase the number of genes in the system, more genes can be incorporated into a single line, or several lines (possibly isogenic) can be developed with separate genes (multilines). The problem foreseen with both types is the development of "super" races, able to overcome all the resistance genes in the system. Alexander et al. (1985) found races of bean rust maintained for five generations on a particular cultivar bore unnecessary virulence genes for cultivars not normally encountered by those races in the field. McCain et al. (1990) found that the bulk crossing of a collection of bean rust races revealed further virulence genes masked in the heterozygous state of the original races. Differences in virulences in several rust species are also noted towards different host species, leading to differentiation into formae speciales. Eshed and Dinoor (1980), however, found that differences between formae speciales and race differentiation overlapped and the situation was not as clear as previously thought after crossing a number of formae speciales of Puccinia coronata. Fillingham et al. (1992) reported two virulence genes which determine race-specificity of Pseudomonas syringae pathovars phaseolicola and pisi also conferred non-host resistance, but two other virulence genes conferred no such resistance.

Addition of further resistance genes into a cultivar increases the selection pressure for a race with matching virulence assuming such increased levels of virulence genes have no effect on the pathogen's reproductive fitness. The possibility of an "ABC-XYZ" system similar to that operating with virulence genes of wheat stem rust (Puccinia graminis Pers. f. sp. tritici Eriks. and E.Henn.) (Vanderplank, 1982) occurring with races of bean rust may have been found by Alexander et al. (1985). They found races virulent on the cv. US No.3 and on cvs. Roma, Early Gallatin or B1349 had lower levels of survival and/or reproduction. Under an "ABC-XYZ" system, virulence genes are grouped into two sets and a combination of a virulence

gene from both groups causes a reduction in fitness. Stavely and Steadman (1992) pointed out that several potential virulence combinations of bean rust have never been found and there may be genetic fitness limitations; this assumes no allelic effects. Inheritance of other rust virulence genes such as for Puccinia recondita, however, is far simpler than that of host resistance genes with little linkage effects (Statler, 1988).

Numerous alternatives to vertical resistance have been reported with rust diseases of other crops. Luke et al. (1984) working with crown rust (Puccinia coronata f. sp. avenae Fraser & Led.) on oats (Avena byzantia C.Koch) found retarded hyphal growth. This led to a reduction in the number and size of uredia and an increase in latent period. Earlier work of Luke et al. (1975) found this resistance to be horizontal and controlled by a few genes with slight partial dominance for susceptibility. Heagle and Moore (1970), also working with crown rust of oats, found the partial resistance due to a lower infection efficiency, reduced hyphal growth, an increase in latent period and lower spore production. Brake and Irwin (1992) working with oat crown rust and the oat cv. Panfive, noted lower infection efficiency, fewer and smaller uredia and lower spore production. Sztejnberg and Wahl (1978) indicated that the slow-rusting of oats (Avena sterilis L.) after infection with Puccinia graminis f.sp. avenae (Eriks. & E.Henn.) was due to restricted colonization of the host. They observed a reduction of urediospore production per pustule in both seedling and flag leaves. Niks (1982) found early abortion of colonies of barley leaf rust (Puccinia hordei Otth.) at the stage of the formation of the first haustorium in partially resistant barley (Hordeum vulgare L.) cultivars. Johnson and Wilcoxson (1979) suggested the partial resistance of barley to barley leaf rust was polygenically inherited.

Lee and Shaner (1984) found that with leaf rust (Puccinia recondita Rob. ex Desm. f. sp. tritici) on wheat the infection process was the same for hypersensitive and partially resistant and susceptible cultivars up to the stage of the formation of substomatal vesicles. But thereafter they found the growth rate of the fungus in the partially resistant cultivars was slower and the number of haustorial mother cells to be fewer. Partially resistant and susceptible cultivars required the same colony size to form a uredial bed before the formation of uredia but with partially resistant

cultivars it took longer and hence a longer latent period. Broers and Jacobs (1989) also found partial leaf rust resistance in wheat to cause a reduction in mycelial growth rate (as did Gavinlertvatana and Wilcoxson, 1978). A higher rate of early colony abortion was noted after the formation of one or two haustorial mother cells. Unlike Lee and Shaner (1984), they found that a smaller colony size was needed to form a uredial bed in partially resistant cultivars. Shaner (1983) reported that pustule size increased more slowly in slow-rusting cultivars. Broers and Jacobs (1989) found partial resistance to be inherited recessively with the additive interaction of 2 to 3 genes depending on the cultivar with the possibility of transgressive segregation. They could not find conclusive evidence against race-specificity of the resistance but found it to be durable over a number of locations and years. Lehman and Shaner (1992) evaluated latent period, infectivity, sporulation, uredial size and growth rates of wheat leaf rust in several susceptible and partially resistant wheat cultivars in the glasshouse. These were then compared to field area under disease progress curve evaluations. All factors were highly correlated with the field evaluations, sporulation and final uredial size being the most important followed by latent period. Infectivity was the least important. Zummo (1988) found decreases in uredium number and size and numbers of uredospores produced in partially resistant maize compared to a susceptible cultivar when working with P.polysora (Underw.). Pataky (1986) found a similar situation in sweet corn resistance to P.sorghi. Scott and Zummo (1989) working with partially resistant maize hybrids also found a longer latent period after infection with P.polysora. Subrahmanyam et al. (1983) reported partial resistance in peanuts (Arachis hypogea L.) to Puccinia arachidis (Speg) consisting of increased latent period and inhibition of the formation of uredia and urediospores. Johnson (1986) found fewer pustules with longer latent periods in asparagus (Asparagus officinalis L.) partially resistant to Puccinia asparagi DC.

Niks (1983) found that in the partially resistant barley cultivar Vada, the number of infection structures without haustoria nearly matched those of the non-host, wheat, after infection with Puccinia hordei. In a vertically resistant cultivar, resistance occurred after the formation of a haustorium. In Vada (and other cultivars partially resistant to P.hordei), a thickening of the haustorial mother cell wall developed at the point of contact with

the host cell. Broers and Jacobs (1989) also found an inhibition of haustorium formation of the infection structures, but this accounted for only a small fraction of the resistance. They suggested the bulk of the resistance was due to poor transfer of nutrients or an inhibition of the fungus. These mechanisms would operate at the haustorium interface which is where vertical resistance genes operate.

Following on from the work of Nelson et al. (1982), in which "defeated" powdery mildew vertical resistance genes exhibited residual and additive resistance to virulent races, Brodny et al. (1986) found a similar situation for wheat stem rust resistance genes. The vertical resistance gene Xa-4 of rice (Oryza sativa L.) to Xanthomonas campestris pv. oryzae (Ishiyama) has also been found to have a residual effect against virulent races of the bacterium (Koch and Parlevliet, 1984). Nelson et al. (1970) suggested that resistance genes against Setosphaeria turcica ((Lutt.) Leonard & Suggs) in maize could act in both a vertical or horizontal manner, depending if the race was virulent or not. In addition, some vertical resistance genes have been shown to give partial resistance to some races but not to others such as the R10, R11 and, to a lesser extent, R2 and R4 genes of potato (Solanum tuberosum L.) which govern resistance to Phytophthora infestans (Mont.) de Bary (Landeo, 1989).

The question arises: What if horizontal and vertical resistance genes are different expressions of the same sort of genes? It is quite possible that "defeated" vertical resistance genes with residual effects, having an evolutionary benefit, are retained and those that do not are lost. A jump from horizontal resistance to vertical resistance is also possible, when a mutation would give the plant an immediate and large benefit. The initial derivation of the resistance is unimportant for plant breeders, but the nature of its durability is. If the two types of resistance are different expressions of the same "family" of genes, why is partial resistance in some cases vertical and in others horizontal (transient or durable in a very loose sense)?

A possible problem with the mass-introduction of horizontal resistance is the adaptation of the pathogen to it. The pathogen may change according to its new situation and become more efficient at colonizing the horizontally

resistant cultivar, thus eroding the level of resistance. The possibility of such a situation in late blight of potatoes has been put forward, by amongst others, Caten (1974) who obtained increased aggressiveness after continued sub-culturing on a single cultivar. James and Fry (1983), however, suggested such increased aggressiveness was due to an overall increase in efficiency and was not cultivar-specific. Leonard (1969) suggested the possibility of increased adaptation in stem rust (Puccinia graminis Pers. f. sp. avenae Eriks. and E.Henn.) of oats (Avena sativa L.) after serial inoculations of a cultivar, but a mixture of races was used. Johnson and Taylor (1972) reported differences between isolates of yellow rust of wheat, based on the weight of spores produced on the same cultivars, but had similar virulences. Again the problem is to determine if the differences were due to specific cultivar/race interactions or if there was a greater overall aggressiveness. Eshed (1978) found high levels of narrow-sense heritability for aggressiveness after crossing different formae speciales of Puccinia coronata. Newton (1992) suggested the powdery mildew fungus of barley can adapt to partial resistance but that it is of an unstable nature.

Race-specificity of slow-rusting has occasionally been found. Parlevliet (1976) found a race of P.hordei to have a shorter latent period compared to other races and that this was more pronounced in slow-rusting cultivars. Cultivar trials planted in Mexico, South Africa and the USA indicated wheat cultivars may have slow stem-rusting resistance in one area but not in others (Wilcoxson, 1981). Presumably as no indication was given, the cultivars were equally adapted to each location.

2.4 Mechanisms of resistance

Beans are resistant to all but a tiny fraction of aerial pathogens; they are not affected by leaf diseases of potatoes, for example. In many cases the alighting spores do not receive the necessary stimuli to germinate and produce infection structures. In others, infection structures are formed, but are incapable of penetration if a similar situation occurs as found with reed canary grass (Phalaris arundinacea L.) and several non-pathogenic leaf spot fungi (Sherwood and Vance, 1976).

When a plant is challenged by an avirulent pathogen, an elicitor is released and this is recognized by the host and prompts it to induce resistance. This is usually associated with cell death (hypersensitivity) and phytoalexin production, but these may be symptoms of resistance rather than the cause (Király et al., 1972; Vidhyasekaran, 1988). Cell death and production of anti-microbial phytoalexins could be added defence mechanisms against nectrotrophic microbes in secondary infections. Perhaps the host responds with a general resistance reaction to cover all occasions, including abiotic damage. Tiburzy and Reisener (1990) indicated the importance of lignin or lignin-like compounds and callose in the resistance mechanisms of wheat to the stem rust fungus. In the Sr5- and Sr6-conditioned reactions, lignification was found over the entire cell and not restricted to the attempted site of penetration as with non-pathogenic fungi. Haustorial growth stops when lignification occurs. But how lignification affects the pathogen still remains unclear.

Elicitors have been found that are specific to a diseased situation with tomato and Cladosporium fulvum (Cooke) and wheat and leaf and stem rust fungi (De Wit and Spikman, 1982; Sutherland and Deverall, 1990; Kogel et al., 1988). In each case it was a glycoprotein of which the carbohydrate part has elicitor activity. In addition, eliciting activity has also been obtained from aqueous exudates from fungal structures such as by Chen and Heath (1990) with cowpea rust (Uromyces vignae Barclay) and cowpeas (Vigna unguiculata (L.) Walpers) and Dixon and Fuller (1977) with Botrytis cinerea (Pers.) in beans.

Chen and Heath (1990 and 1992) reported a cultivar-specific elicitor and this was only found in aqueous exudates from differentiated basidiospore germlings and from intercellular washing fluids from infections derived from uredospores with cowpea rust and cowpeas. Elicitor activity was not found in undifferentiated basidiospore germlings, urediospore germlings or intercellular washings from leaves with a basidiospore-derived infection. Hence the stage of development is very important for the production of the elicitor. Reports of cultivar/race-specific elicitors are rare. Beissmann et al. (1992), however, reported an elicitor from compatible stem rust infections of wheat containing the Sr5 gene that could induce resistance in plants with or without the Sr5 gene. They suggested the elicitation of

resistance was suppressed within susceptible plants. Coleman et al. (1992), however, found an glycoprotein elicitor after interaction between beans and a race of C.lindemuthianum that was active at nanomolar concentrations. Suboptimal concentrations of the elicitor induced 9.7 times the elicitation of phaseollinisoflavin in a resistant cultivar than in a cultivar susceptible to that race. At greater concentrations both cultivars reacted similarly, indicating that the differential reaction is easily swamped.

Vanderplank (1982) suggested that a susceptible reaction is specific; i.e. a plant would respond with a resistant reaction unless a specific mechanism prevents it. Presumably all pathogens can elicite resistance but either elicitation or the mechanism of resistance is suppressed. Skip and Samborski (1974) working with the Sr6 gene of wheat stem rust resistance, found that prior treatment of at least one day at 25°C was necessary for a susceptible reaction by a race that normally induces resistance at 20°C and vice versa. This shows that temperature sensitivity resides with the host and de novo synthesis of a receptor is required. Samborski et al. (1977) found that moving infected leaves from 25°C to 20°C led to resistance reactions primarily where new fungal growth was occurring, but not amongst the established colony, i.e. a state of susceptibility had been induced. It seems logical for a plant to have a general resistance mechanism for any possible challenge and a pathogen would then need to specifically overcome the resistance. Beissmann et al. (1992) suggested that in compatible stem rust infections of wheat, elicitation of resistance was suppressed. Perhaps a resistance gene product interferes with the suppression of resistance in what would have been a susceptible reaction. A virulence gene product would then in turn interfere with the resistance gene product to induce a return to susceptiblity. In the cases of induced susceptibility and resistance, simultaneous inoculations of virulent and avirulent races lead to a susceptible reaction (Ouchi et al., 1979). The induction of susceptibility, requires a time lag of 15 - 18 hours and de novo synthesis, as indicated by inhibitors of nucleic acid and protein syntheses (Ouchi et al., 1979). Induction of resistance requiresa time lag of only six hours. Perhaps induction of resistance protects the resistance mechanism at a receptor site, but induction of susceptibility requires active interference of the resistance mechanism. Jakobek and Lindgren (1992) found that a prior inoculation in beans with a susceptible isolate of Pseudomonas syringae pv.

phaseolicola suppressed elicitation of the host defences by glutathione application eight hours later. Suppressor activity of the bacterium was inactivated by protein synthesis inhibitors.

On the other hand, if resistance is the specific case, presumably elicitors are formed for each race of a disease. Perhaps each virulence gene modifies an elicitor(s) or codes for a separate elicitor. If each pathogen race codes for a separate elicitor, the host's receptor(s) must recognize an inordinately large number of elicitors for each pathogen and/or its races it will encounter. If the virulence gene modifies the initial elicitor then presumably only a small modification of the general receptor would be required (presumably by the resistance gene product). This may account for the high allelism of specific resistance genes compared to virulence genes. With Beissmann et al. (1992), however, an elicitor was found in diseased tissue without causing a resistance reaction. Perhaps this and other general elicitors such as those of Sutherland and Deverall (1990) follow elicitation pathways separate to the cultivar-specific elicitation. Yamaoka et al. (1990) reported elicitor activity in crude preparations from sorghum infected with Colletotrichum graminicola ((Ces.) Wilson), but no response was noted after application of the preparations to nine other crops including maize. Anderson (1978) on the other hand, found elicitors derived from Colletotrichum pathogens of lucerne and dodder that were active in beans. The lack of identification of cultivar-specific elicitors may be due to production only occurring during an incompatible race/cultivar infection and these are then quickly bound. Anderson (1980), however, found race-specific elicitors from races of Colletotrichum lindemuthianum. In addition, Chen and Heath (1990) found large quantities of a cultivar-specific elicitor in basidiospore germlings of cowpea rust without any great difficulty. As the host and race virulence genes of Pseudomonas syringae and Puccinia coronata appear to overlap (Fillingham et al., 1992; Eshed and Dinoor, 1980) it may be that the elicitor activity overlaps.

In several cases of symbiotic interactions, an infection of a host occurs. With nodulating bacteria in legumes, there is a fairly high degree of specificity (Vidhyasekaran, 1988). With vesicular arbuscular mycorrhizal fungi, however, there is very little specificity and a single species can infect several host families. Presumably there is no elicitor or the fungus

prevents recognition (Bowen, 1987).

When a partial resistance reaction occurs, presumably there is poor production or recognition of the elicitor or the suppression of the resistance mechanism is only partially successful. The degree of hypersensitive response to a race will often vary. With beans, resistance reactions of apparent symptomless infections to necrotic lesions up to 1 - 2 mm in diameter can be obtained after infection with avirulent rust races.

2.5 Breeding Methodology

Beans are an important crop and hence there has been a large input into their genetic improvement with a resultant extensive literature. Numerous governmental bodies and international organizations such as the Centro Internacional de Agricultura Tropical (CIAT), Cali, Colombia, devote a lot of resources into finding resistance to economically important diseases. CIAT has developed several bean disease nurseries which contain a range of resistant cultivars and, if appropriate, a set of differential cultivars (Anon., 1990). Unfortunately, these cultivars may not be locally adapted, suit local consumer preferences, be resistant to the local races of the disease in question and/or resistant to other diseases. They may, however, serve as parents for a crossing programme.

Most breeding programmes work in a step-wise manner. One cultivar has good quality characteristics, but may be susceptible to a disease and is crossed with another cultivar with resistance. If the progeny is selected without further crossing it is usually called pedigree selection, but if the progeny is crossed to either of the original parents one or more times it is called back-crossing (e.g. Van Rheenen and Muigai, 1984b). The new cultivar is released and perhaps another disease (or race of the old disease) may become economically important, besides the first disease. Another cross is done and in addition to selection for quality, selection for two resistances is now required. Quite often the two parents may have resistances to different diseases and the progeny is selected for all the disease resistances. The work is still done in a step-wise manner. In breeding for multiple disease resistance this may be considered too slow as each step normally involves

extensive field testing. The alternative would be to make a cross involving resistances to all the diseases. As this possibility generally does not exist it would require extensive crossing to create a breeding line with the required resistances. To expedite the creation of such a line, a reverse dichotomous crossing programme could be initiated. In a reverse dichotomy, cultivars with perhaps just a single disease resistance are crossed in pairs. The progeny is then tested for the relevant resistances and crossed with the progeny of another pair. This is continued until all desired resistances are combined into a single breeding line. The breeding line is now available for crossing to cultivars with the desired agronomic and quality characteristics.

Selection for a combination of disease resistances requires testing and elimination of susceptible progeny for all the diseases. Much of the literature about resistance breeding is about reliable means of testing for the presence of resistance in the field and/or glasshouse. In most cases of vertical resistance there is usually a good correlation between the field and glasshouse as it is normally a "yes/no" situation. For horizontal resistance, environmental conditions may have quite a dramatic effect on the expression of resistance and it is quite important to check for expression under field conditions such as the crop might be grown under.

In testing the resistance of a plant to several diseases the question arises if tests should be conducted simultaneously or sequentially and if so in what order. For common blight, Webster et al. (1980) found a reasonable correlation between disease ratings at the seedling stage and mature plants, but Aggour and Coyne (1989) reported a poor correlation. Zaiter et al. (1989a) found differences in reaction to common blight in a single disease situation compared to a mixed infection such as common blight and rust. Comparing plants and leaves of the same age and nutrition (which can affect symptom expression, such as with common blight (Patel and Walker, 1963)) may also create problems for a multiple disease-testing programme. With vertical resistance, the degree of susceptibility of the plant is irrelevant as only those rated as resistant are retained. With quantitative resistance (as with common and Ascochyta blight resistances in this study) there is no distinct line between resistance and susceptibility. As a result, selection is somewhat subjective to environmental conditions. Sequential inoculations

of rust, common blight, angular leaf spot and anthracnose pathogens were made on primary, first, second and third trifoliate leaves, respectively, by De Faria (1988) without any significant interaction between the diseases.

Glasshouse evaluation is usually far easier and more reliable as conditions are under greater control than in the field. For beans there is an added advantage that under glasshouse cultivation at least three generations per year can be obtained compared to a maximum of two in the field. Hence to obtain a bean breeding line with combined vertical resistances, maximal advancement would be obtained via glasshouse evaluation. This obviously precludes selection for agronomic characteristics such as lodging.

Genetic abnormalities sometimes arise in cross progenies such as those reported by Coyne (1965). More recently, cripple plants have been reported after crossing small-seeded cultivars with medium to large-seeded cultivars. Beans have two centres of domestication, with large-seeded types from the Andes and small-seeded types from Mexico (Evans, 1976). The two types differ in some respects, one of which is the dwarf lethal (DL) genes. Van Rheenen (1979) initially reported a sub-lethal combination of two dominant genes and Singh and Gutierrez (1984) suggested the link between the two groups. As few members of the groups have the relevant dwarf lethal gene, it is usually only an occasional cross that fails due to this problem. This is indeed fortunate, as most of the disease resistances were originally obtained from the small-seeded group (Gepts and Bliss, 1985; Melis 1989).

Plant growth habit is also strongly linked to seed size. Adams (1982) indicated the ideal growth habit as type-II (according to Van Schoonhoven and Pastor-Corales, 1987) which has a bush growth habit suitable for mechanical cultivation, but is also indeterminate and hence more adaptable to stress. Large-seeded types are usually type-I or type-III. Type-I is a determinate bush and type-III is a viny type plant prone to lodging and associated problems. The local cv. Bonus has a type-III growth habit. Kelly and Adams (1987) indicated difficulty in combining larger seed size and type-II growth habit and suggested cyclic recurrent selection was required. Beaver and Kelly (1994) successfully combined large seed size with a type-II bush by recurrent selection, in spite of an unfavourable association of the desired habit with small seed size in the first cross.

Bean rust has been known to overcome vertical resistance and hence the search for durable horizontal resistance. In this study it was decided to take several susceptible cultivars and, by recurrent selection, create a horizontally resistant cultivar for commercial production. Hill et al. (1963) reported good progress in breeding lucerne (Medicago sativa L.) resistant to Uromyces striatus Schroet. var medicaginis (Pass.) Arth. by recurrent selection. Sharp et al. (1976) obtained good resistance to stripe rust in winter wheat from susceptible or intermediate cultivars after recurrent selection. Lyons et al. (1987) found significant gains in breeding for resistance to white mould (Sclerotinia sclerotiorum (Lib.) de Bary) of beans by recurrent selection. Resistance mechanisms to white mould vary but are generally inherited quantitatively with low heritabilities. If Ascochyta blight becomes a major problem then the same method may have to be used as there are no oligogenic sources of resistance to Ascochyta blight at present.

Common beans (P.vulgaris) are naturally self-pollinating. Stoetzer (1984) found ca. 5 % natural cross-pollination under field conditions in Ethiopia and suggested carpenter bees (Xylocopa spp.) were the main cause. Brunner and Beaver (1988) found outcrossing rates of <1 to 4 % except in one case where it was 18 % in trials in Puerto Rico. Wells et al. (1988) found a higher degree of cross-pollination in the presence of bumble bees (Bombus sonorus Say) of 67 % in one cultivar, but a mean of 27 % for six cultivars tested in California. Ibrarra-Perez and Waines (1991) reported an average out-crossing rate of 8 % from two cultivars also tested in California. With the low degree of natural outcrossing it would be difficult to maintain sufficient progress in a recurrent crossing programme. Male sterility such as the indehiscent anther mutant of Wyatt (1984) would ensure a higher level of outcrossing. Pollen from plants that have indehiscent anthers is viable but selfing is prevented as it is not shed. Unfortunately, this mutant has been lost. No breeding work with cytoplasmic or genetic male sterility has been reported (Wells et al., 1988). Although good progress was made in breeding for horizontal resistance in wheat using a chemical male gametocide (Beek, 1983; De Milliano, 1983), equally good progress was made using more traditional crossing methods in Morocco (Pieters et al., 1990).

In order to estimate the possible success of a breeding programme,

information is required about the inheritance of a trait being selected for. With oligogenic inheritance it is relatively easy to estimate the number of genes involved. With polygenic inheritance, however, it is more difficult, as the effect of the environment obscures individual gene effects as with most horizontal resistance mechanisms. The relative heritability of the trait as a whole can be measured by a diallel crossing programme and comparison of the progeny. The nature of the genetic information obtained from a diallel depends on the nature of material included in the experimental design and the model upon which the analysis is based (Hayman, 1954; Griffing, 1956; Gardner and Eberhardt, 1966). Variations on the experimental design include using a full diallel or excluding the reciprocal crosses and/or parent lines. If one assumes the diallel parent lines are a random sample from a population then the genetic information gleaned from the diallel refers to the entire population. But in most cases the diallel parents are a specific set of lines deliberately chosen and as such, the information gleaned refers only to this subset.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Glasshouse cultivation

Plants were grown in composted pine bark or a mixture of soil and pine bark, with the latter favoured for seed production. Several pot sizes were used, the main sizes were 150 mm diameter (dia.)(5 - 7 plants were grown for short term use) and 250 mm dia. (upwards of 40 plants for short term use and usually five plants for seed production were grown). Roguing was facilitated by use of extruded polystyrene (Speedling®) trays in the seedling stages of the project to breed for multiple-disease resistance. A range of temperatures was used during this project but no supplementary lighting was provided. Watering and fertilization was done when necessary.

Thrips and occasionally aphids were controlled by dimethoate (Aphicide®) and red spider mites by tetradifon (Redspidercide®). Whiteflies were controlled by use of a sulphur burner.

The bean cultivars (a list is given in Appendix 1) used in this project were obtained from the bean germplasm maintained by the Oil and Protein Seed Centre (OPSC), Potchefstroom, or from Dr. R.J.M. Melis, Proseed, Ukulinga, Pietermaritzburg. Most of the cultivars were originally from CIAT.

3.2 Methods of pathogen storage

BCMV was stored in infected seed. The halo and common blight, angular leaf spot, anthracnose and Ascochyta blight pathogens were kept in infected leaves, dried and stored in a cold room (4°C). Rust urediospores were kept in a deep freezer (-10 to -20°C).

In the shorter term, anthracnose and Ascochyta blight fungal cultures were maintained on oatmeal agar (OMA) for three to six months. Re-isolations were carried out from infected plants to maintain pathogenicity of isolates.

Oatmeal agar was made by cooking 100 g oatmeal in 500 ml distilled water in a microwave oven at medium power for 10 minutes. Seventeen grammes of agar was melted in 500 ml of distilled water and this was added to the filtrate after sieving the oatmeal through a layer of melkdoek (similar to muslin cloth). Volume was made up to 1 ℓ and the mixture autoclaved for 15 minutes.

3.3 Inoculation technique

Plants were kept at 100 % relative humidity (r.h.) by one of three methods:
1. Dew chamber - Water evaporated by a hot water bath was condensed in the chamber by cooling in the surrounding room.
2. Plastic bags - Transpiration of the plants maintained 100 % r.h.
3. Switching off air-conditioning units of the section of the glasshouse, spraying water and leaving the tap dripping.

3.3.1 BCMV

Leaves of infected plants with typical mottling and/or leaf-curling symptoms were blended with 0.01 M (pH 7) phosphate buffer in a 10:1 (m/v) ratio and filtered through a layer of melkdoek. The filtrate was gently rubbed onto leaves with melkdoek and washed off with tap water shortly thereafter.

3.3.2 Common blight

Leaves with typical necrotic lesions bounded by chlorosis were blended with distilled water in a 10:1 (m/v) ratio and filtered through a layer of melkdoek. The filtrate was gently rubbed onto leaves with melkdoek and the leaves were then pierced with a florist's pin cushion or frog, numerous small nails set in a metal base (cf. Andrus, 1948). It was not necessary to maintain plants at 100 % r.h.

Plants used for bulking-up were often inoculated by screwing up a few infected leaves with a little tap water and rubbing this against the leaf.

3.3.3 Halo blight

Leaves with good halo symptoms and parts of the plant with chlorotic systemic symptoms were blended with distilled water in a 10:1 (m/v) ratio and filtered through a layer of melkdoek. Filtrate was gently rubbed onto leaves with melkdoek. Plants were maintained at 100 % r.h. overnight.

3.3.4 Angular leaf spot

Leaves with typical angular lesions and synnemata were shaken up with a dilute (0.05 %) solution of Tween 40® (polyoxyethylene sorbitane monopalmitane). This was filtered through a layer of melkdoek and the filtrate sprayed onto leaves. Plants were kept at 100 % r.h. overnight. As there was usually a mixed pathogen population in the inoculum, rust was also inoculated. This was not a problem in these experiments but can be avoided by aging the leaves and allowing the urediospores to lose viability.

3.3.5 Anthracnose

Cultures of C.lindemuthianum maintained on OMA were washed with distilled water and the washings (containing conidia) were sprayed onto the plants, which were then maintained at 100 % r.h. overnight. Alternatively, infected leaves were washed with distilled water to obtain the inoculum.

3.3.6 Ascochyta blight

Tests were done on the cvs. PC 235-4-E1, Carioca 80, PAI 6 and PAI 127 to determine the best method of inoculation. Plants were sprayed with a conidial suspension and half of the plants were then damaged with a pencil; all plants were then maintained at 100 % r.h. for three or four days. Following the results of this test, it was decided to use the following method: Cultures of P.exigua maintained on OMA were washed with distilled water and the washings (containing conidia) were wiped onto leaves. The leaves were then pierced with a florist's pin cushion. Plants were maintained at 100 % r.h. for three days.

3.3.7 Rust

Suspensions of urediospores in 0.05 % Tween 40® were sprayed onto plants, which were maintained at 100 % r.h. overnight. Alternatively, if only a small quantity of urediospores was available for inoculation, they were transferred by means of a camel-hair paint brush. Fresh urediospores were used whenever possible.

3.4 Crossing

Bean crosses were made without emasculation on buds one day before opening. The standard was opened and folded back, the style was forced out by applying pressure to base of the wings and a style from a naturally opened flower with pollen was hooked under the style of the flower to be crossed. The standard was then closed and flower labelled. Selfing is usually avoided as beans are protogynaceous. Crosses were usually done so that plants with dominant marker genes for easily observed characters (such as pink versus white flowers) were crossed onto plants with the recessive gene.

There was a high success rate of pollination (>95 %) but the number of abscising pods depended on the load of the plant. Usually, a maximum of five pods per plant was used. The number of selfs depended on the number of crosses being done and the time available to be meticulous. Many selfs were obtained when, for example, six hours of crossing were done per day. Following the increase of the F_1 of the rust cross (RC), ca. 10 % were selfs based on seed colour. Fouilloux and Bannerot (1988) suggested that it took five minutes of crossing time per hybrid seed obtained. The crosses done, excluding the horizontal resistance programme, are given in Appendix 2.

3.5 Sources of resistance

This section is in addition to information supplied in section 2.2 The diseases and sources of resistance.

3.5.1 BCMV

I-gene resistance to BCMV is common in many current cultivars of beans. Of the cultivars used in the multiple-disease resistance crossing programme, only GN Nebraska No.1 Selection 27 and GN Tara did not have the I-gene. All the cultivars used in the horizontal rust resistance crossing programme were deliberately chosen for having the I-gene.

Temple and Morales (1986) reported very close linkage between an undesirable testa colour and the I-gene. This linkage was broken and the cvs UI 50 and UI 51 were released; these were used to introduce the I-gene into the Potchefstroom Cross (PC) lines used in this study (Edington, 1989a).

3.5.2 Common and halo blight

The cvs GN Nebraska No.1 Selection 27 and GN Tara have quantitative resistance to common blight and race non-specific resistance to halo blight. In addition, GN Tara has race-specific resistance to race 1 of the halo blight bacterium hence the need to test with race 2. The cv. GN Jules also has race non-specific resistance to halo blight, but in a test with halo blight from Potchefstroom (Race 1), halos of 5 - 6 mm in diameter compared to those of GN Nebraska No.1 Selection 27 of 3 - 5 mm were found. After natural infection with halo blight from Potchefstroom of GN Nebraska No. 1 selection 27 and GN Tara pods, lesions of ca. 2 mm diameter were observed, although lesion size was more variable for GN Tara.

The cv. XAN 159 was recently obtained from the OPSC. It exhibited no common blight symptoms in field trials at Delmas (A.J.Liebenberg, pers. comm., 1991) and has hypersensitive resistance to common blight from Potchefstroom and Ukulinga (Unpublished data). It is reported to have very good leaf and pod resistance to Dominican Republic, Puerto Rican and Nebraskan isolates of the bacterium (Arnaud-Santana et al., 1991). XAN 159 was derived from a P.acutifolius interspecific cross (Anon., 1984) and has a single dominant gene for resistance. Incorporation of this resistance would have been far easier than that of the great northern beans but was initially not available. It could be utilized in future crosses.

3.5.3 Angular leaf spot

The cv. Carioca 80 was resistant to angular leaf spot in trials at KwaGubeshe (near Pietermaritzburg), Natal in 1987/88 and 1988/89 (Anon. 1988a and 1989). Carioca 80 is a small-seeded carioca bean and in the two trials, it was the seventh and second highest yielding cultivar, respectively (Mkuze was sixth and eighth, respectively).

3.5.4 Anthracnose

Edington (1990) identified a race of the fungus from Natal which closely resembles the Alpha-Brazil race except that Evolutie was susceptible. Fouilloux (1975) reported the cv. Cornell 49242 as susceptible to the Alpha-Brazil race but Drijfhout and Davis (1989) indicated it was resistant. Cornell 49242 is resistant to the Natal race and was used. Cornell 49242 is a small-seeded black bean and resistance is conditioned by the Are-gene.

3.5.5 Ascochyta blight

At this stage only poor sources of Ascochyta blight resistance are available in South Africa. Anonymous (1988a) found the carioca cultivars to be quite susceptible but the cvs PAI 6 (second highest yielding cultivar in the trial) and PAI 127 to have a degree of resistance in trials conducted at KwaGubeshe. Both are small-seeded, reverse red-speckled types.

In 1990, Mr. G. Nevill (Pers. comm., 1990) planted an Ascochyta blight resistance trial at Cedara (near Pietermaritzburg), Natal. Most of the cultivars were too late-maturing to be of any use but seed of ASC 4, BAT 795 and VRA 81018 was obtained.

3.5.6 Rust

The cvs Carioca 80, PAI 6 and PAI 127 were reported as rust-resistant at KwaGubeshe in 1988 (Anon., 1988a). Carioca 80 and PAI 6 were also reported as resistant in 1989 (Anon., 1989), PAI 127 was not included in the trial. PAI 6 and PAI 127 were susceptible to rust collected at Potchefstroom but Carioca 80 was resistant (unpublished data).

The cvs. BAT 1427 and ZAA 49 were rated as fully resistant in an International Bean Rust Nursery (IBRN) trial planted at Delmas in the 1987/88 season but proved to be susceptible to rust from Potchefstroom after glasshouse tests (unpublished data). Cornell 49242 was also rated as partially resistant in Delmas IBRN trials and was susceptible to rust from Potchefstroom. Another cultivar, Riz 43, fully resistant at Delmas and susceptible to rust from Potchefstroom was not used in this project. The speckled sugar beans PC 252-D2 and PC 256-D1 (shortly to be released as the cv. Kranskop) had good but not full resistance to rust at Dundee, Natal (Edington, 1989a) and Delmas, but poor resistance at Ukulinga (unpublished data). PC 232-1-D2 and PC 235-4-E1 are speckled sugar types rejected by the OPSC but found to be rust resistant and high yielding by Mr J. Chapman (Grain Crops Research Institute, Dundee) at Dundee and Mr A. Jarvie (Pannar Seed, Greytown, Natal) at Delmas, respectively (unpublished data). All the PC lines are susceptible to the single-pustule isolate (SPI) M1B.

3.6 Race surveys

A knowledge of what races are present in an inoculum source can aid a breeder in determining the possible durability of resistance sources. Comprehensive studies have yet to be done on the races of the halo blight, angular leaf spot, anthracnose and rust pathogens in South Africa. A study of the *P.griseola* races present in South Africa has recently been initiated by Mrs. M.M. Liebenberg at the OPSC, Potchefstroom.

3.6.1 Halo blight

The four differential cvs Canadian Wonder, Red Mexican UI 3, Tendergreen and Edmund were inoculated with isolates of the bacterium from Warden (Orange Free State) and Delmas (both obtained from the OPSC) and samples of infected material from Greytown, Potchefstroom and Ukulinga. Ten to twelve plants of each cultivar were inoculated in the primary leaves prior to full expansion and rated 14 days post inoculation.

3.6.2 Anthracnose

Cultures of C.lindemuthianum from Cedara, KwaGubeshe, Empangeni and Greytown (all in Natal) were obtained after plating out small sections of infected leaves onto water agar. These were maintained on OMA before inoculation of the differential cultivars. Ten to twelve plants of each cultivar were inoculated on the primary leaves (three-quarters expanded) and rated 14 days post inoculation.

3.6.3 Rust

Single-pustule isolates of rust, M1B (from Pietermaritzburg) and U6A (Ukulinga), were obtained from Mr B.D. Garman, Department of Microbiology and Plant Pathology, University of Natal, Pietermaritzburg (UNP). Three SPI's from Bethal, Beth (U93Ba46), U93Ba32 and U93Ba40, were provided by Mrs. M.M. Liebenberg, OPSC, in 1994. Further SPI's were obtained from rust samples from Cedara provided by Mr E.B. Birch and from Potchefstroom provided by Dr A.J. Liebenberg, both in 1992. Urediospores from a single pustule in the cv Teebus were transferred to healthy Teebus plants by means of a camel-hair paint brush. A single urediosorus from this infection was then used to inoculate further Teebus plants. The final Teebus plants were used to bulk-up the infection for inoculation of the differential cultivars. The SPI M1B is avirulent and the SPI U6A causes a ring reaction in the cv. Teebus. An inoculation of a sample of rust from Ukulinga in 1992 resulted predominantly in ring symptoms in Teebus, identical to those caused by the SPI U6A and as a result no single-pustule isolations were attempted with this rust. Ten to twelve plants of each differential cultivar were inoculated on the primary leaves prior to full expansion with the single pustule isolates and were rated 14 days post inoculation.

Further single-pustule isolates were made from a sample of rust from Wartburg, Natal, provided by Mr A. Jarvie in 1994. The same procedure as above was carried out but on cv. KID 16 plants before inoculation of rust differential cultivars.

3.7 Breeding for horizontal resistance to rust

3.7.1 Breeding trial

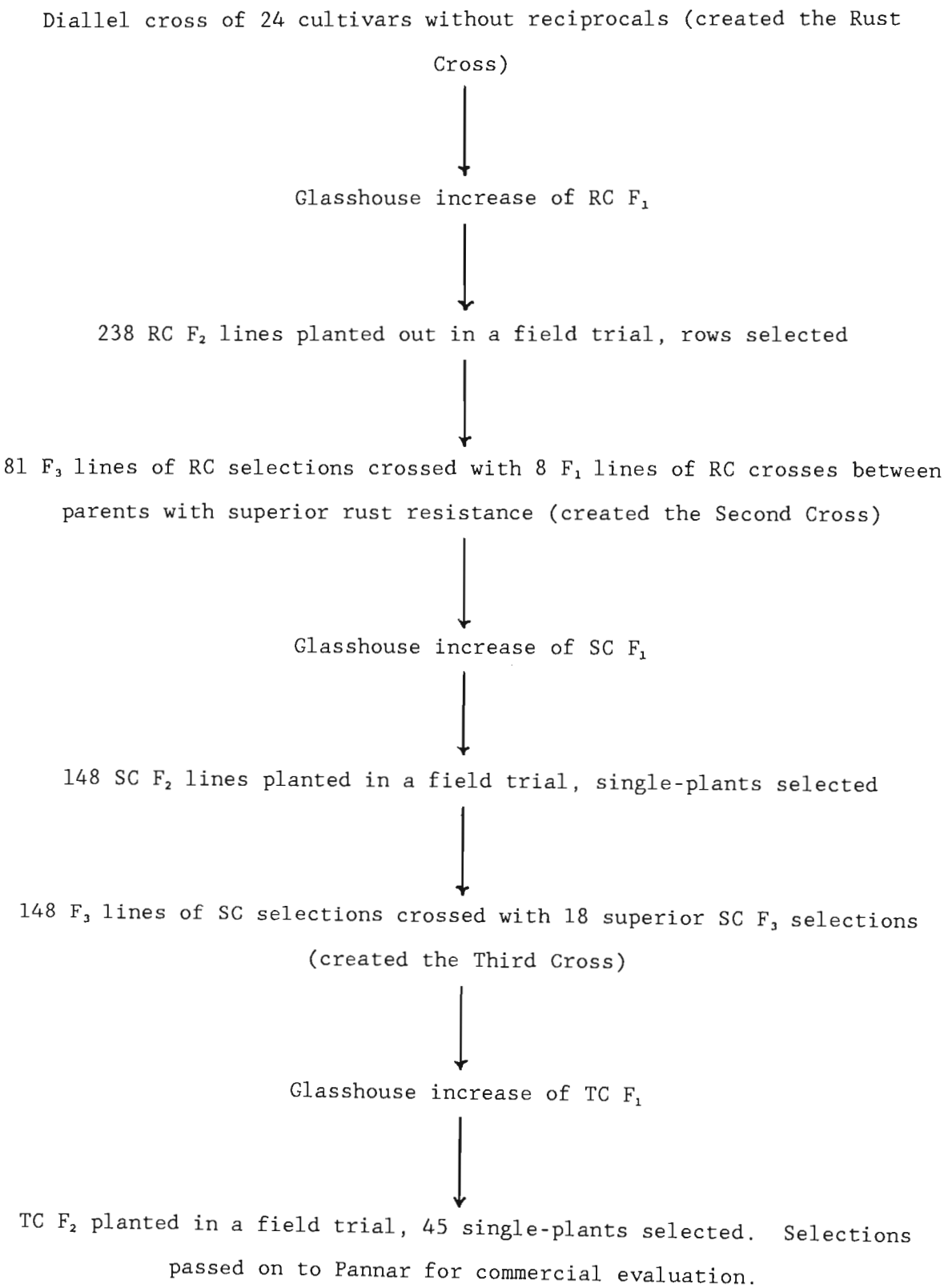
Twenty-seven cultivars were selected from CIAT adaptation and rust nurseries grown by the OPSC at Delmas. Selection was based on rust susceptibility, large seed size, presence of the I-gene for resistance to bean common mosaic virus and absence of severe halo blight symptoms. The 27 cultivars and five OPSC breeding lines (PC-lines) were planted in the disease garden at the UNP on the 19th of September, 1988. To prevent complications of a differential race reaction (Parlevliet 1983), single-pustule isolates were used in this and following trials (checks were made by planting cultivars resistant to the SPI). The trial was inoculated with the SPI M1B as the cv. Teebus, planted as a spreader, proved to be resistant. Teebus is usually very susceptible to rust in field trials (Internal OPSC Reports). Ratings of pustule size were done on the 18th of November according to the CIAT scale (Van Schoonhoven and Pastor-Corrales, 1987). The cvs PVA 2280, ICA 15489, AND 366, GLP X 1125, Golden Gate Wax, PC 349-7 and PC 351-19 were resistant and work was discontinued with them. The cv. 1274 was susceptible but as it was similar (and probably closely related with a common parent) to the cvs 1266, 1271 and 1273, was also discarded.

Epicure was also resistant to the SPI M1B but developed symptoms of ring necrosis of the slightly incompatible reaction upon infection with local races. Crosses were made with Epicure but abandoned as it became obvious that the slightly incompatible reaction was partial vertical resistance. Further work on the slightly incompatible reaction is given in section 3.8.

The remaining 23 susceptible cultivars and Epicure were crossed in a diallel without reciprocals (rust cross). The RC F_1 was increased in the glasshouse before planting in a field trial at Ukulinga (details of experimental design and site are given in Appendix 3). Of the 276 possible crosses, 238 F_2 lines were planted.

Border rows of the cv. PC 222-5-6-P2 of 5 m inter- and 0.1 m intra-row spacing were planted 14 days before the main trial on the 5th of October, 1989. Four-metre test rows were planted on the 19th and 20th of October,

Figure 3. Crossing diagram for the horizontal resistance programme.



perpendicular to the border rows, at 0.75 m inter- and 0.1 m intra-row spacings. The 0.5 m gap between the ends of the test rows and the border rows allowed easy access. All planting was done by hand. Besides the RC lines, three replications of the parent cultivars were also planted.

Fertilizer (2N:3P:2K, 6.3 %:9.4 %: 6.3 %) was worked into the land before planting at a rate of 400 kg ha^{-1}. Weed control was by hand hoeing; flooding prevented early weed control. Inclement weather initially led to flooding with excessive rainfall, a spring opened up in the middle of the trial. This was followed by drought later in the season. Irrigation was applied as required. The land had been previously used for bean trials and root rots, initially aided by the wet weather, were prevalent in the trial.

Border rows were successfully inoculated with a suspension of urediospores of the SPI M1B in a 0.05 % solution of Tween 40® by a hand sprayer on the 30th of October. No rust symptoms were observed on the cvs Teebus and Epicure in the trial until late, indicating a pure epidemic. Rust ratings were made on the 8th, 15th and 21st of December. On the 8th of December plants were rated for pustule size and % leaf area affected by rust. Pustule size ratings were done according to the CIAT scale, but little variation was noted and no further ratings of pustule size were done. The percentage leaf area affected by rust was adjudged by viewing the row.

Poor environmental conditions led to a negligible yield and no yield results were taken for the parent cultivars. In many of RC lines there was no yield and in the others, only a handful of seed was harvested. Several late-maturing lines were rejected to favour earliness. As the cv. KID 16 closely resembled KID 20 and was slightly more susceptible (both were very susceptible), only the KID 20 crosses were retained.

Eighty-one RC F_3 lines were planted in the glasshouse for crosses with RC F_1 lines of crosses between parents that had shown good field resistance to the SPI M1B and where such seed was left over after the initial RC F_1 increase. The F_1's were RC 69 (1266 x 1312), RC 72 (1266 x ICA 15522), RC 79 (1266 x AND 340), RC 126 (1312 x ICA 15522), RC 133 (1312 x AND 340), RC 139 (1312 x 312 AND 689), RC 184 (ICA 15522 x 312 AND 689) and RC 254 (AND 340 x 312 AND 689). This generated 148 lines of the second cross (SC),

which were planted at Ukulinga after the F_1 was increased in the glasshouse.

The SC trial was identical to the first trial except that the inter-row spacing of the border rows was 4 m and the length of the test row was 3 m. In addition, to hand hoeing, weed control was aided by a pre-planting application of Trifluralin (Treflan®) at a rate of 1 ℓ ha^{-1}.

Border and test rows were planted on the 9th and 23rd of October, 1990, respectively and the border rows were successfully inoculated on the 8th of October. Ratings of % leaf area affected by rust were done on the 18th and 27th of December and 3rd and 10th of January. Weather conditions were more favourable and periods of short drought were compensated for by irrigation. Yields of the parent cultivars were measured by harvesting the whole row.

One hundred and forty-eight single-plant selections were made based on the rust resistance of the row from which it was taken and seed characteristics. Of these selections, 18 were deemed to be superior and these were crossed with the other selections and amongst themselves. This generated 181 lines of the third cross (TC) which was planted at Ukulinga after the increase of the F_1 in the glasshouse.

The TC trial was identical to the second trial except that Treflan® was applied at a rate of 1.5 ℓ ha^{-1}. Border and test rows were planted on the 10th of January and 27th and 28th of January, 1992, respectively, and the border rows were inoculated on the 1st of February. Parent cultivars were rated for % leaf area affected by rust on the 17th, 26th and 31st of March and the 8th and 16th of April and their yields were measured. Single-plant selections were again made based on the rust resistance of the row from which it was taken and seed characteristics.

3.7.2 Diallel trial

In order to examine the nature of the rust resistance of the cultivars, a full F_1 diallel with reciprocals was planted each year alongside the breeding trial. The diallel trial was treated under the same cultural conditions as the breeding trial except that the inter-border row spacing was 3 m and the intra-test row spacing was 0.2 m. In the first trial 10

seeds were planted, but in the other two, a maximum of 8 seeds per replication was planted. Three replications of the parent cultivars and two of the crosses, where enough seed was available, were planted.

The eight cultivars used in the first trial were chosen somewhat arbitrarily as no information was available on the resistances of the cultivars at that stage. With the results of the first breeding trial, the cvs PC 222-5-6-P2, 1273 and KID 16 were chosen for being susceptible, ICA 15522, GLP X 1132 and 269 AND 631 for being moderately resistant and 1266 and 259 AND 621 for being the most resistant. The cvs 1273 and 1266 are probably closely related, they look similar, but differ quite markedly in rust resistance. Except for GLP X 1132 and 269 AND 631, the cultivars are determinate.

The first trial was abandoned after the first rating but greater success was achieved with the following two trials. Ratings of % leaf area affected by rust and final yield of the individual plants were recorded.

3.7.3 Infection trials with other single-pustule isolates

To test for the partial resistance of the 23 parent cultivars to other SPI's natural conditions of infection are required. Three pots of each cultivar were planted in a plastic tunnel without heating or cooling and with half-walls of fine mesh (ca. 4 mm^2). Overhead boom irrigation aided disease spread. Unfortunately, the only time when the tunnel could be used without fear of contamination was in the autumn when the onset of cold nights curtailed rust infection. In the test with the SPI Ced-1 the plants were transferred to a greenhouse with air conditioning. The air conditioning was expected to distribute the spores evenly during the day and at night it was switched off to provide 100 % r.h.. Even with glasshouse units being heated on either side of the unit, it was still too cold for rust infection.

A small field trial was planted at Ukulinga on the 7th of March, 1994, consisting of 2 m rows of the 23 parent cultivars with 0.1 m and 0.75 m intra- and inter-row spacings, with no replication. The trial was irrigated and hoed as necessary. The cv. Bonus planted alongside the trial was inoculated on the 17th of March, 1994, with the SPI Beth as a source of inoculum for natural infection of the trial.

3.7.4 Leaf hair counts

Plants of the cultivars 1266, 1273, ICA 15522 and KID 16 were grown to just past full expansion of the first trifoliate. The middle leaflet of the first trifoliate was examined for adaxial and abaxial leaf hairs using a Zeiss Stemi SV6 dissecting microscope.

Fifteen plants of each of the 23 parent cultivars were grown to past full expansion of the first trifoliate. The abaxial side of the middle leaflet of the first trifoliate was examined for the presence of leaf hairs. At a later stage, the third trifoliate of glabrous cultivars was inspected for the presence of leaf hairs.

Further plants of the cvs 1266 and 1273 were grown to a stage past full expansion of the third trifoliate. Abaxial leaf hairs were counted using the Zeiss Stemi SV6 dissecting microscope.

Cultivars used as parents in the second and third diallel crosses were grown to the second trifoliate stage so that the first trifoliate was fully expanded. The middle leaflet of the first trifoliate was removed and leaf hairs within a field of view of a Wild dissecting microscope were counted on the adaxial side. In the first test, four counts were made per leaflet and three leaflets per cultivar. In the second and third tests, three counts were made per leaflet and eight leaflets per cultivar. Unfortunately, in the first test, the area viewed and the total leaflet area were not recorded but were probably similar to the latter two. In the other tests the area viewed was 0.5 mm^2 and the area of each leaflet was measured by a Delta-T Devices image analyser after counting.

3.7.5 Stoma number

Leaflets of several cultivars were viewed for stomata at 100 times magnification under a Zeis Axiophot microscope with incident light.

3.7.6 Latent period tests

The plants used for the second and third leaf adaxial leaf hair counts in

section 3.7.4 were initially used for a latent period test. Five plants per pot and two pots per cultivar were maintained at 22°C day/12°C night, except for inoculation of the primary leaves at full expansion with the SPI M1B when they were kept overnight in a dew chamber. Inoculation was non-quantitative and the order of pustule eruption was judged on a visual basis.

Additional plants of 1266, 1273, ICA 15522 and KID 16 plants were inoculated on the unifoliate leaves and this infection was used as the source of urediospores for the inoculation of the second trifoliate leaves of the same plants. Observations were made to see if there were differences in latent period of the second trifoliate leaves of the cultivars.

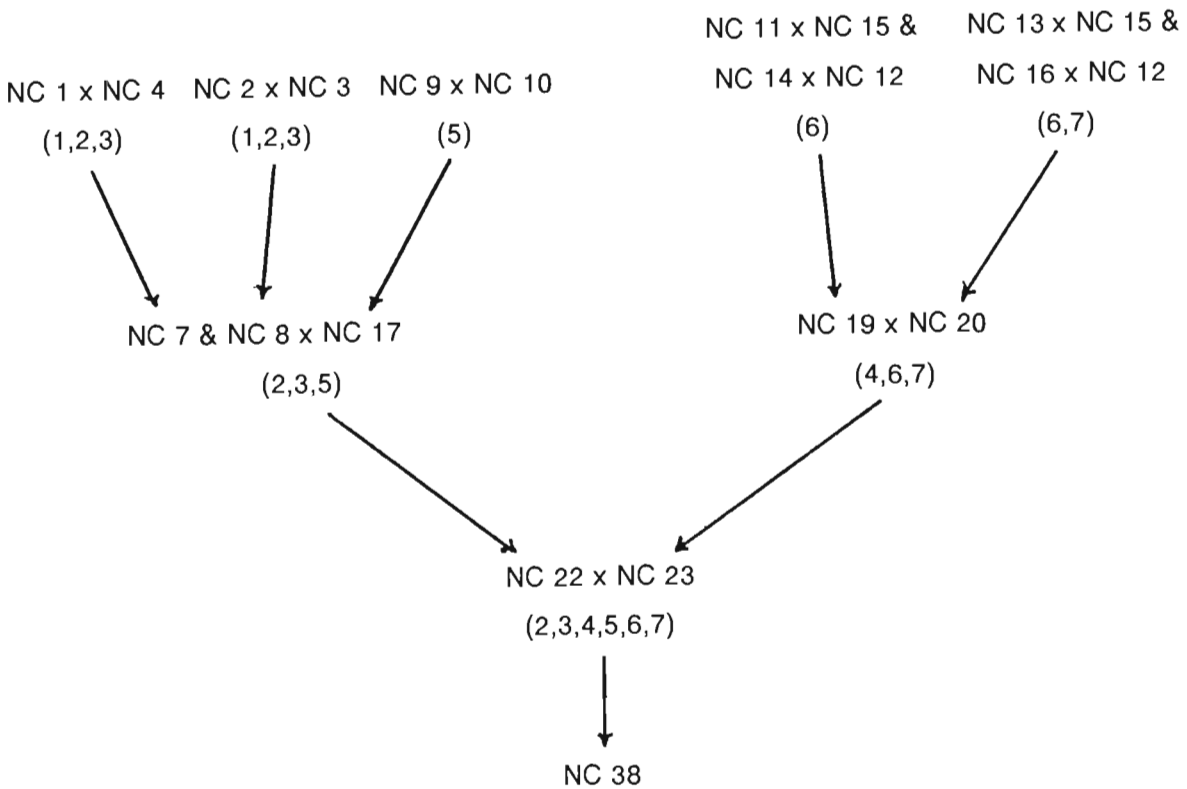
Ten plants of the 23 parent cultivars were inoculated at the unifoliate stage with the SPI Wart-2, as a further test of the latent period in unifoliate leaves. Six plants of the eight diallel cultivars were grown to past the fourth trifoliate stage and inoculated with the the SPI Beth.

3.8 Breeding for multiple disease resistance

Crosses were made in a reverse dichotomy (Figure 4.) and at each F_2 stage the plants were tested with the relevant pathogen(s). In some cases where the dominant resistances to angular leaf spot, anthracnose and rust pathogens were involved and inoculum was available, the F_1 was also tested. Where plants with the dominant resistances tested positive for resistance, the genes may be heterozygous and additional testing will help to eliminate unwanted susceptible alleles. Where resistance was oligogenic, susceptible plants were rogued, but where resistance was quantitative (Ascochyta and common blights), 30 - 50 % of the plants with a greater degree of symptom development were rogued. The number of plants tested for resistance at each stage varied, but was never less than 50 unless a F_1 cross was tested.

The great northern cvs GN Nebraska No.1 Selection 27 and GN Tara were both crossed with PC 252-D2 and PC 256-D1. PC 252-D2 and PC 256-D1 had improved rust resistance compared to Bonus and outyielded it by an average of 34 % and 38 %, respectively, in checkrow trials at Bethlehem, Orange Free State and Delmas. In addition, both have the I-gene linked to a favourable testa

Figure 4. Crossing diagram for the vertical resistance programme.



NC 1 = GN No. 27 Sel. 1 x PC 252-D2 (1)
NC 2 = GN No. 27 Sel. 1 x PC 256-D1 (1)
NC 3 = GN Tara x PC 252-D2 (1)
NC 4 = GN Tara x PC 256-D1 (1)
NC 9 = Cornell 49242 x BAT 1427 (5)
NC 10 = Cornell 49242 x ZAA 49 (5)
NC 11 = PC 232-1-D2 x PAI 6 (4,6)
NC 12 = PC 232-1-D2 x PAI 127 (4,6)
NC 13 = PC 232-1-D2 x Carioca 80 (4,7)
NC 14 = PC 235-4-E1 x PAI 6 (4,6)
NC 15 = PC 235-4-E1 x PAI 127 (4,6)
NC 16 = PC 235-4-E1 x Carioca 80 (4,7)

The figures in parentheses indicate which disease resistance(s) the F_2 of the cross was tested for; the diseases were:

1 = BCMV 2 = Halo blight 3 = Common blight
4 = Angular leaf spot 5 = Anthracnose 6 = Ascochyta blight
7 = Rust

colour (Edington, 1989a). Crosses between GN Tara and Charlevoix (Natal cross (NC) 5 & 6) indicated the presence of specific resistance to race 1 of the halo blight bacterium besides the race non-specific resistance.

The F_2 plants were inoculated with BCMV in the primary leaves just prior to full expansion. Plants were maintained at day temperatures above 27 °C and after four days, unifoliate leaves with pinpoint necrotic lesions were removed. Plants without lesions were inoculated again and after another four days, any plants that did not display pinpoint lesions were rogued. The cross between GN Nebraska No.1 Selection 27 and PC 252-D2 (NC 1) was then crossed with the cross between GN Tara and PC 256-D1 (NC 4) and vice versa (NC 2 x NC 3). The F_2 (NC 7 and 8, later referred to as NC 7) was again tested for presence of the I-gene and the first trifoliate leaves were inoculated with halo blight from Potchefstroom (race 1). Due to problems with finding a suitable common blight host cultivar, inoculation of the F_2 with the bacterium was unsuccessful but that of the F_3 was.

The cv. Cornell 49242 was crossed with BAT 1427 (NC 9) and ZAA 49 (NC 10) and these crosses were crossed together (NC 17). BAT 1427 and ZAA 49 are large-seeded types with an undesirable seed colour, but were resistant to rust at Delmas in the 1987/88 season. The F_2 of each stage was tested for anthracnose resistance. Some of the smaller seeds of the NC 17 F_3 were discarded. The rust resistances of BAT 1427 and ZAA 49 were disregarded after found to be susceptible to rust from Potchefstroom and as the rust race situation in South Africa is still unclear. It was decided to use Carioca 80 as the source of rust resistance.

NC 7 was then crossed with NC 17 and the F_2 (NC 22) tested for common and halo blight and anthracnose resistances.

The cvs PAI 6, PAI 127 and Carioca 80 were crossed with both PC 232-1-D2 (NC 11, NC 12 and NC 13, respectively) and PC 235-4-E1 (NC 14, NC 15 and NC 16, respectively). The PAI F_2's were tested for Ascochyta and angular leaf spot resistances. The Carioca 80 F_2's were tested for angular leaf spot and rust (from Potchefstroom) resistances. The PAI 6 cross with PC 232-1-D2 was crossed with the PAI 127 cross with PC 235-4-E3 and vice versa (both NC 19) and the F_2 was tested for Asochyta blight resistance. The Carioca 80 cross

with PC 232-1-D2 was crossed with the PAI 127 cross with PC 235-4-E1 and the Carioca 80 cross with PC 235-4-E1 with the PAI 6 cross with PC 232-1-D2 (both NC 20) and tested for rust and Ascochyta blight resistances. NC 19 and NC 20 were crossed and the F_2 (NC 23) was tested for Ascochyta, angular leaf spot and rust resistances.

NC 22 was crossed with NC 23 and the F_2 (NC 38) was tested for the following resistances in chronological order: anthracnose, halo blight (from Delmas; race 2), angular leaf spot and rust together and Ascochyta and common blights together (aided by overhead boom irrigation).

The final cross was advanced a generation by single-seed descent by Mr A. Jarvie and plants of the F_4 were planted in a field trial in the disease garden. Five rows of NC 38 seed was planted at 0.1 m intra- and 0.75 inter-row spacing on the 28th of January, 1994 in the University's disease garden. The rows were surronded by the cv. Bonus planted at a spacing of 0.1 m, as a disease spreader. Two rows of Bonus and a single row of the cv. Teebus were planted in the middle of the trial perpendicular to the NC 38 rows as a test of infection of the trial.

The whole trial was inoculated with P.syringae pv. phaseolcola (from Greytown) on the 16th of February, C.lindemuthianum (Greytown) on the 17th of February, P.exigua var. exigua (Cedara) on the 6th of March, P.griseola (Cedara) on the 7th of March, P.griseola (Greytown) on the 23rd of March, X.campestris pv. phaseoli (Greytown) on the 24th of March.

3.8.1 Field trials by Pannar Seed

An advantage of this breeding programme was that redundant material could be used for other purposes. Once a cross has been done, the parent lines can be tested in the field as they were now superfluous. All redundant material was passed onto Mr A. Jarvie of Pannar Seed for inclusion in his field trials. This allowed selection of potential material which might otherwise have been discarded. These lines could possibly be used in a back-cross with lines from further into the multiple-disease resistance programme. Some details of the site are given in Appendix 3.

3.9 Genetics of resistance

3.9.1 Angular leaf spot

The F_1 and F_2 of the crosses betweeen PAI 127 and Carioca 80 and PC 232-1-D2 (NC 12 and NC 13, respectively) were tested for resistance to angular leaf spot. Carioca 80 was crossed with PC 235-4-E1 (NC 16) and PAI 127 (NC 26) and the F_2 were tested for angular leaf spot resistance.

3.9.2 Full rust resistance

The cv. Mkuze is known to be rust resistant in the main bean growing areas of South Africa according to the results of the National Bean Cultivar Trials (Internal Report of the OPSC, 1991). Mkuze was crossed with Carioca 80 (NC 24) and the F_2 was tested with rust from Pietermaritzburg.

3.9.3 The slightly incompatible reaction of rust

Yarwood (1951) reported a ring reaction very similar to that found in this study of Epicure plants to that in rust infected plants followed by infection with five viruses. As a result, tobacco cv. Samsun plants were inoculated to test for virus symptoms. The same inoculation technique as used for BCMV inoculations was used other than that healthy bean leaves were utilized.

Epicure was initially investigated as a possible source of rust resistance. Ten to twelve plants of Epicure were inoculated with samples of rust from Cedara, Empangeni and Potchefstroom on the unifoliates prior to full expansion. This was besides inoculations conducted in section 3.6.3 with single-pustule isolates.

The degree of ring necrosis observed in Epicure in the initial testing of the 32 cultivars for resistance to the SPI M1B in the University's disease garden was affected by temperature. Epicure plants were inoculated and maintained at 20/20°C and 30/20°C to test for temperature-sensitivity. A similar test was conducted with plants of NC 16 bearing ring necrosis after infection with the SPI M1B. Plants were maintained for 4 and 8 hours at

30°C, respectively, when they were transferred to join other plants at 18°C, at which temperature inoculation occurred. One group was maintained at 30/20°C, including the period of inoculation. In addition, another group was maintained at 18°C before inoculation and overnight thereafter to allow infection before transfer to 30/20°C day and night.

Epicure was crossed with PC 235-4-E3 (RC 53, i.e. from the horizontal breeding programme) and PVA 2280 was crossed with PC 235-4-E1 (NC 21) and the F_2's were inoculated with the SPI M1B. In addition, the F_3 of RC 53 was tested with the SPI M1B. Epicure, Redlands Greenleaf C, PC 235-4-E3, RC 10 F_1 (PC 222-5-6-P2 x Epicure), RC 73 F_2 (1266 x Epicure) and a RC F_2 of an unknown cross with Epicure were planted in the disease garden in August of 1989 and natural infection occurred. Epicure was crossed with Redlands Greenleaf C (NC 28) and the F_2 was inoculated with rust from Potchefstroom.

Two sets of the F_2 plants of the cross between Carioca 80 and PC 235-4-E1 (NC 16) were tested with rust from Potchefstroom and Pietermaritzburg, respectively. Plants of NC 16 bearing ring symptoms were crossed with PC 222-5-6-P2 (NC 27) and inoculated with the SPI M1B.

PI 165426 was found to have ring symptoms after natural infection with rust from Potchefstroom (it is resistant to the SPI M1B). It was crossed with Epicure (NC 37) and two sets of the F_2 plants were tested with the SPI's Ced-1 and Potch-1, respectively. Plants bearing ring symptoms derived from crosses with Epicure (RC 53) and Carioca 80 (NC 16) were crossed together (NC 42) and the F_2 was inoculated with the SPI M1B.

Plants of the cross RC 53 which had given mild and severe ring symptoms upon infection with the SPI M1B were crossed with PC 222-5-6-P2 (NC 40 & NC 41, respectively) and the F_1 and F_2 were inoculated with the SPI M1B.

A cross (NC 18) was made to introduce rust resistance into the cv. Broadacres (a green speckled or pinto type) from Mkuze. Some of the F_2 plants exhibited ring symptoms after inoculation with rust. The F_3 were used in crosses (NC 25) with NC 16 plants that had borne ring lesions.

The eight diallel cultivars, PC 222-5-6-P2, 1266, 1273, ICA 15522, KID 16,

GLP X 1132, 259 AND 621 and 269 AND 631, were crossed with RC 53 plants bearing ring symptoms (NC 29 to NC 36, respectively). The F_1 of these crosses was inoculated with the SPI M1B in the unifoliate leaves. Further inoculations of these plants on the second trifoliate leaf and the whole plant at flowering time with the SPI M1B failed.

3.10 Breeding for Ascochyta blight resistance

Ascochyta blight is, at present, only of local importance in Natal. Its precise importance is not known, but with the increasing cultivation of the more susceptible cv. Mkuze and cultivars bred from carioca-type sources it may be worth more attention.

Three isolates of the fungus from Cedara, KwaGubeshe and Ixopo (Natal) were maintained on OMA. Differences in growth rates on agar were observed with the KwaGubeshe isolate being quite a lot slower. Inoculations to maintain the pathogenicity and re-isolate the isolates also indicated the slower growth of the KwaGubeshe isolate *in vivo*.

As noted in 3.8, PC 232-1-D2 and PC 235-4-E1 were crossed with PAI 6 and PAI 127 and the resultant NC 19 was tested for Ascochyta resistance. The NC 19 F_3 was planted in the disease garden with Mkuze, Carioca 80, PAI 6 and PAI 127. Single-plant selections from NC 19 were made and some seed was given to Mr A. Jarvie (Pannar Seed) for field testing. A further planting in the disease garden was made with the cvs Mkuze, ASC 4, BAT 795 and VRA 81018 and NC 19. Conidia were sprayed onto the plants after poor natural infection was noted, and a reasonable disease spread was obtained.

Plants of NC 19 were crossed with BAT 795 (NC 39) and the F_2 was planted in the disease garden with NC 19 and the cvs BAT 795 and Mkuze (the latter cultivar failed to emerge).

CHAPTER FOUR

RESULTS

4.1 Inoculation technique

4.1.1 BCMV

Non-destructive testing for the presence of the I-gene has previously involved removing and inoculating a leaflet (Edington, 1989a). Tests in this project were done by inoculating a leaf still attached to the plant. Loss to "blackroot" was prevented by removing inoculated leaves as soon as possible after necrotic lesions formed. A susceptible plant can also form necrotic lesions (Trujillo and Saettler, 1972; Wassimi *et al.*, 1991), but these lesions tend to form later and are more diffuse. In one test, 5 % of the plants retained as positive developed typical mosaic symptoms. Thus those that give a false positive will most probably identify themselves. In the same test, 8 % of the plants were lost due to "blackroot". It is possible that this method may select against plants particularly susceptible to "blackroot". To test a cultivar for the presence of the I-gene, at least ten plants should be subjected to a lethal test for "blackroot".

4.1.2 Common blight

Initial tests with common blight failed until Nep 2 was used as the inoculum source and plants were maintained at day temperatures of greater than 25°C.

4.1.3 Ascochyta blight

Plants of PC 235-4-E1, Carioca 80, PAI 6 and PAI 127 inoculated by spraying a conidium suspension alone had little infection compared to plants sprayed and the leaf damaged by pencil scoring. The fungus is a weak pathogen and often requires damage for infection. Damage was standardized by use of a florist's pin cushion. Spraying often resulted in stem infection, leading to girdling and loss of the plant. By wiping the conidial suspension onto leaves, initial stem infection was avoided. If the infected leaf does not senesce, infection spreads up the petiole and into the stem. The leaf must

be removed before infection reaches the stem to save a plant. Three days post-inoculation (dpi) at 100 % r.h. was adequate for all inoculations.

In one test, plants were transferred to a plastic tunnel with overhead boom irrigation and no heating or cooling after removal of the plastic bags at four dpi. In this test there was no further significant development of disease in PAI 6 compared to PAI 127, Carioca 80 and PC 235-4-E3, showing PAI 6 may be more resistant compared to the others. With the latter cultivars, the fungus infected the petiole and girdled the main stem if the inoculated leaves failed to senesce. In another test in which plants were maintained at a constant 20°C, no differences in levels of resistance were noted, but differences in virulence of the two isolates became obvious. A third isolate from Cedara was similar to that from Ixopo and grew faster than the KwaGubeshe isolate on OMA. Inoculations to maintain pathogenicity by re-isolating the fungus also indicated the slower growth of the KwaGubeshe isolate compared to the other two.

Table 1. Results of an Ascochyta blight inoculation trial.

Cultivar	Isolate	Lesion size[1]	Dead plants 14 dpi[2]	Dead plants 21 dpi
PAI 6	Ixopo	8	5	5
PAI 6	KwaGubeshe	6[3]	1	2
PAI 127	Ixopo	7	3	4
PAI 127	KwaGubeshe	6	0	1
Carioca 80	Ixopo	8	5	5
Carioca 80	KwaGubeshe	7	0	0

1 = Width of lesion in mm at 4 dpi, after removal of plastic bags.
2 = Number of plants dead out of six, the totals are cumulative.
3 = Lesion width very variable for the KwaGubeshe/PAI 6 treatment.

4.2 Race surveys

4.2.1 Halo blight

Samples of halo blight from Greytown, Potchefstroom, Ukulinga and Warden

gave similar results, typical of race 1 (Table 2.). The halo blight from Delmas was found to be of race 2. The cvs GN Nebraska No.1 Selection 27 and GN Tara were resistant to all samples.

Table 2. Results of testing of samples of halo blight in the differential cultivars.

	Differential Cultivar			
Source	Canadian Wonder	Red Mex[1]	Tendercrop	Edmund
Delmas	S[2]	S	S	R[3]
Greytown	S	R[4]	S	R
Potchefstroom	S	R	S	R
Ukulinga	S	R	S	R
Warden	S	R	S	R

1 = Red Mexican University of Idaho 3

2 = Susceptible, large halo development often with systemic chlorosis.

3 = All the resistance reactions of Edmund were small initial water-soaked lesions followed by necrosis with limited halo development (3 - 5 mm dia.).

4 = Resistant, hypersensitive reaction with very limited halo development (1 - 2 mm dia.).

4.2.2 Anthracnose

The differential cultivars gave similar reactions after inoculations with four samples of C.lindemuthianum from Cedara, Empangeni, Greytown and KwaGubeshe (Table 3). The isolates are of the same race and reactions most closely resemble that of the Alpha-Brazil race except the cv. Evolutie which should be resistant (Drijfhout and Davies, 1989).

Table 3. The reactions of the differential cultivars to infection with isolates of C.lindemuthianum from Cedara, Empangeni, Greytown and KwaGubeshe.

Susceptible Cultivars	Resistant Cultivars
Aiguille Vert	Cornell 49242
Evolutie	Michigan Dark Red Kidney
Michelite	Sanilac

Mexique 222	Perry Marrow
PI 167399	Coco a la Creme
PI 165426	Ab 136

A susceptible reaction was numerous dark necrotic lesions along the vein and inter-veinal spots. A resistant reaction was no apparent symptoms except Cornell 49242, when occasional necrotic lesions of 1 mm dia. were observed.

4.2.3 Rust

Results of inoculations of rust differential cultivars with single-pustule isolates are given in Table 4. Inoculations were not quantitative as the aim was not to identify races of rust per se but to obtain SPI's to test the partial resistance of the 23 cultivars in horizontal breeding programme.

The SPI's Wart-1, Wart-2 and Beth gave similar results and may be isolates of the same race. None of the SPI's was virulent in Nep 2 (usually susceptible in the field), which suggests that at least another race is present in South Africa. In addition, inoculation of Epicure with rust from Empangeni also gave a fully susceptible reaction. If it is assumed that the SPI U6A is a different race to M1B and not just a variant of it, then there are at least eight rust races in South Africa.

4.3 Breeding for horizontal resistance to rust

4.3.1 Breeding trial

Ratings of pustule size of the 23 susceptible cultivars within the 1988 trial in the disease garden showed no major differences in pustule size. Pustule sizes were rated 5 (0.5 - 0.8 mm dia.) or 6 (> 0.8 mm dia.). Ratings of the cultivars in the RC trial also showed little difference with only a single replication of 259 AND 621 being rated as 4 (0.3 - 0.5 mm dia.) and the rest as 5 or 6. Sizes indicated are the maximal size observed and not an average size. A possible skewing of the distribution of pustule size towards a smaller size was not observed. It appears that pustule size does not have an important role in the partial resistance observed.

Table 4. Results of testing single-pustule isolates of rust in differential cultivars.

	Isolates									
Cultivar	M1B	U6A	Ced-1	Potch-1	Potch-2	Potch-3	Wart-1	Wart-2	Beth	U93Ba40/32
Aurora	1	1	3	5/6	5/6	1	2	2	2	2
A x S 37[1]	1	1	2	1	1	1	2	2	2	2
Brown Beauty	5/6	5/6	5/6	5/6	5/6	5/6	5/6	5/6	5/6	5/6
CSW 643[2]	1	1	5/6	5/6	5/6	1	2	2	2	2
CNC[3]	3	1	1	1	1	1	1	2	2	1
Early Galatin	5/6	5/6	5/6	5/6	3	1	5/6	5/6	5/6	5/6
Ecuador 299	1	1	3	3	3	3	2	2	2	2
Golden Gate Wax	3	3	5/6	5/6	1	3	3	3	3	3
Kentucky W. 765[4]	3	3	3	5/6	5/6	5/6	2	2	2	2
Kentucky W. 780	3	3	5/6	5/6	5/6	5/6	4N++	4N++	4N++	4N++
Kentucky W. 814	2	2	2	5/6	5/6	5/6	3	3	3	3
Mexico 235	1	1	1	1	1	1	2	2	2	2
Mexico 309	3	3	1	1	1	1	1	3	1	3
Mountain WHR[5]	4N++	4N++	4N++	4N++	4N++	4N++	4N++	4N++	4N++	4N++
Nep 2	2	2	2	1	3	2	2	2	2	2
Olathe	3	3	5/6	5/6	5/6	5/6	2	2	3	3
Pinto 650	3	3	5/6	5/6	5/6	5/6	3	5/6	5/6	5/6
Redlands GL C[6]	5/6	5/6	5/6	3	3	3	*	*	*	*
Redlands Pioneer	3	3	5/6	5/6	3	5/6	3	2	3	3
US No. 3[7]	3	3	5/6	5/6	1	1	3	3	3	3

50151	1	1	1	1	1	1	2	2	2	2
Epicure[8]	3	3	4N++	4N++	4N++	4N++	*	*	*	*
Natal SS[9]	5/6	5/6	5/6	1	3	1	5/6	5/6	5/6	5/6
Teebus	1	2++	5/6	5/6	5/6	5/6	4N++	4N++	4N++	4N++

The key is from Stavely et al. (1983) where,

1 = Resistant, no apparent symptoms

2 = Necrotic lesions: 2 = < 0.3 mm dia. 2+ = 0.3 - 1.0 mm dia. 2++ = 1 - 3 mm dia. 2+++ = > 3mm dia.

3 = Pinpoint uredia (< 0.3 mm dia.)

4 = Uredia 0.3 - 0.5 mm dia.

5 = Uredia 0.5 - 0.8 mm dia.

6 = Uredia > 0.8 mm dia.

4N++ = Uredia of size 4 surronded by ring necrosis of size ++; under warmer conditions a 2++ reaction was obtained.

* = Not tested

1 = Actopan x Sanilac 37 2 = Californian Small White 3 = Compuesto Negro Chimaltango

4 = Kentucky Wonder 5 = Mountain White Half Runner 6 = Redlands Greenleaf C

7 = United States Number 3 7 = The following three cultivars were tested for local interest

8 = Natal Speckled Sugar

Successful ratings of % leaf area affected by rust were made of each trial except the first trial in the disease garden. The inoculator rows (Teebus) of the disease garden trial proved to be resistant to the SPI M1B and the test rows were inoculated at too late a stage. Percentage leaf area affected by rust should include leaf senescence and drop and ultimately plant death. In these trials, however, these factors were not included due to the presence of common blight and drought stress which can also cause early senescence. In the TC trial, drought and rust disease stress acted synergistically. Some lines in the SC and TC field trials were rejected as too susceptible to common blight. The greenhouse increase of the SC F_1 was contaminated with common blight. Most of the lines were susceptible and a few of the more susceptible lines were rogued. It is most probable that a cultivar selected from this programme will be common blight susceptible.

Ratings of % leaf area affected by rust (Tables 5 to 7) were somewhat subjective, especially at the lower ranges. Ratings are, however, proportional to each other on a specific rating date. The first trial ratings were too high, with experience gained, more accurate ratings were made of the latter two trials. It was difficult to compare determinate with indeterminate plants. Determinate plants have a vegetative stage, stop growing and flower. Indeterminate plants continue to grow after the induction of flowering and the rating was a visual mean of young and old leaves. Some determinate cultivars such as the ICA cvs occasionally produced a fresh flush of growth after the vegetative stage. If a cultivar had a greater tendency to retain its leaves, even if heavily infected, compared to another then it would obviously receive a higher rating. Fortunately, there were no marked differences in this trait. Automatic measurement of % leaf area affected by rust by image analysis, for example, would remove the subjectivity but may make the rating an inordinately long process. Venette and Venette (1991) have used image analysis to count uredia but obtained erroneous readings with small uredia. Attempts were made in the first trial to see if date of flowering had any effect on the rust rating. The cvs KID 16 and KID 20, amongst the most susceptible, were the first to flower but PC 222-5-6-P2, also very susceptible, was the last to flower of the determinate cultivars. A similar lack of correlation was also noted in the indeterminate cultivars.

Table 5. Rust ratings of the 24 parent cultivars and Teebus in the 1989/90 field trial at Ukulinga.

	Percentage leaf area affected by rust		
Date	8.12.	15.12.	21.12.
Cultivar			
PC 222-5-6-P2	20[2*]	43[2]	25[2]
PC 223-4-1-D1	28[2]	47	28[2]
PC 235-4-E3	28[2]	48	32
1266	10[2]	30	7
1271	28[2]	43	32
1273	25	43	33
1312	17	30[2]	7
ICA 15116	20	39	30
ICA 15521	23	43	32
ICA 15522	17	37	26
Epicure	0	0	0
PVA 992	28	50	42
KID 16	32	54	46
KID 20	30	49	40
COS 10	31	50	42
AFR 241	29	49	45
AND 340	20	37	31
GLP X 1132	25	40	27
339 AFR 290	27	43	35
358 AFR 290	28	46	35
259 AND 621	13[2]	28[2]	18[2]
269 AND 631	27	44	32
312 AND 689	13	27	25
380 HAL 10	22	41	35
Teebus	0	0	<1
LSD[1] (5 %)	7	10	8
LSD (0.1 %)	13	17	15

Please see page 65 for key.

Table 6. Rust ratings and yield of the 23 parent cultivars and Teebus in the 1990/91 field trial at Ukulinga.

	Percentage leaf area affected by rust					Yield[‡]
Date	11.12.	18.12.	27.12.	3.1.	10.1.	
Cultivar						
PC 222-5-6-P2	11	15	20	21	20	18
PC 223-4-1-D1	7	11	14	15	15	36
PC 235-4-E3	7	10	14	16	14	80
1266	4	3	3	4	4	191
1271	6	10	12	13	16	48
1273	6	10	12	12	16	51
1312	4	3	3	2	2	106
ICA 15116	5	6	7	9	10	*
ICA 15521	5	6	8	10	10	*
ICA 15522	4	4	6	9	10	55
PVA 992	9	11	14	16	16	53
KID 16	10	14	19	27	26	31
KID 20	7	13	17	22	24	29
COS 10	10	14	18	20	19	16
AFR 241	9	14	19	22	24	21
AND 340	6	6	7	10	11	59
GLP X 1132	5	7	7	8	8	136
339 AFR 290	9	10	15	15	17	70
358 AFR 290	10	10	14	15	15	92
259 AND 621[²]	2	2	1	2	2	207
269 AND 631	7	7	7	9	10	32
312 AND 689	3	3	4	5	6	56
380 HAL 10	7	8	12	15	17	72
Teebus	<1	<1	1	3	5	–
LSD[†] (5 %)	2	3	4	3	4	38
LSD (0.1 %)	4	5	6	5	7	66

Please see page 65 for key

Table 7. Rust ratings and yield of the 23 parent cultivars, Teebus and Umlazi in the 1992 field trial at Ukulinga.

	Percentage leaf area affected by rust						Yield[3]
Date	6.3.	17.3.	24.3.	31.3.	7.4.	16.4.	
Cultivar							
PC 222-5-6-P2	7	13	14	17	15	15	18
PC 223-4-1-D1	7	9	10	10	8	8	52
PC 235-4-E3	7	10	11	11	8	9	55
1266	2	2	1	2	4	S[4]	178
1271	7	10	10	13	14	13	45
1273	8	10	11	14	14	12	47
1312	6	6	6	7	6	5	103
ICA 15116[2]	5	5	6	6	6	5	63
ICA 15521	4	5	6	8	7	8	93
ICA 15522	4	5	6	7	8	S	124
PVA 992	7	9	11	14	13	12	22
KID 16	7	9	14	18	17	S	34
KID 20[2]	8	10	11	14	16	15	53
COS 10	7	9	9	12	12	14	29
AFR 241	6	9	11	12	11	S	19
AND 340	4	5	6	7	7	S	100
GLP X 1132	6	7	8	8	8	6	117
339 AFR 290	5	7	8	9	10	10	44
358 AFR 290	5	7	10	14	15	S	83
259 AND 621	1	1	1	1	3	S	141
269 AND 631	4	6	7	7	8	6	94
312 AND 689	3	4	2	3	4	4	164
380 HAL 10	3	5	7	8	7	S	76
Umlazi	0	0	0	3	4	S	195
Teebus	0	<1	1	2	3	S	-[5]
LSD[1] (5 %)	1	3	2	3	3	3	48
LSD (0.1 %)	2	5	4	5	5	5	85

Please see page 65 for key.

Key for Table 5.

* = Values given are the means of three replications except where the number of replications is given by a suffix.
† = Least significant differences (LSD) are given for 5 and 0.1 % levels of probability for all cultivars excluding Epicure and Teebus.
The 8th of December is 50 days after planting.

Key for Table 6.

‡ = Yield is given in g per single 3 m row.
† = LSD's are given for all cultivars except Teebus.
* = Pods were blighted and yield was negligible. The third replication of 312 AND 689 was similarly affected.
§ = The yield of Teebus was not measured.
2 = Only two replications of 259 AND 621 were planted due to shortage of seed. Other values are the means of three replications. Yield is given for the same number of replications.
The 11th of December and 10th of January are 49 and 79 days after planting, respectively.

Key for Table 7.

‡ = Yield is given in g per 3 m row.
† = LSD's are given for all cultivars except Teebus and Umlazi except yield, which includes Umlazi.
2 = One replication of KID 20 was discarded due to poor emergence and a third replication of ICA 15116 was not planted due to a shortage of seed. Other values are means of three replications. Yield is given for the same number of replications.
§ = The yield of Teebus was not measured.
¶ = Senescent.
The 6th of March and 16th of April are 39 and 80 days after planting, respectively.

Various plant architectural forms were noted among the crosses. Before commencement of this project, strong selection pressure for determinate types was mooted. Determinate types, however, tend to yield less than indeterminate types, but mature quicker. A determinate plant flowers and sets pods over a short period and if favourable conditions are then experienced, it can often outyield a similar indeterminate plant. But when stressed, pods will drop and determinate plants respond less readily to favourable conditions at a later stage than would an indeterminate plant. Mechanized agriculture is aided by synchronized maturity and hence flowering. A Type IIa bush (Van Schoonhoven and Pastor-Corrales, 1987) would be ideal as it is still indeterminate. Most of the selections were Type IIIa sprawling bushes and a few were determinate Type I bushes.

Plants next to the border rows were initially more infected, but thereafter infection was more even. The partial resistance of the cultivars tested in these trials does not appear to rely to any great extent on the reduction of inoculum potential. The resistance thus acts by either slowing down and/or preventing infection and/or growth to sporulation.

The first field trial initially suffered from excessive rainfall and flooding in parts, with 100 mm of rain recorded during one day (climatic data is given in Appendix 3). Rating was made awkward by ankle-deep mud in places. This was followed by drought in the latter part of the season. Root rots were prevalent throughout the trial. Crosses with the PC lines appeared to be more susceptible and cognisance was taken of this during selection. The second trial was far more successful and the season was more "normal". The third trial was grown in 1992, the year of "the greatest drought in living memory in southern Africa". Fortunately, although the trial was affected, good results were obtained. The highest daily temperature measured in the three trials was 39°C in the first trial, but this was an isolated maximum. In the third trial, 38°C was recorded in a period of 13 days of temperatures in the thirties.

With a few exceptions, the intra-row rust resistances of the RC crosses were fairly even but there were some quite large inter-row differences. None of the crosses in the three trials had such a high degree of resistance as the more resistant parents. It is possible that as the rows were rated as a

unit, individual plants may have had better resistance. Field selection was done by comparing single plant yields; those from rows with better rust resistance and/or with a seed type closest to a speckled sugar were favoured. Several lines were rejected as too late-maturing. Forty-five single-plant selections were made from the Third Cross field trial. Their pedigrees are given in Appendix 4 and the seed was passed on to Mr A. Jarvie of Pannar Seed, Greytown, for evaluation before possible cultivar release (see section 4.4.1).

When the data of the 23 parent cultivars were analysed over all seasons (Tables 8 and 9) there were very highly significant (0.1 % level) differences between cultivars for rust ratings and yield. Blocks mean sum of squares (MS) were also very highly significant for rust ratings and yield, but there were no significant interactions between the blocks and cultivars. Seasons MS for the rust ratings was very highly significant and was mostly due to exaggerated ratings of the first trial. There was also a very highly significant interaction for rust ratings between seasons and cultivars indicating a genotype x environment interaction. The yield seasons and seasons interaction cultivars were not significant.

Table 8. Analysis of variance of the rust ratings of 23 parent cultivars of the 1989/90, 1990/91 and 1992 field trials at Ukulinga.

Source	DF	SS	MS	VR
Blocks	2	1148.20	574.1	25.62 ***
Blocks*cultivar	44	802.8	18.25	0.78 NS
Seasons	2	43949.66	21974.83	938.19 ***
Cultivar	22	16233.72	737.9	31.5 ***
Seasons*cultivar	44	2339.08	53.16	2.37 ***
Residual	88[1] (4)	2061.19	23.42	
Total	202 (4)	66534.66		

Means of the three ratings of the first trial, the five ratings of the second trial and the first five ratings of the third trial were analysed with an angular transformation and weighted according to the number of ratings in the trial. Coefficient of variation (CV) = 21.2 %.

1 = Allowances made for four missing plots by iteration.

Table 9. Analysis of variance of the yields of the 23 parent cultivars of the 1990/91 and 1992 third field trials at Ukulinga.

Source	DF	SS	MS	VR
Blocks	2	40490	20245	9.87 ***
Blocks*cultivar	44	36091	820	0.40 NS
Seasons	1	771	771	0.38 NS
Cultivar	22	259139	11779	5.74 ***
Seasons*cultivar	22	40972	1862	0.91 NS
Residual	35[1] (11)	71768	2051	
Total	137 (11)	449231		

1 = Allowances made for 11 missing plots by iteration. CV = 61.4 %.

The most susceptible and resistant cultivars (Table 10) were KID 16 and 259 AND 621, respectively. In the first trial at 57 days after planting 259 AND 621 had 52 % of the rust severity of KID 16, in the second trial at 72 days after planting 7 %, and in third trial at 64 days after planting 6 %. If yield is compared in the latter two trials, the difference between the two was 531 %. If one was to consider the yield difference between PC 222-5-6-P2 and 1266 (the yield extremes over the two trials) then the difference was 1028 %. Hence if these differences are attributable to horizontal resistance (bearing in mind a CV of 61.4 %) then there is considerable potential in breeding for resistance.

To compare the yield results (Tables 6, 7 and 10) would be of a dubious nature as no allowances were made for border effects and only a single row of each cultivar was grown and harvested. Ideally, percentage reduction in yield of the cultivars due to rust infection compared to the cultivars grown rust-free under the same conditions should be compared. Hence no allowance can be made for the better adaptation of cultivars to agronomic conditions other than rust infection. The yield results of the 1990/91 trial were very highly significantly negatively correlated with the rust ratings, with correlation coefficients of -0.56 to -0.68. The latter two ratings of the 1992 trial (7th & 16th of April) had significant (5 % level) negative correlation with the yield of -0.30 and -0.27, respectively. Some cultivars were maturing by this stage. Of the other ratings, only the 24th of March

Table 10. Significant differences in rust ratings and yield of the 23 parent cultivars after analysis of the 1989/90, 1990/91 and 1992 Ukulinga field trial data by Duncan's multiple range test (DMRT) at the 5 % level.

	Rust Ratings[1]			Yield[2]	
Cultivar	Mean	DMRT	MUD	Mean	DMRT
PC 222-5-6-P2	25	abc[3]	19	19	abcdefghij
PC 223-4-1-D1	23	abcd	16	44	efghij
PC 235-4-E3	25	abc	19	68	ghij
1266	14	d	7	183	j
1271	25	abc	19	46	efghij
1273	25	abc	19	59	fghij
1312	16	bc	9	101	hij
ICA 15116	22	abcd	16	72	hij
ICA 15521	22	abcd	16	90	hij
ICA 15522	20	abcd	14	90	hij
PVA 992	27	a	21	38	defghij
KID 16	29	a	25	33	cdefghij
KID 20	29	a	24	34	cdefghij
COS 10	27	a	22	22	bcdefghij
AFR 241	27	a	22	20	abcdefghij
AND 340	21	abcd	14	83	hij
GLP X 1132	21	abcd	15	127	ij
339 AFR 290	24	abc	18	59	fghij
358 AFR 290	26	ab	20	86	hij
259 AND 621	14	d	7	170	j
269 AND 631	22	abcd	16	63	fghij
312 AND 689	16	d	8	122	ij
380 HAL 10	23	abcd	17	71	ghij

1 = Means of the three ratings of the 1989/90 trial, the five ratings of the 1990/91 trial and the first five ratings of the 1992 trial were analysed with an angular transformation and weighting according to number of ratings in the trial. Grand mean of the transformed data (Mean), upon which the DMRT was done, and of the untransformed data (MUD) are given.

2 = Means of the 1990/91 and 1992 trials were analysed, the figures given are in g per 3 m row. The mean given is the grand mean.

3 = Cultivars followed by the same letter are not significantly different. Allowances were made for missing plots by iteration.

rating was very highly significantly negatively correlated with yield with a correlation coefficient of -0.54. The drop in correlation coefficients was most probably due to increased drought stress in the 1992 trial. A correlation coefficient of -0.678 was obtained after comparing the yields and ratings of the latter two trials.

Rust ratings of the cultivars within individual field trials were always very highly significantly correlated with each other when compared over different dates. This shows that many ratings of a trial are not required. Correlations ranged from 0.81 to 0.93 in the 1989/90 trial, from 0.54 to 0.95 in the 1990/91 trial and from 0.61 to 0.91 in the 1992 trial (excluding the last rating, as some cultivars were senescent by that stage).

Cultivars found to be highly resistant were 1266, 259 AND 621 and 312 AND 689. The cv. 1312 was highly resistant in the first two trials, but only moderately resistant in the third. In the third trial, 1312 had a relatively high initial rating, but this remained more or less constant during the trial. Cultivars identified as moderately resistant were ICA 15116, ICA 15521, ICA 15522, AND 340 and GLP X 1132 and those as moderately susceptible were PC 223-4-1-D1, 269 AND 621 and 380 HAL 10. The highly susceptible cultivars were PC 222-5-6-P2, PC 235-4-E3, 1271, 1273, PVA 992, KID 16, KID 20, COS 10, AFR 241, 339 AFR 290 and 358 AFR 290.

Cultivars found to be high-yielding were 1266 and 259 AND 621. Those that were moderately high yielding were GLP X 1132 and 312 AND 689. Those with a moderate yield were 1312, ICA 15521, ICA 15522, AND 340, 358 AFR 290. Those with a moderately poor yield were PC 235-4-E3, ICA 15116, 269 AND 631, 380 HAL 10. Those with a poor yield were PC 222-5-6-P2, 1271, 1273, PVA 992, KID 16, KID 20, COS 10, AFR 241, 339 AFR 290. The cultivars with a red speckle on a cream background (speckled sugar type seed) were PC 222-5-6-P2, PC 223-4-1-D1, PC 235-4-E3, 1271, 1273, 1312, COS 10 and AFR 241.

Umlazi was bred for vertical rust resistance in Natal and is adapted to conditions at Ukulinga. It would be interesting to compare the single-plant selections (which have also been selected at Ukulinga), Umlazi and other sugar bean cultivars in yield trials . All the parent cultivars are in the list of pedigrees (Appendix 4) of the final selections (TC lines) except KID

16 and 380 HAL 10. Crosses with KID 16 were deliberately discontinued and, presumably, none of the 380 HAL 10 crosses was found to have merit.

4.3.2 Diallel trial

The test trial results are given in Appendix 5 and a summary is given in Tables 11 and 13. It was intended that three replications be planted each year but due to failure of glasshouse air conditioning in both years, plants bearing crosses were lost and only two replications were planted.

Table 11. Ratings of % leaf area affected by rust of the 1990/91 and 1992 diallel trials at Ukulinga.

♀ Parent \ ♂ Parent	1	2	3	4	5	6	7	8
1 PC 222-5-6-P2	**15**	5	12	9	15	7	4	8
2 1266	4	**2**	4	5	5	3	3	4
3 1273	12	5	**11**	8	13	7	4	7
4 ICA 15522	11	5	8	7	8	5	2	6
5 KID 16	13	7	12	8	**18**	8	5	9
6 GLP X 1132	7	4	6	4	7	**6**	2	6
7 259 AND 621	5	3	5	4	5	2	**2**	3
8 269 AND 631	8	4	7	5	8	6	3	7
GCA	2.8	-2.5	1.7	-0.2	3.2	-1.3	-3.3	-0.5

Each figure given is a grand mean of the means of four ratings made for the second diallel trial and means of the first three ratings made for the third diallel trial. The diagonal (top left to bottom right) is the results of the parent cultivars. General combining ability (GCA) is also given.

Table 12. Analysis of variance of rust ratings of the 1990/91 and 1992 diallel trials at Ukulinga.

Source	DF	SS	MS	VR
Blocks	1	74.715	74.715	54.02 ***
Blocks x crosses	63	108.377	1.720	1.24 NS

Seasons	1		374.761	374.761	270.98 ***
Crosses	63		3077.500	48.849	35.32 ***
GCA	7		2598.131	371.162	268.37 ***
SCA[1]	28		433.071	15.467	11.18 ***
Reciprocal	28		46.300	1.654	1.20 NS
Crosses x seasons	63		218.544	3.464	2.50 ***
GCA	7		105.058	15.008	10.85 ***
SCA	28		74.351	2.654	1.92 *
Reciprocal	28		39.135	1.398	1.01 NS
Residual	56[2]	(8)	88.525	1.383	
Total	247	(8)	3943.421		

1. SCA = specific combining ability.

2 = Allowances made for eight missing plots by iteration. CV = 18.1 %

The analyses of variance of the rust rating and yield data (Tables 12 and 14) are a combination of two Genstat 5 Release 2.2 programmes (given in Appendix 6). Treatment effects were analysed using linear models, firstly as a design with 64 crosses (56 crosses and 8 selfs) and secondly as an 8 x 8 diallel. The Griffing (1956) method was done by modifying a linear equation.

Table 13. Yields of the 1990/90 and 1991 diallel trials at Ukulinga.

♂ Parent	1	2	3	4	5	6	7	8
♀ Parent								
1 PC 222-5-6-P2	2	14	5	10	3	11	19	10
2 1266	19	**11**	13	13	11	20	17	14
3 1273	5	12	**3**	7	5	11	15	6
4 ICA 15522	11	11	6	**5**	9	16	11	12
5 KID 16	4	10	4	9	**2**	10	12	8
6 GLP X 1132	12	**13**	10	11	10	**9**	19	10
7 259 AND 621	17	15	12	13	14	21	**12**	16
8 269 AND 631	10	16	7	9	8	13	19	**5**
GCA	-1.3	2.8	-3.3	-0.9	-3.2	2.0	4.4	-0.4

Each figure given is the mean of the two trials. The diagonal is the results of the parent cultivars. General combining ability is also given.

Table 14. Analysis of variance of the yields of the 1990/91 and 1992 trials at Ukulinga.

Source	DF	SS	MS	VR
Blocks	1	48.095	48.095	6.35 ***
Blocks x crosses	63	312.212	4.956	0.65 NS
Seasons	1	1647.243	1647.243	217.33 ***
Crosses	63	5381.278	85.417	11.27 ***
GCA	7	3511.466	501.638	66.18 ***
SCA	28	1551.873	55.424	7.31 ***
Reciprocal	28	317.94	11.355	1.50 NS
Seasons x crosses	63	1383.381	21.958	2.90 ***
GCA	7	625.272	89.325	11.79 ***
SCA	28	286.712	10.24	1.35 NS
Reciprocal	28	471.397	16.836	2.22 ***
Residual	561 (8)	424.444	7.579	
Total	247 (8)	9196.653		

1 = Allowances made for eight missing plots by iteration. CV = 25.7 %.

Blocks MS for rust ratings and yield were very highly significant, but the interactions with the crosses were not significant. Differences due to the seasons were also very highly significant for rust ratings and yield. The weather experienced during the third trial was more extreme with hotter conditions. Interactions between seasons and crosses were very highly significant for rust ratings and yield indicating a genotype x environment interaction.

The second programme allowed further partitioning of the crosses and crosses x seasons sums of squares (SS) by analysing the data as an 8 x 8 diallel. For rust ratings and yield over both seasons, inheritance due to general and specific combining abilities (GCA and SCA, respectively) was very highly significant. The degree of inheritance due to GCA, additive gene effects, was far greater than that due to SCA, dominance and epistatic effects, as

evinced by the partitioning of the crosses SS. Percentages of GCA, SCA and reciprocal were 84, 14 and 2, respectively, for rust ratings and 65, 29 and 6, respectively, for yield. Reciprocal inheritance was not significant for yield or rust ratings. The data were also analysed by Griffing's (1956) method and virtually identical results were obtained.

Due to the limit on the number of the parameters of the Genstat regression analysis of variance, it was not possible to calculate the reciprocal x seasons SS directly. Hence they were obtained by subtraction of the GCA and SCA interactions with seasons from the crosses x seasons SS. The GCA x seasons MS were very highly significant for both rust ratings and yield. The SCA x seasons MS were significant for rust ratings but not yield. Reciprocal x seasons MS were not significant for rust ratings, but were very highly significant for yield.

The importance of the GCA, SCA and reciprocal interactions with seasons as determined by the partitioning of the sums of squares was different to that of over both seasons (Tables 11 and 13). Percentages of GCA, SCA and reciprocal were 48, 34 and 18, respectively, for rust ratings and 45, 21 and 34, respectively, for yield. The GCA interaction was still the most important factor in the inheritance of improvement in rust resistance and yield but to a lesser extent compared to over both seasons. The SCA interaction had far greater importance compared to SCA over both seasons for rust ratings although the overall significance declined. The SCA interaction for yield declined in importance and significance. Importance of the reciprocal interactions increased for both rust ratings and yield, but still remained non-significant for rust ratings.

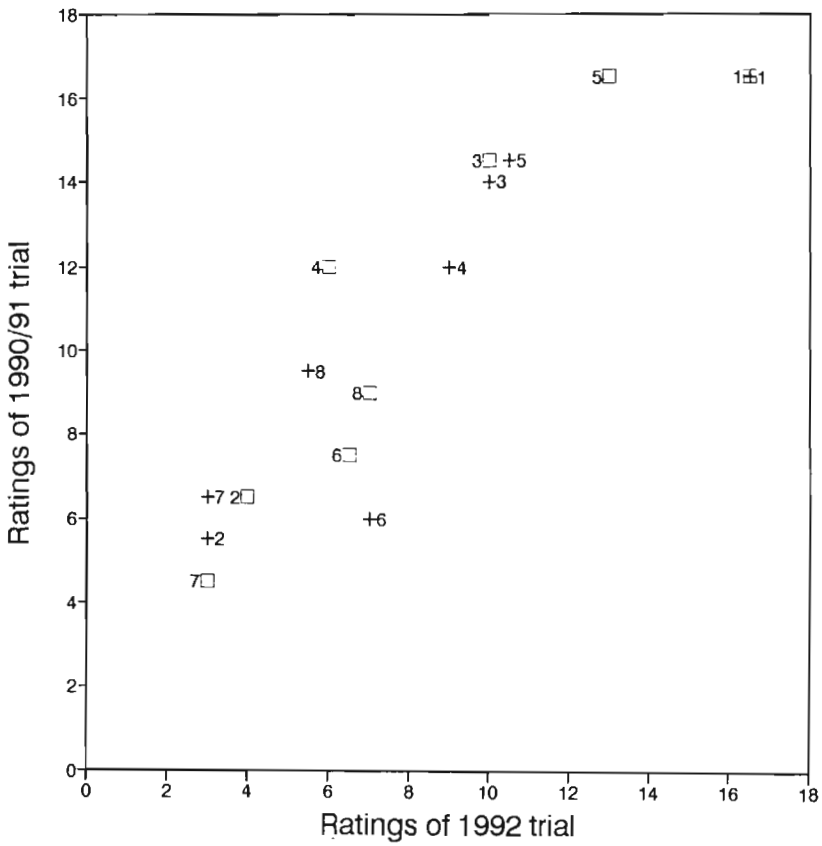
Sums of squares for GCA and SCA can also be obtained by hand. Reciprocal differences of the diallel were eliminated by averaging reciprocal crosses. The GCA SS were obtained by calculating main effects ($\Sigma_i\Sigma_j\Sigma_k(x_{i00}-\bar{x})$) and the SCA SS from calculating interaction effects ($\Sigma_i\Sigma_j\Sigma_k(x_{ij0}-x_{i00}-x_{0j0}+\bar{x})$). Interactions with seasons formulae were $\Sigma_i\Sigma_j\Sigma_k(x_{i0k}-x_{i00}-x_{00k}+\bar{x})$ for GCA and $\Sigma_i\Sigma_j\Sigma_k(x_{ijk}-x_{ij0}-x_{0jk}-x_{i0k}+x_{i00}+x_{0j0}+x_{00k}-\bar{x})$ for SCA, where there are i male parents, j female parents and k seasons and a 0 signifies values averaged for that factor. Alternatively, the SAS macro of Linda (1993) could be used to analyse diallel crosses according to Griffing's methodology.

Figures 5a to 5h illustrate genotype x environment interactions for the diallel cultivars. Divergence from a central 45° diagonal of the graph shows that the crosses of a cultivar do better in one season compared to the other. Deviation from a 45° angle of the plotted values shows a genotype x environment interaction. The closer the plotted values are to a fitted line points to a greater consistency of performance of the parent in both environments. Greater the scatter of the points, the greater the adaptation of the parent to one of the environments.

The crosses generally did better in the 1990/91 trial compared to the 1992 trial. Genotype x environment interactions of the rust ratings of the crosses appeared to correspond with resistance. There were negligible genotype x genotype interactions for the cvs PC 222-5-6-P2 and KID 16 (the most susceptible), high levels for 1266 and 259 AND 621 (the most resistant) and intermediate levels for cultivars with intermediate resistance.

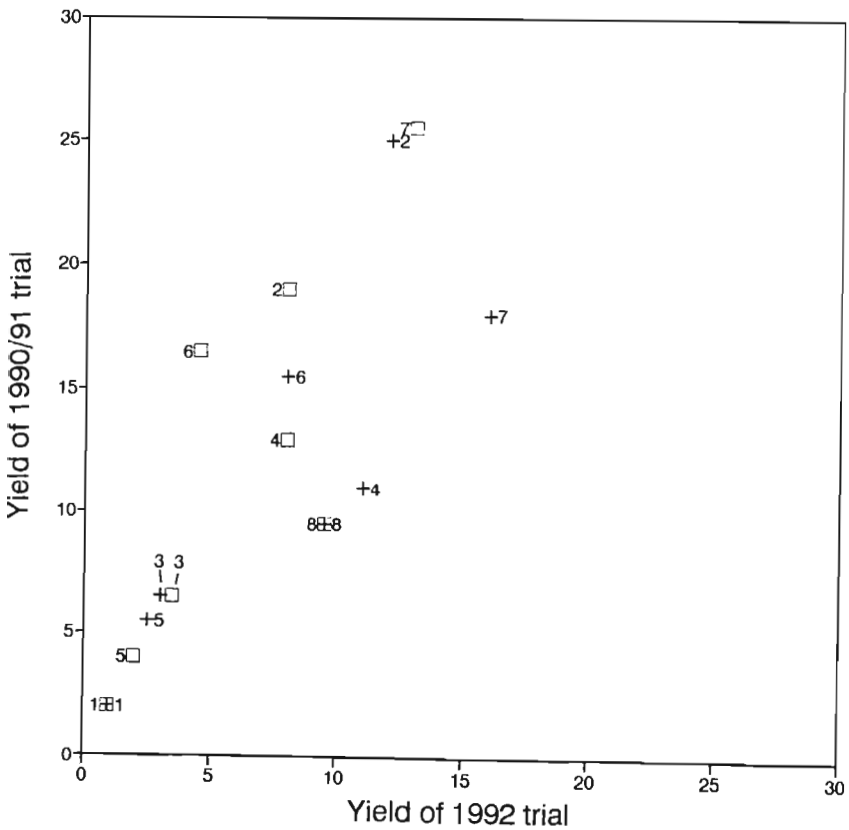
The reverse trend was observed, although more obscurely, for the yield results, with a greater genotype x environment interaction for the susceptible cvs PC 222-5-6-P2 and KID 16. As noted earlier, there was a synergism between drought and rust disease stress, especially in the 1992 trial. Hence the yield of a resistant cultivar is more stable over a range of environments compared to a susceptible cultivar.

Levels of heritability were estimated by regression of progeny values on mid-parent values. For the 1990/91 trial, heritabilities of rust resistance were 0.97 ± 0.079 and 0.827 ± 0.079, respectively, for the reciprocal male and female crosses, with a mean of 0.899 ± 0.056. For the 1992 trial, the heritabilities were 0.623 ± 0.096 and 0.583 ± 0.091, with a mean of 0.603 ± 0.065. Heritabilities of improved yield for the 1990/91 trial were 1.322 ± 0.205 and 1.241 ± 0.187, respectively, for the reciprocal male and female crosses, with a mean of 1.282 ± 0.137. For the 1992 trial, heritabilities were 1.639 ± 0.448 and 1.678 ± 0.396, respectively, with a mean of 1.659 ± 0.294. Lower estimates of the 1992 trial show the greater influence of environment on both characters.



i. Rust ratings (% leaf area affected by rust)

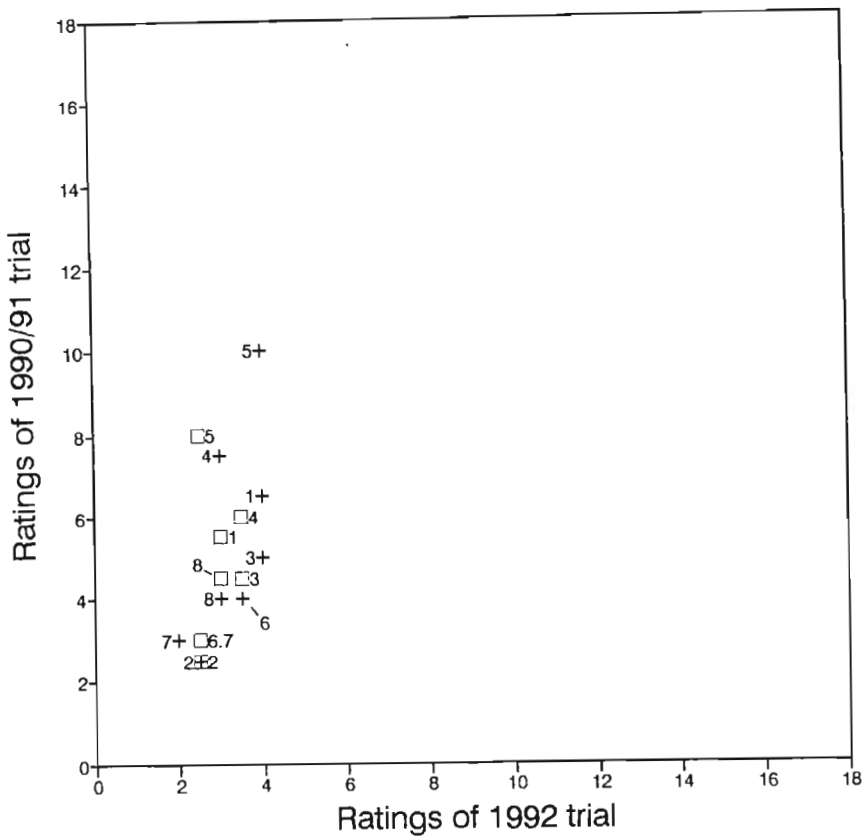
1 = PC 222-5-6-P2
2 = 1266
3 = 1273
4 = ICA 15522
5 = KID 16
6 = GLP X 1132
7 = 259 AND 621
8 = 269 AND 631



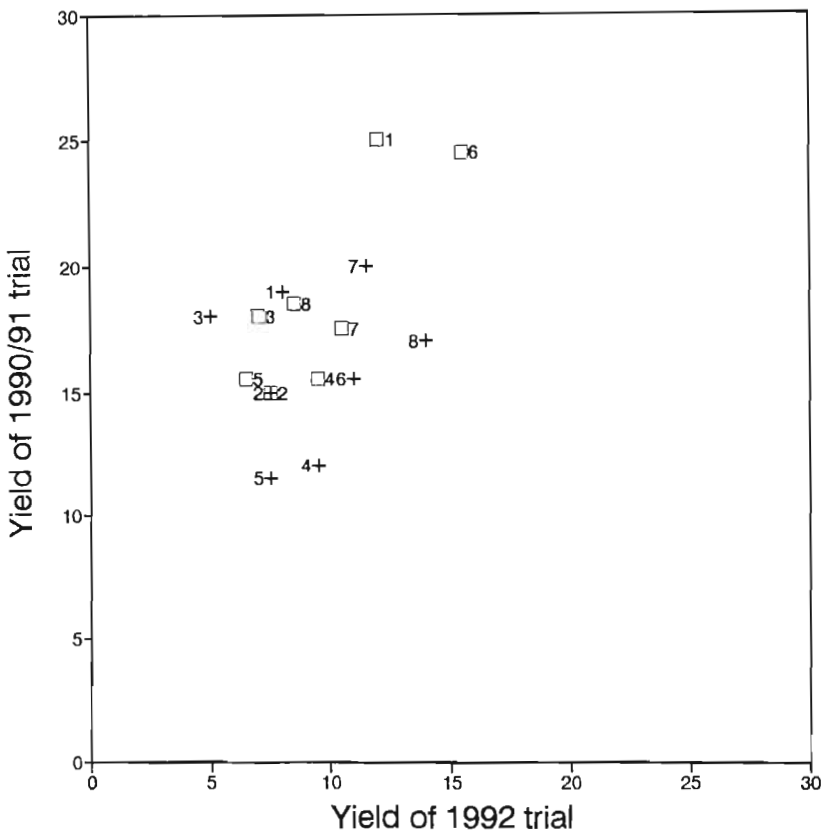
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5a. Genotype x environment interaction for PC 222-5-6-P2 (parent 1).



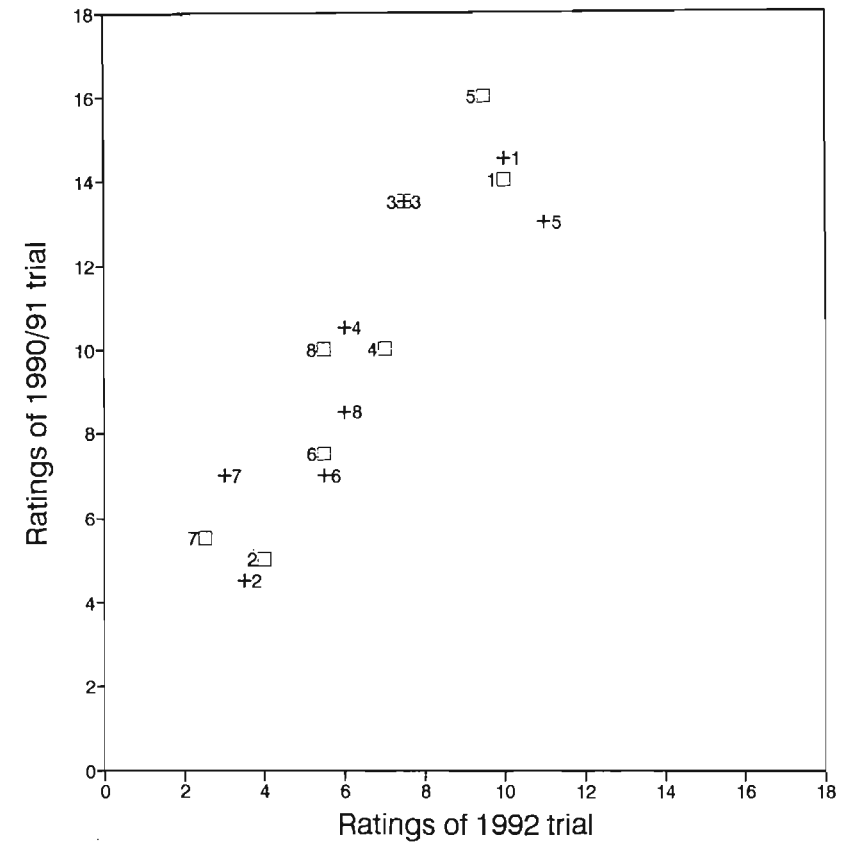
i. Rust ratings (% leaf area affected by rust)



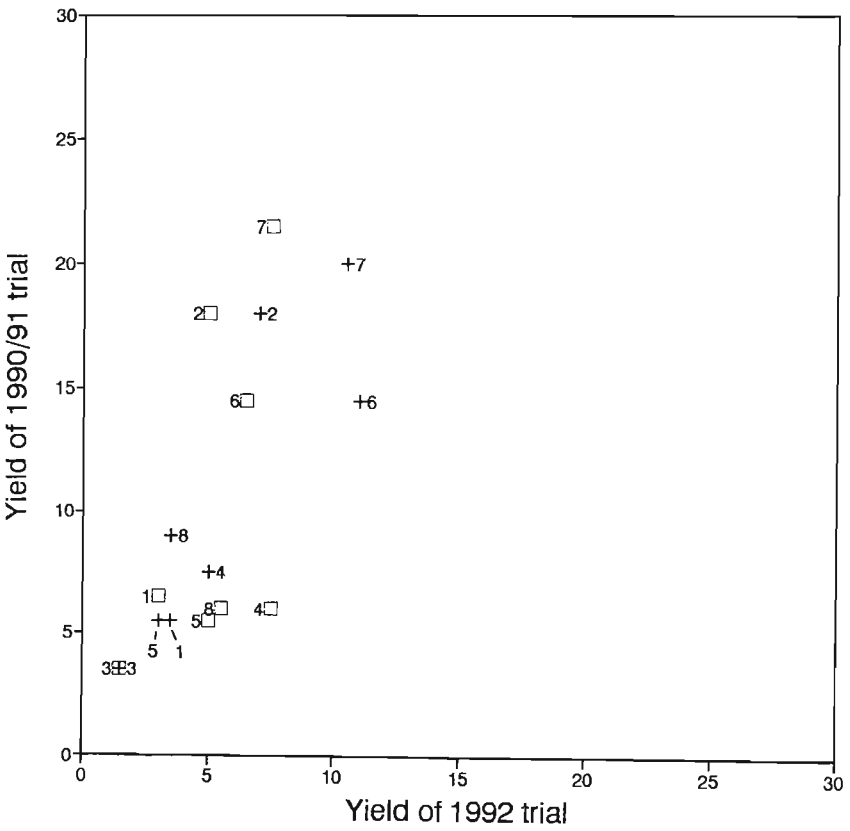
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5b. Genotype x environment interaction for 1266 (parent 2).



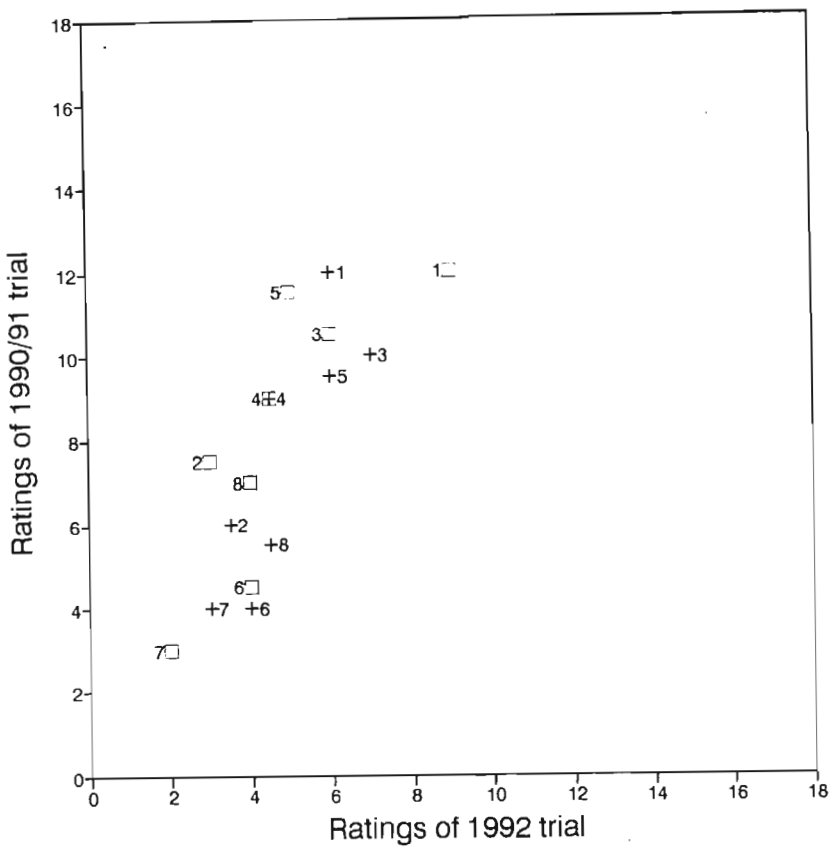
i. Rust ratings (% leaf area affected by rust)



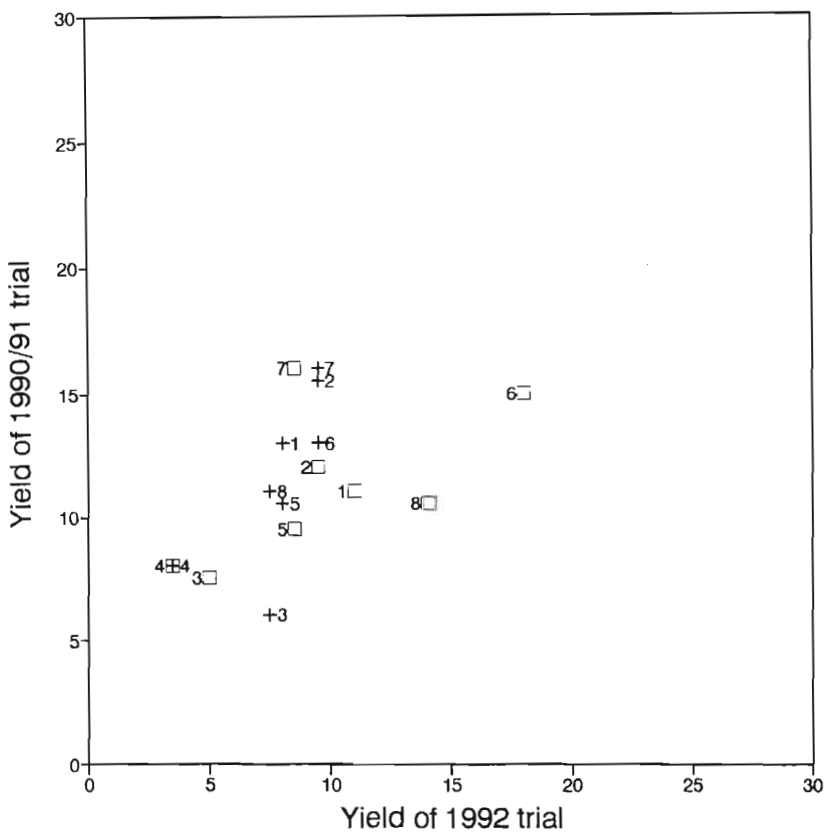
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5c. Genotype x environment interaction for 1273 (parent 3).



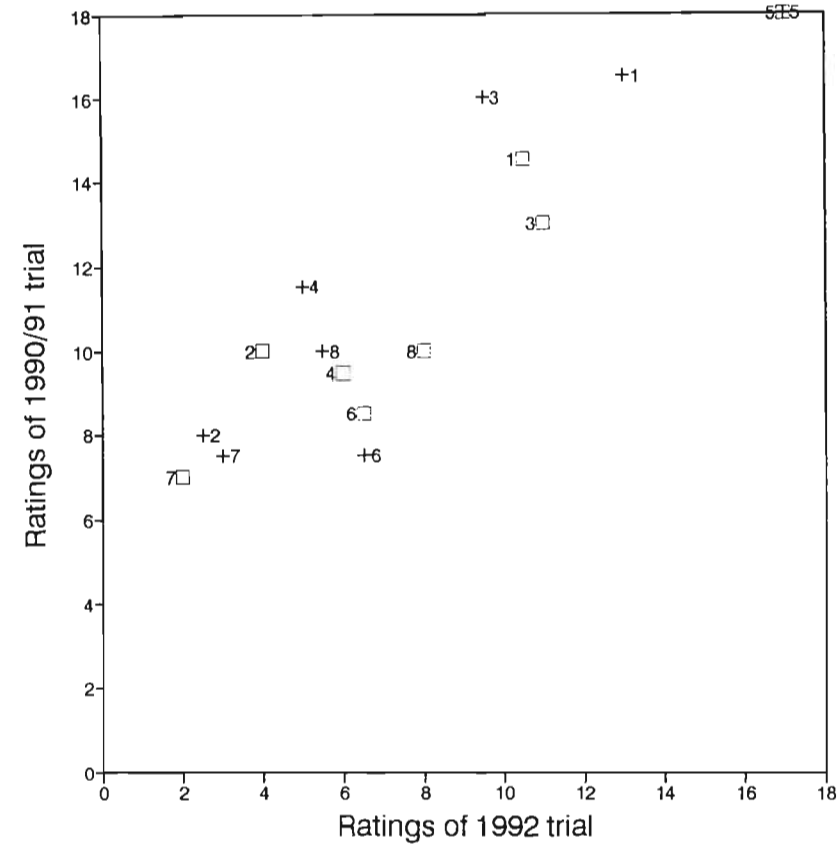
i. Rust ratings (% leaf area affected by rust)



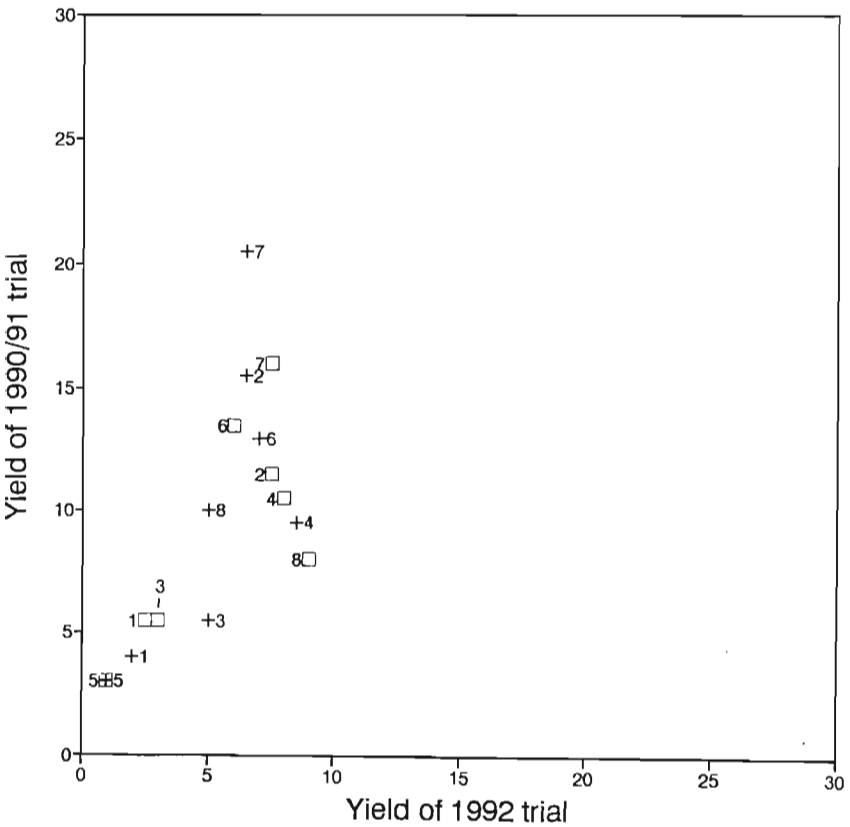
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5d. Genotype x environment interaction for ICA 15522 (parent 4).



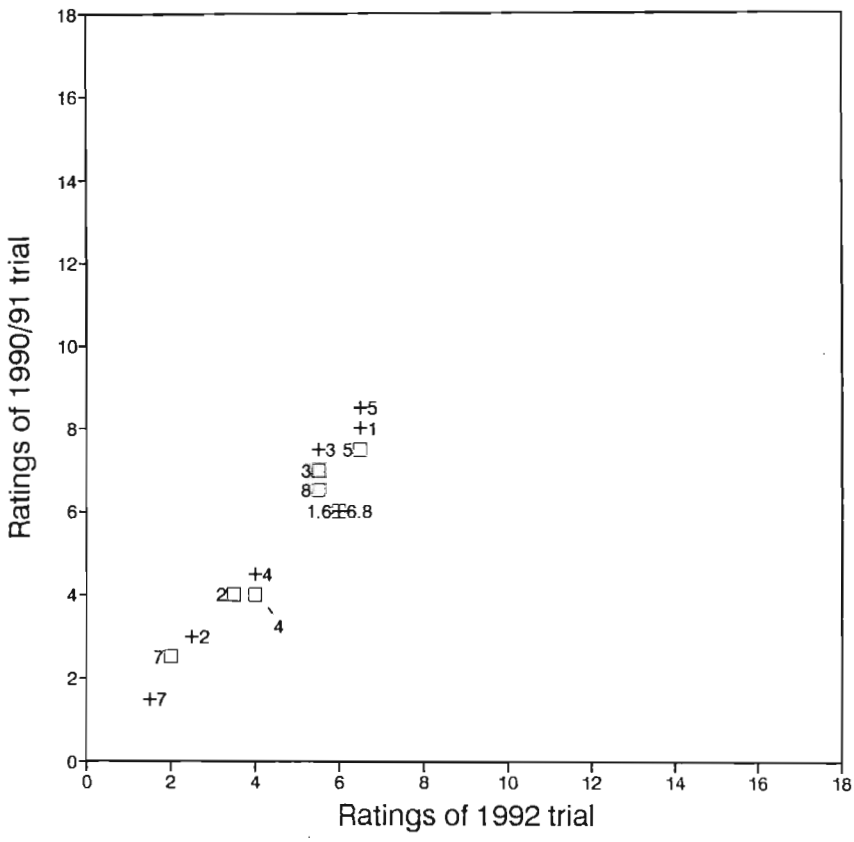
i. Rust ratings (% leaf area affected by rust)



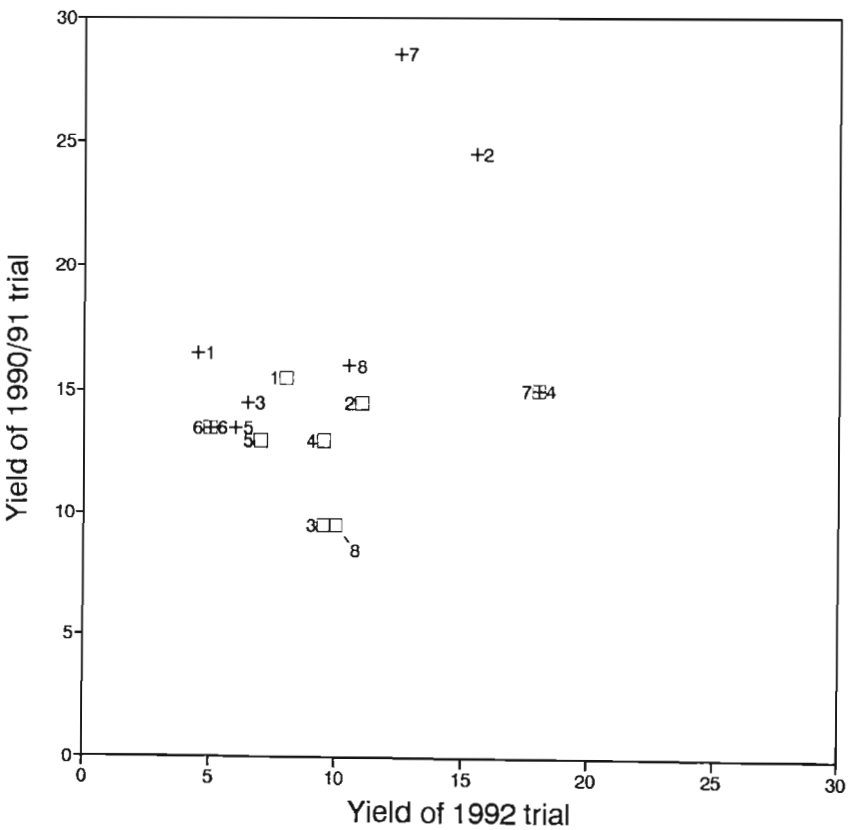
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5e. Genotype x environment interaction for KID 16 (parent 5).



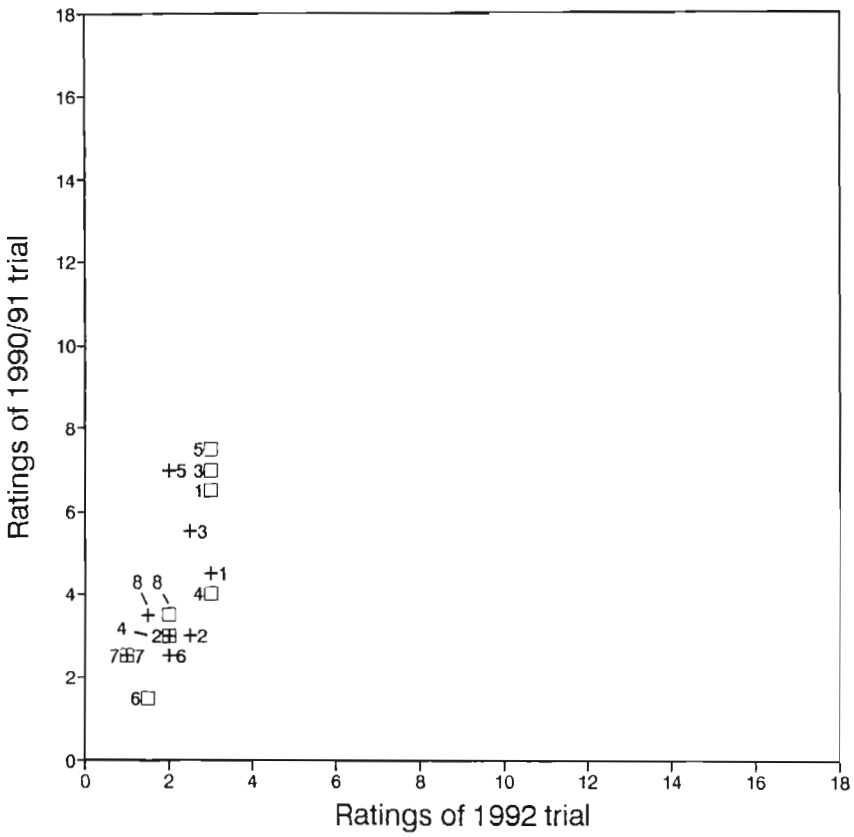
i. Rust ratings (% leaf area affected by rust)



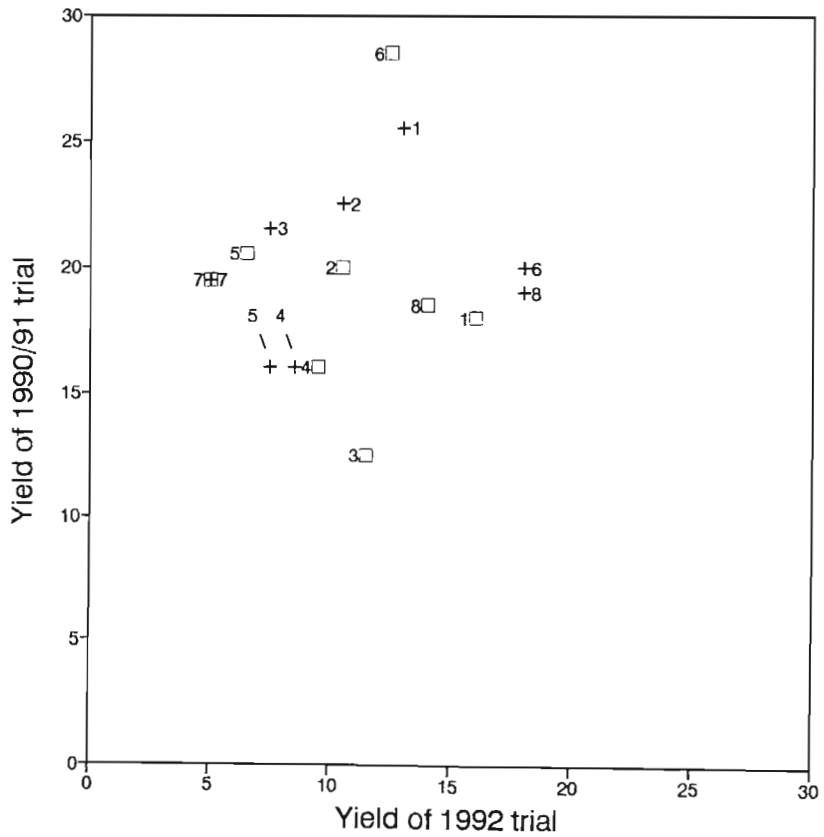
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5f. Genotype x environment interaction for GLP X 1132 (parent 6).



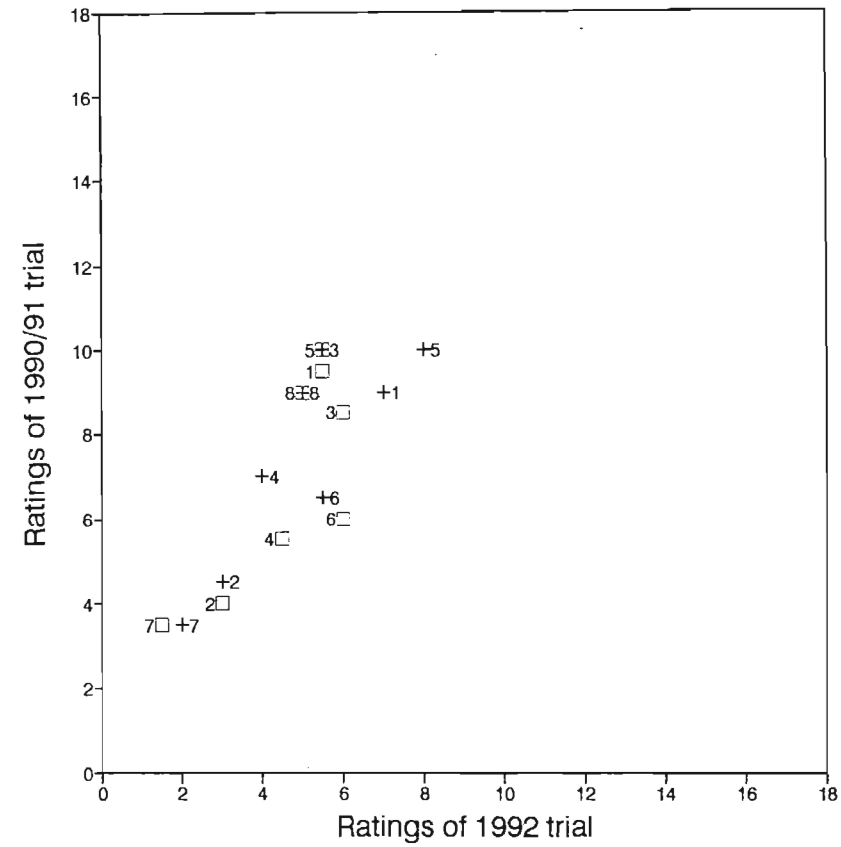
i. Rust ratings (% leaf area affected by rust)



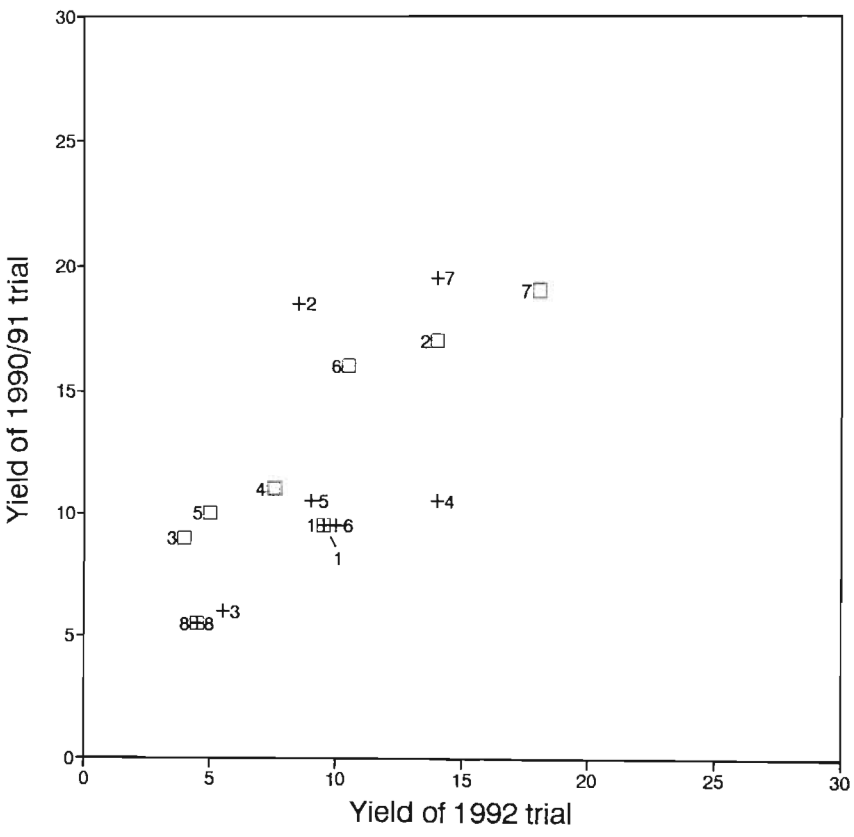
ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5g. Genotype x environment interaction for 259 AND 621 (parent 7).



i. Rust ratings (% leaf area affected by rust)



ii. Yield (single plant yield in grammes)

□, + are reciprocal crosses

Figure 5h. Genotype x environment interaction for 269 AND 631 (parent 8).

4.3.3 Infection trials with other single-pustule isolates

Success with these trials was limited (Table 15). Of the single-pustule isolates provided by Mr B.D. Garman, only two were viable (M1B and U6A). Further SPI's were expected from Mr Garman and the OPSC, but the people concerned left for other employment. A further limiting factor was to obtain a pure infection under conditions of natural spread, the SPI Beth test was the only one conducted in the field.

Table 15. Percentage leaf areas affected by rust of the 23 parent cultivars after infection with the SPI's U6A, Ced-1 and Beth.

Cultivar	U6A	Ced-1	Beth	Cultivar	U6A	Ced-1	Beth
PC 222-5-6-P2	6	4	25	KID 16	3	3	20
PC 223-4-1-D2	5	4	25	KID 20	5	5	12
PC 235-4-E3	7	3	20	COS 10	4	7	10
1266	0.5	1	3	AFR 241	3	3	20
1271	4	4	18	AND 340	0.5	2	10
1273	3	7	15	GLP X 1132	3	3	15
1312	0.5	1	7	339 AFR 290	4	4	3
ICA 15116	7	1	10	358 AFR 290	5	2	5
ICA 15521	4	*	8	259 AND 621	*	*	1
ICA 15522	3	*	5	269 AND 631	2	2	15
PVA 992	3	1	15	312 AND 689	2	2	1
				380 HAL 10	7	4	7

* = Occasional pustule noted. Plants were rated close to physiological maturity, for the SPI Beth it was 66 days after planting (12th May).

Most of the cultivars had equivalent levels of infection with the three single-pustule isolates compared to the SPI M1B results. The SPI Beth trial was complicated by an uneven infection. The border row of the cv. Bonus (planted perpendicular to the test rows) extended from PC 222-5-6-P2 to only AFR 241 due to a shortage of seed. In addition, the border row was planted at the same time as the main trial and not two weeks before as with the breeding and diallel trials (4.3.1 and 4.3.2). The epidemic developed adequately for the first ten cultivars and on a later time scale for cultivars towards the other end (380 HAL 10). Levels of infection of the

cvs KID 20, COS 10, 339 AFR 290 and 358 AFR 290 were somewhat lower than expected in comparison to the infection levels with the SPI M1B.

Poor infection was obtained in both tunnel trials, but there was a reasonable relationship between these results and the SPI M1B trials. Perhaps the poor infection obtained will only be overcome if the trials are conducted in the field. Similar tests with the SPI's Potch-1 and Wart-2 failed due to a lack of sufficient natural infection.

4.3.4 Leaf hair counts

All the 23 parent cultivars have long straight leaf hairs (acicular trichomes) along the veins and small, curved inter-veinal leaf hairs (unciform trichomes) when viewed on the abaxial side. The susceptible cultivars tended to be glabrous (leaf hairs absent) and the resistant cultivars were pubescent (a number of leaf hairs present) in the inter-veinal spaces (Table 16).

Table 16. Abaxial leaf hairs of the 23 parent lines.

Cultivar	First trifoliate	Third trifoliate
PC 222-5-6-P2	G (1/15 P)	G (1/15 P)
PC 223-4-1-D2	G	G
PC 235-4-E3	G(P)[1]	P
1266	P	P
1271	P	P
1273	P	P
1312	P	P
ICA 15116	P	P
ICA 15521	P	P
ICA 15522	P	P
PVA 992	G	G
KID 16	G	G
KID 20	G	G
COS 10	G	G
AFR 241	P	P
AND 340	P	P

GLP X 1132	G (1/15 P)	G (1/15 P)
339 AFR 290	G	G
358 AFR 290	G	G
259 AND 621	P	P
269 AND 631	P	P
312 AND 689	P	P
380 HAL 10	P (7/15 G)	P (5/15 G)

Fifteen plants were rated as G = Glabrous (no inter-veinal acicular leaf hairs present) or P = Pubescent (a number of leaf hairs present).
1 = Some plants had a very small number of leaf hairs present.

There were some important exceptions. The cvs 1271, 1273 and AFR 241 were as pubescent as 1266 but are highly susceptible. Several PC 235-4-E3 plants had a few hairs in the inter-veinal spaces towards the petiole on the first trifoliate. Examination of third trifoliate leaves indicated all the plants to be pubescent. PC 235-4-E3 had similar levels of rust infection compared to PC 223-4-1-D2 (glabrous), both are closely related. The moderately resistant GLP X 1132 was glabrous except one plant. The moderately susceptible 380 HAL 10 consisted of equal numbers of pubescent and glabrous plants on the first trifoliate (examination of the third trifoliate revealed 10/15 pubescent plants).

Abaxial acicular leaf hair numbers of the first trifoliate of the cvs 1266, 1273, ICA 15522 and KID 16, when veiwed under a dissecting microscope, appeared to correlate well with resistance except 1273 (Figure 6). Highly resistant 1266 had many leaf hairs; ICA 15522, moderately resistant, had ca. half the number of 1266; the highly susceptible KID 16 had no inter-veinal leaf hairs. But the highly susceptible 1273 also had many leaf hairs,

Figure 6. Leaf hairs of the first trifoliate leaf (magnification x 25).
a. Adaxial side, 1266.
b. Abaxial side, resistant 1266.
c. Abaxial side, susceptible 1273.
d. Abaxial side, moderately resistant ICA 15522. Note fewer hairs than 1266 and 1273.
e. Abaxial side, susceptible KID 16.

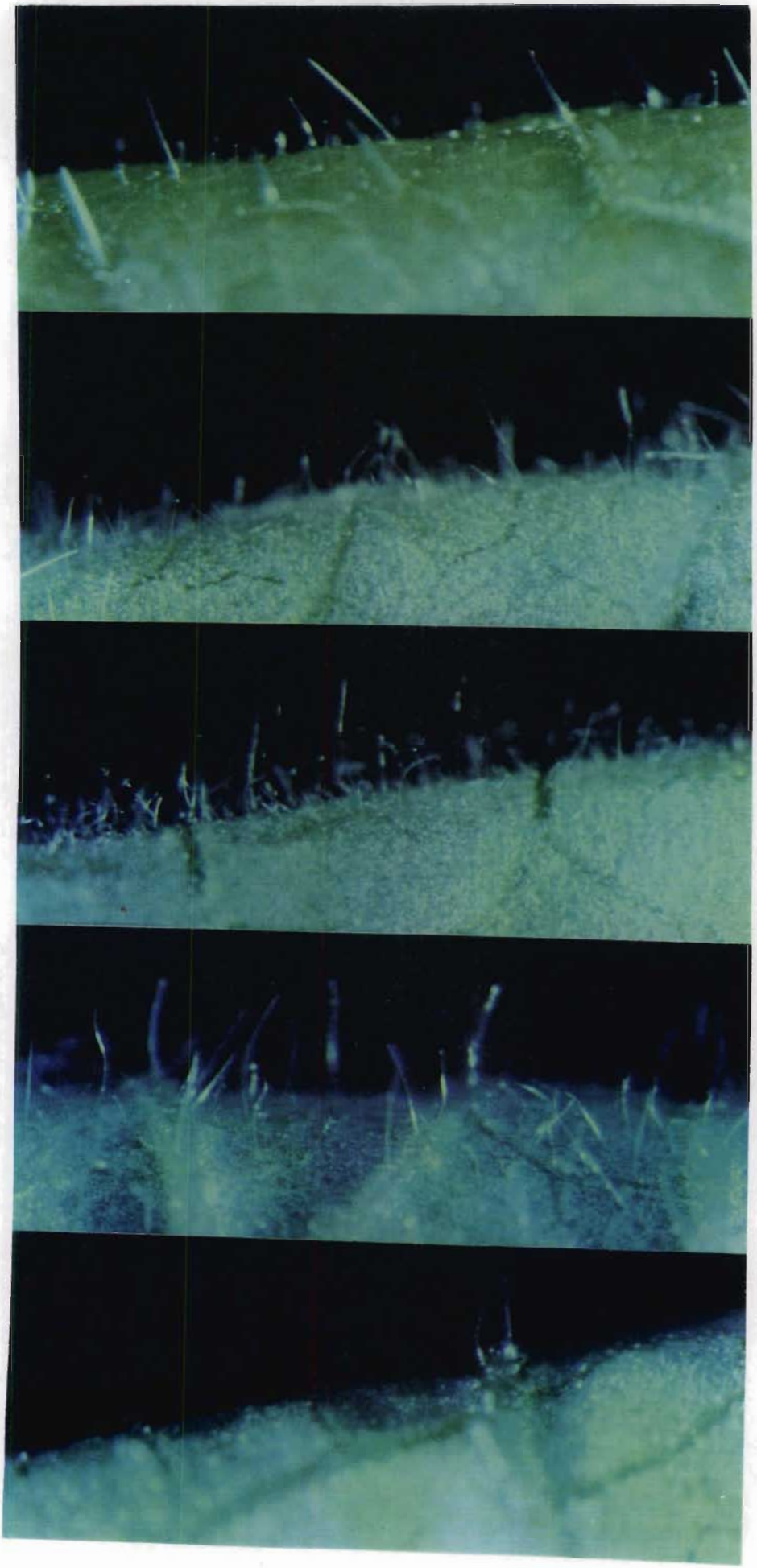


Figure 6
a

b

c

d

e

possibly more than 1266.

The number of acicular leaf hairs on the abaxial side of the third trifoliate leaves of 1266 was similar to that of 1273 (Table 17), although there was a large variation in hairs counted.

Table 17. Abaxial leaf hair counts of the third trifoliate leaves of the cvs 1266 and 1273.

1266	Mean	1273	Mean
20, 23, 29, 28, 35, 30	28	14, 20, 20, 28, 27, 18	25
18, 21, 18, 24, 22, 28	25	25, 25, 33, 26, 20, 24	26
24, 22, 20, 31, 23, 27	25	36, 36, 39, 34, 36, 30	35
20, 22, 18, 24, 24, 22	22	35, 39, 33, 24, 21, 28	30
38, 29, 25, 27, 32, 37	31	16, 18, 28, 25, 17, 20	21
17, 26, 32, 22, 22, 27	24	35, 26, 30, 36, 32, 33	32
21, 29, 14, 24, 26, 21	22	25, 23, 23, 30, 25, 27	26
23, 25, 28, 27, 31, 27	27	28, 24, 20, 31, 37, 28	28
Grand mean	26	Grand mean	28

Six counts were made on the middle leaflets of eight trifoliate leaves over an area of 0.5 mm^2 with the places chosen at random.

Adaxial acicular leaf hair counts were also very variable (Table 18), even allowing for leaflet area.

Table 18. Adaxial leaf hair counts

Cultivar	Rust rating[1]	1[2]	2	3
PC 222-5-6-P2	20	10	6	7
1266	4	27	7	11
1273	16	24	10	12
ICA 15522	10	5	13	8
KID 16	26	14	7	8
GLP X 1132	8	16	9	6
259 AND 621	2	12	9	6
269 AND 631	10	16	12	14

1 = % leaf area affected by rust of the cultivars in the 1990/91 breeding trial on the 3rd of January.
2 = The first test is the mean of four counts of a single leaflet with three replications. The second and third tests are the means of three counts of a single leaflet with eight replications. The figures given are the number of hairs in a microscope field of view (ca. 1.0 mm²)

Table 19. Analysis of variance of the first leaf hair count.

Source	DF	SS	MS	VR
Cultivar	7	14.81	2.116	3.31 *
Residual	16	10.23	0.640	
Total	23	25.04		

Analysed with a square root transformation. CV = 19.7 %.

Table 20. Analysis of variance of the second and third leaf hair counts.

Source	DF	SS	MS	VR
Block	1	0.5	0.5	0.06
Cultivar	7	512.25	73.170	8.68 ***
Residual	119	1003.125	8.43	
Total	127	1515.875		

Analysed with a square root transformation. CV = 16 %.

There were significant differences between the number of leaf hairs on the adaxial surface of the cultivars (Tables 17 and 18). More leaf hairs did not necessarily correlate with greater resistance (cf. cvs 1266 and 1273).

Density of abaxial and adaxial leaf hairs is unimportant in the partial resistance found in this study. Leaf hair length was variable, even between leaves of the same cultivar, perhaps further investigation is warranted.

4.3.5 Stoma number

Numerous stomata were found on the abaxial side of trifoliate leaves of all

the cultivars examined. No attempt was made to count them but a very cursory scan revealed no major differences. The susceptible cultivars PC 222-5-6-P2 and KID 16 had many stomata on the adaxial side compared to the resistant cultivars 1266, 259 AND 621 and 312 AND 689 and the moderately resistant ICA 15522, although the number was considerably smaller than on the abaxial side. Susceptible cultivars 1273 and AFR 241 also bore few stomata on the adaxial side. PC 222-5-6-P2 and KID 16 were glabrous, but the glabrous PVA 992 also had few stomata on the adaxial side.

Number of adaxial stomata appears to have little relation to the partial resistance of the cultivars to the SPI M1B, based on the trends observed. The relationship of the abaxial stomata remains to be clarified. No attempt was made to look for a relationship between stomatal structure and the partial resistance to the SPI M1B.

4.3.6 Latent period tests

Differences between latent periods in the primary leaves after inoculation with the SPI M1B of the first three ranking groups of Table 19 were minimal. But the difference between 1266 (11 dpi) and PC 222-5-6-P2 was two days. In a second test, the magnitude of differences was smaller with a difference of a day between eruption of pustules in 1266 and PC 222-5-6-P2. In ICA 15522, KID 16, GLP X 1132, 259 AND 621 and 269 AND 631 pustules had erupted by 11 dpi and in 1266 and 1273 it was possibly earlier (< 1 day).

Table 21. Rankings of the eight diallel cultivars according to length of latent period.

Cultivar	Rust rating[1]	LP 1	LP 4
PC 222-5-6-P2	20	4	1
1266	4	1	4
1273	16	2	1
ICA 15522	10	3	3
KID 16	26	2	3
GLP X 1132	8	1	3
259 AND 621	2	2	3
269 AND 631	10	1	2

Latent period (LP) 1 and LP 4 are the results are from the primary leaf and fourth trifoliate tests.
1 = % leaf area affected by rust of the cultivars in the second breeding trial on the 3rd of January.

A test of primary leaves of the 23 parent cultivars with the SPI Wart-2 indicated a similar trend as above. The cvs 1266, 1271, 1273, KID 16 and PVA 992 were the first to erupt (eight dpi) and the cvs PC 222-6-P2, PC 223-4-1-D2, PC 235-4-E3 and 339 AFR 358 were the last to erupt (10 dpi).

Results of the primary leaf inoculations of 1266, 1273, ICA 15522 and KID 16 revealed no differences. Inoculations of the second trifoliate revealed no differences for 1266, 1273 and KID 16. A poor infection of ICA 15522 was obtained with a delayed eruption, but this may be due to experimental error. All the plants were beginning to be stressed by pustule eruption after the second inoculation as five plants were maintained in 150 mm pots.

Results (from two tests with the SPI Beth) of the latent period test in the fourth trifoliate leaf begin to match the resistances of the cultivars. The cvs PC 222-5-6-P2 and 1273 had the shortest latent periods (8 dpi), closely followed by 269 AND 631. The others erupted the following day, except 1266, erupting a further day later.

The order of eruption of PC 222-5-6-P2 and 1266 were reversed when comparing the primary and fourth trifoliate leaves. Hence a test of the primary leaves is inadequate to test latent periods for adult plant resistance. Latent periods of 1266 and 1273 were closely matched in the primary and second trifoliate leaves. It was only in the fourth trifoliate that differences in latent period matched resistance. In addition, the latent periods of KID 16 and 259 AND 621 did not correspond with resistance. Longer latent period *per se* is probably not the cause of resistance but a characteristic of it.

4.4 Breeding for multiple disease resistance

No major problems were experienced in this crossing programme, except the

programme was held up on occasion by the lack of availability of inoculum. With angular leaf spot resistance testing, for example, sometimes only the F_3 was tested. An occasional plant with genetically conditioned chlorosis or mottling was discarded, but a trend was not observed.

Large seed size was not maintained as the number of crosses increased. As noted earlier, most of the resistance sources were small-seeded cultivars. In spite of selection in favour of larger seed size, where possible in earlier generations, back-crossing is essential to maintain seed size. As large seed size was maintained in the earlier crosses, it is unlikely that the resistances are closely linked to seed size.

With the large variety of seed colours and markings it is not unexpected that the final breeding line has little seed with a red speckle on cream or white background of the desired speckled sugar type. Back-crossing to a sugar bean cultivar would improve the chances of obtaining a disease-resistant cultivar with a larger seed size and desired seed colouring.

Disease testing at the seedling stage was aided by using small 24-cell Speedling® trays. These trays were of a convenient size for use in the dew chamber and plastic bags, and individual cells made roguing easier. As the plants outgrew the cell size, they were potted-out. At this stage testing for Ascochyta and common blight resistances occurred.

Rating of plants for resistance to angular leaf spot, anthracnose, halo blight and rust pathogens was very easy. In tests for Ascochyta and common blight resistances, where resistance was not discrete, tests were somewhat subjective. For Ascochyta and common blights, plants with a greater degree of symptom development were rogued and it was hoped that this matched their levels of resistance (this would not eliminate disease escapes). Ideally, evaluation of plants for resistance to Ascochyta and common blight should be done under field conditions with spreader rows.

Reasonable success was achieved with the field trial conducted in the disease garden with the NC 38 line advanced one generation by single-seed descent. This was inspite of fairly severe damage caused by a hail storm on the 9th of March. Inoculation of Teebus with C.lindemuthianum resulted

in severe anthracnose symptoms but only occasional lesions were noted in five NC 38 plants. Mild halo blight symptoms were noted in three NC 38 plants out of a total of 81. There is a possibility of disease escapes as the inoculation for anthracnose and halo testing was followed by hot and dry weather. A plant that had anthracnose also had angular leaf spot symptoms. Another plant had rust and ALS symptoms. Of the remaining 59 plants on the 29th of March, a total of two had rust and three had ALS. A successful infection of Ascochyta blight was achieved. A range of severity was observed on the test plants and the cv. Bonus with the former bearing milder symptom development. With both groups, however, little re-infection of new leaves occurred. The inoculation with X.campestris pv. phaseoli resulted in too few lesions for an accurate assessment of the relative resistances. By this stage little new growth was being produced due to the advanced age and plants were severely affected by the Ascochyta infection as it followed on from the hail damage.

Several plants in this trial were lost due to abiotic damage (such as hail and wind) and biotic damage (such as snails and root rots) which would not have occurred under glasshouse cultivation. In addition, the halo blight infection probably did not identify all the susceptible plants as the inoculation was followed by hot and dry conditions. Again this could have been avoided under glasshouse conditions.

4.4.1 Field trials by Pannar Seed

Results of field trials of material passed on to Pannar Seed as provided by Mr A. Jarvie are as follows:

NC 7 (Cross for halo and common blight resistance): Nineteen selections were made from 1991 summer trials; presently in 1992 winter trial awaiting selection and harvesting. Further field trials have indicated no selections of agronomic interest.

NC 19 (Cross for Ascochyta blight and angular leaf spot resistance): Eighteen selections were made from 1991 summer trials; presently in 1992 winter trial. Nine additional selections made from F_4 populations were planted in 1991 winter nursery and of these, two lines were planted in 1992

summer trials, but were later rejected.

NC 20 (Cross for angular leaf spot and rust resistance): Nineteen selections were made from 1991 summer trials; presently in 1992 winter trial. Further field trials have indicated no selections of agronomic interest.

NC 13 (Cross for angular leaf spot and rust resistance): Six selections were made from 1991 summer trials; due to shattering only three selections were retained after 1991 winter trials. Five selections were retained after the 1992 summer trials, these had good yield and growth habits and improved resistance to shattering. Of the four selections grown in the 1993 summer trials, one was not rust resistant and, unfortunately, this was the only one without shattering. A single selection has been retained for its good rust and angular leaf spot resistance. The selection's other drawbacks include hardseedness (leads to poor and slow germination), a low, flat bush and late maturity.

NC 38 (Cross for all the disease resistances): General performance in the 1993 summer trials was poor, but forty-two single-plant selections were made based on yield, a larger seed size and disease resistance. Single-plant selections (one row per selection) were rated for BCMV, angular leaf spot, Ascochyta blight, common blight and rust during the 1994 summer trials planted at Greytown. The trial was planted somewhat late (17th January) to favour disease spread, although an earlier date would have favoured halo blight incidence. Disease incidence occurred by natural spread.

Nineteen selections were rated as rust-resistant, with no or very few pustules. Twenty-one selections were rated as resistant to angular leaf spot, with no sporulating lesions. Nineteen selections were rated as resistant to Ascochyta blight, with no apparant lesions. Thirteen plants were rated as common blight resistant, with no apparant lesions. The mean rating of common blight within the trial was 0.97 and 1.8 in adjacent trials indicating good level of resistance in the NC 38 population as a whole. The low frequency of resistance compared to that in the NC 38 field trial reported in the previous section, is due to selection for yield and seed size during the preceding trial. Two speckled sugar bean cultivars were also planted in the 1994 trial. Kranskop had no rust, a moderate infection

of angular leaf spot, and occasional lesions of Ascochyta and common blight. PAN 127 had a moderate infection of rust, fairly moderate infection of angular leaf spot and Ascochyta blight and occasional lesions of common blight. Yields were not measured due to hail damage.

The selections were also tested for BCMV resistance by planting ten seeds of each selection in a BCMV-infection nursery. Thirty-three selections were rated as homozygous for the necrotic reaction due to the I-gene and three were rated as susceptible. One selection was rated as symptomless (no necrotic or typical mottle symptoms) and five selections were rated as heterozygous with plants exhibiting necrosis and other plants with no symptoms. Three selections were rated as resistant all five diseases. Susceptibility to BCMV was presumably inherited from the Great Northern cultivars and maintained in the heterozygous state whilst testing for the presence of the I-gene was conducted.

SC lines (Crosses for horizontal rust resistance): Forty-one SC lines were planted at Greytown and/or Delmas in the 1991/2 season. Two lines, SC 126-1 and SC 180, were selected for use as parents. Both exhibited good hybrid vigour in the F_1 of their crosses. Results of the rust ratings were highly variable. Of the 33 lines rated at both places, 11 were resistant at both localities, 12 were susceptible at both localities, seven were resistant at one locality but susceptible at the other and three were resistant at one locality and segregating for resistance and susceptibility at the other. Selections of the SC lines were based on the rust resistance of the RC line as a unit from which it came from and there is a possibility of disease escapes. In addition, several SC lines were chosen from a RC line with poor resistance due to good seed characteristics (yield, colour and speckle). It is somewhat worrying that some lines had a good level of resistance at one locality but not the other. Horizontal resistance implies a similar level of resistance at all localities assuming the same disease pressure.

Results of the 1993 summer trials at Greytown were more promising, although the trial was planted late and may have reduced rust incidence. The SC lines planted generally had good rust resistance, but there was none with immediate commercial potential. General performance of the TC lines was superior to the SC lines and single-plant selections have been made. The

SC and TC lines included several that were resistant to angular leaf spot and they varied greatly in response to scab (Elsinoë phaseoli Jenkins), which was a major problem in the trials.

The 1994 summer trials of the SC and TC material were decimated by hail.

4.5. Genetics of resistance

4.5.1 Angular leaf spot

Both F_1's of the crosses of Carioca 80 and PAI 127 with PC 232-1-D2 were rated as resistant at 14 dpi. At 28 dpi the F_1's had lesions half the size of the susceptible check, PC 232-1-D2. In the cross with Carioca 80 (NC 13), a F_2 ratio of 20 resistant plants to 7 susceptible plants was obtained at 14 dpi. By 28 dpi, the plants were rated as 15 resistant and 12 susceptible. In the cross with PAI 127 (NC 12), a F_2 ratio of 12 resistant plants to 13 susceptible plants was obtained at 14 dpi and 8 resistant plants to 17 susceptible plants at 28 dpi. In two additional tests of Carioca 80 crossed with PC 235-4-E1 (NC 16), F_2 ratios of 32:14 and 46:15 of resistant to susceptible plants were obtained. In the cross between Carioca 80 and PAI 127 (NC 26), 60 plants of the F_2 showed no symptoms.

It would appear that the angular leaf spot resistance of Carioca 80 is governed by a single dominant gene and that of PAI 127 by two dominant genes. One of the resistance genes in PAI 127 is probably the same as that as found in Carioca 80. In both cases the resistance is not fully dominant. A similar situation to that of PAI 127 may occur in Mkuze, a ratio of 40 resistant to 36 susceptible plants was obtained after inoculating the F_2 of a Mkuze cross with the susceptible Natal Speckled Sugar (I.P.Holmes, pers. comm., 1989). The resistance of Mkuze was postulated as conditioned by the epistatic interaction of two dominant genes.

4.5.2 Full rust resistance

All fifty-two plants of the F_2 of a cross between Mkuze and Carioca 80 (NC 24) were rated as resistant (pin-point lesions) after inoculation with a

sample of rust from the University's disease garden. Results of testing a Carioca 80 cross with a susceptible cultivar are given in 4.5.3.

4.5.3 The slightly incompatible reaction

Inoculation of the tobacco cv. Samsun failed to reveal any virus symptoms and it is assumed the slightly incompatible reaction is a result of rust infection alone.

Tests with samples of rust from Cedara, Empangeni and Potchefstroom inoculated onto in the cv. Epicure resulted in typical ring syptoms. A fully susceptible reaction was, however, found after inoculation with the sample from Empangeni.

Inoculations of Epicure plants maintained at 20/20°C and 30/20°C day and night temperatures, respectively, with a rust sample from the University's disease garden, indicated a higher degree of necrosis associated with the warmer temperature (Figures 7 and 8). At 30°C day temperature there was collapse of necrotic tissue and no sporulation. At 20°C pustules of ca. 0.5 mm dia. formed with necrotic rings of ca. 0.5 mm width formed by 14 dpi. It is interesting that necrosis followed the veins for a small distance giving the appearance of a transmitted elicitor in plants maintained at 20°C day. In contrast to the Epicure tests, inoculations of NC 16 plants with ring necrosis symptoms after inoculation with the SPI M1B revealed minimal temperature sensitivity. At seven dpi the plants maintained at 30°C after infection (those maintained at 30/20°C day and night and at 18°C until after infection) had slightly more collapse of tissue due to ring necrosis. No differences were observed in plants maintained at 18°C after inoculation (0, 4 and 8 hours at 30°C before inoculation). Wei (1937) found temperature to be one of several environmental factors influencing the ring (mesothetic) reaction. In addition, leaf age was also an important factor in determining symptom severity as also indicated by Wei (1937), younger leaves exhibited more necrosis.

The F_2 of the cross between Epicure and PC 235-4-E3 (RC 53) gave 19 resistant (pin-point lesions or uredia) plants and six plants with ring symptoms after inoculation with the SPI M1B. Further tests of the F_3

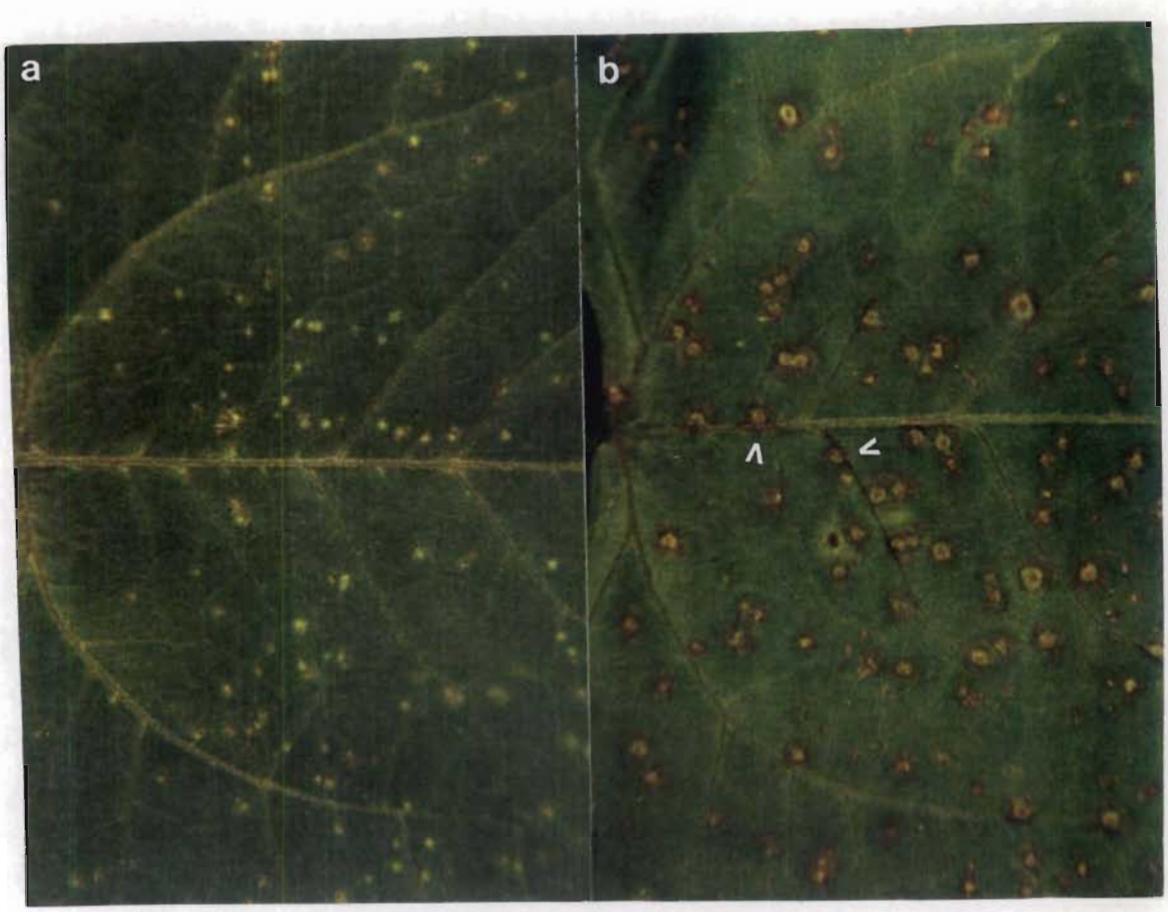


Figure 5. Primary leaves of Epicure 7 (a) and 14 dpi (b) after inoculation with rust from Pietermaritzburg and maintained at 20°C day and night. Note increased necrosis in the vein where a pustule abuts (arrows).

Figure 6. Primary leaf of Epicure 7 dpi after inoculation with rust from Pietermaritzburg and maintained at 30°C day and 20°C night. Little further symptom development occurred.

indicated the presence of two dominant resistance genes where one gene confers full resistance which is dominant over the other gene which conditions the ring reaction which, in turn, is dominant over full susceptibility. A similar situation was found in the cross between PVA 2280 and PC 235-4-E1 (NC 21). The F_2 of NC 21 gave 45 resistant plants, 12 plants with ring symptoms and three fully susceptible plants. The crosses between Epicure and susceptible lines from the horizontal resistance programme also indicated that the conditioning of the ring reaction is by a single dominant gene. The RC 10 F_1 (Epicure x PC 222-5-6-P2), RC 73 F_2 (Epicure x 1266) and the unknown RC F_2 (Epicure x a susceptible) gave 5/5, 10/13 and 18/25 plants with ring reactions, respectively, after natural infection in the disease garden. In addition, PC 235-4-E3 had a severe infection, Redlands Greenleaf C was moderately infected and Epicure had typical ring lesions. The F_2 of the cross (NC 28) between Epicure and Redlands Greenleaf C gave 15 plants with ring reactions and 8 susceptible plants. Although this does not fit that well to a 3:1 ratio, it would appear that Redlands Greenleaf C does not have genes which conditions a ring reaction to rust infection with the rust race(s) tested.

The F_2 of a cross between Carioca 80 and PC 235-4-E1 (NC 16) was tested in two sets with rust samples from Potchefstroom and Pietermaritzburg, respectively. The two tests resulted in ratios of 28 resistant: 7 ring-bearing: 5 susceptible plants and 38 resistant: 5 ring-bearing: 3 susceptible plants, respectively, with a total ratio of 67:12:8. This suggests the presence of three dominant resistance genes with one gene that confers full resistance dominant over the other two, where epistatic interaction of the dominant alleles conditions the ring reaction. The results of an inoculation of the F_2 of a cross (NC 27) between ring-bearing plants of NC 16 and PC 222-5-6-P2 with the SPI M1B, however, indicated a single dominant gene conditioning the ring reaction, 42 plants had ring symptoms and 11 were susceptible.

Of the 72 plants of the F_2 of the Epicure cross with PI 165426 (NC 37) tested with the SPI Ced-1, 49 plants bore pinpoint lesions (same as PI 165426) and 23 had ring lesions (same as Epicure). In another test with the SPI Potch-1, all 30 plants tested bore ring lesions identical to both parents. It is unlikely that Epicure and PI 165426 have different genes

conferring the ring reaction. Of the 83 plants of the F_2 of a cross (NC 42) between plants bearing ring symptoms of NC 16 and RC 53, 77 plants bore ring symptoms after inoculation with the SPI M1B. The gene conditioning ring necrosis in Carioca 80 and Epicure is the same. The remaining six plants were cripples with general chlorosis and died shortly after expansion of the unifoliate leaves. The cripple condition was possibly conditioned by two recessive genes, one from each cultivar, with the combination leading to death. Although there is a possibility that the plants were not true breeding for this condition; heterozygosity or homozygosity of the dominant allele is not ruled out.

Differences in reaction to infection with the SPI M1B were noted amongst F_3 plants of RC 53. In the more severe form, symptoms were similar to those originally found in Epicure and occurred 3 - 4 dpi. A milder form was also found at pustule eruption *ca*. 11 dpi. Reciprocal crosses with PC 222-5-6-P2 revealed no maternal factors affecting inheritance of the two types in the F_1. The F_2 of the severe ring cross (NC 37) reacted as expected, but the mild form (NC 36) was similar to that of susceptible RC 53 plants. At the time of pustule eruption there was no necrosis and it only became apparent 20 dpi. In the F_1 of the mild form crosses, necrosis also appeared at the time of pustule eruption at 15 dpi. Perhaps the mild form is more sensitive to environmental conditions than the severe form.

In the cross (NC 18) between Broadacres and Mkuze an occasional plant was found with a ring reaction after rust infection. No attempt was made to obtain ratios, but it appeared that full resistance was dominant over the ring reaction which in turn was dominant over full susceptibility as with Carioca 80. Inoculations of a cross (NC 25) between ring-bearing plants derived from Carioca 80 (NC 16) and Mkuze (NC 18) failed to identify if the gene(s) conditioning the ring reaction were the same, as the Mkuze plants were heterozygous. Symptoms of the F_1 were, however, identical to the parent lines.

Good results were obtained after inoculations of the F_1 of the crosses between RC 53 and the diallel cultivars (NC 29 to NC 36). Faint symptoms of necrosis were visible four dpi and good symptom development was noted the following day except the cross with PC 222-5-6-P2. Similar symptoms were

observed for the PC 222-5-6-P2 cross except that it was two days later than in the other crosses with no other chronological differences noted. Plants of RC 53 inoculated as controls with the crosses had similar symptoms to most of crosses except crosses with 1266, 1273 and KID 16. In the 1266 and 1273 crosses, which were similar to each other, several ring symptoms developed later than others on a time scale similar to that of the PC 222-5-6-P2 cross. With the KID 16 cross, ring symptoms were slightly larger (ca. 0.5 mm), but this may have been due to the leaves being slightly older than those of RC 53. It would appear that the longer latent period of PC 222-5-6-P2 is conditioned by dominant gene(s) as the time scale for symptom development is similar to that of the latent period tests in 4.3.5.

4.6 Breeding for Ascochyta blight resistance

Plants of Carioca 80, Mkuze, PAI 6, PAI 127 and NC 19 (PAI crosses for Ascochyta blight resistance from the multiple-disease resistance programme), grown in the disease garden, initially had low levels of infection of Ascochyta blight, but incidence increased after onset of flowering. The PAI lines had a slightly lower incidence than Carioca 80 and Mkuze and single-plant selections were made from NC 19. NC 19 plants susceptible to rust after natural infection were rejected and a sugar bean seed type was favoured in the selections.

The NC 19 selections were mixed and increased in the glasshouse. The next trial in the disease garden with NC 19, Mkuze, ASC 4, BAT 795 and VRA 81018 was irrigated daily by an over-head sprinkler and this dramatically increased Ascochyta blight incidence. Differences in resistance were noted before flowering, the latter three cultivars were all late-flowering, but once flowering had begun the cultivars appeared to have similar levels of resistance. All the cultivars were quite susceptible with Mkuze being only slightly more so.

The following trial with NC 19, BAT 795 and NC 39 (BAT 795 x NC 19) also had a high level of incidence and low levels of resistance with no apparent differences. As a result, work was discontinued with NC 39.

It would appear that the resistance to Ascochyta blight examined in this study is easily overcome under high disease pressure. Perhaps all that can be hoped for is to select for a line that would not be as susceptible as Mkuze or Carioca 80.

CHAPTER FIVE

DISCUSSION

5.1 Breeding for multiple disease resistance

Success was attained in combining resistances to the major foliar pathogens of beans together in one breeding line. It is presently undergoing field testing by Pannar Seed and single-plant selections are being made. The breeding line is available for use as a source of disease resistance to all the major foliar pathogens of beans in South Africa. On the Highveld, for example, the important diseases of beans are BCMV, halo and common blight and rust and hence a cross with the disease-resistant line might be selected for these disease resistances alone.

I-gene resistance to BCMV, derived from the cvs UI 50 and UI 51 which have a combination of a large seed size and the I-gene that is not linked to an undesirable testa colour, was used. Race non-specific resistance to halo blight was obtained from the great northern cvs GN Nebraska No 1 Selection 27 and GN Tara. The great northern cultivars were also the source of common blight resistance. Angular leaf spot resistance used, was found in the cvs Carioca 80 (conditioned by a single dominant gene), PAI 127 (conditioned by two dominant genes) and PAI 6. The Are-gene from Cornell 49242 was used for resistance to anthracnose. PAI 6 and PAI 127 were used as sources of moderate Ascochyta blight resistance. Carioca 80 was used as a source for rust resistance. This resistance may be the same as that of another carioca bean, Mkuze (A286), which has proven to be resistant in the National Cultivar Trials (Internal Reports of the OPSC).

A secondary aim was to achieve the combined resistances in a line with a large seed size and red speckle of a sugar bean type. Unfortunately, this was not achieved. The seed size of the breeding line declined as the number of resistances combined increased. Perhaps the breeding line could be used to increase the disease resistance of small-seeded types. Seed size was maintained in the early stages when crosses for just one or

two disease resistances were made, indicating only a loose linkage between seed size and disease resistance. Hence seed size could have been increased if several back-crosses had been made within the programme, but this would have delayed the combining of the disease resistances. The possible back-cross parent used will also help determine the seed colour, speckle and shape of the final product. The breeding line has several different colour segregants, few of which are the desired type.

Inoculation of the pathogens used simple techniques and could be easily done under conditions prevalent at most plant breeding companies. Little equipment other than glasshouse and field space is required. Inoculation of the anthracnose and Ascochyta blight pathogens was aided by use of pure cultures, but washings of diseased plants can also be used. Glasshouse inoculations of pathogens to which vertical resistance was available (angular leaf spot, anthracnose, BCMV, halo blight and rust) were more than adequate in eliminating susceptible plants. The vertical resistances operate in seedling and adult stages of the plant allowing testing at any stage. With Ascochyta and common blight resistances used in this project, which have a continuous distribution from susceptible to resistant plants, field testing would be superior. In the field, natural spread of disease would allow selection on several dates and under conditions similar to which a new cultivar would be grown under. Use of the common blight vertical resistance of the cv. XAN 159 would obviate the need for field testing for common blight resistance.

In the period that this project was undertaken, 1988 - 1994, extremes of climate were encountered. Towards the latter part, drought made field testing of disease resistance difficult. This was particularly evident in the testing of lines bred for Ascochyta blight-resistance given to Pannar Seed for field evaluation. Poor symptom development was noted as conditions were unfavourable for spread of cool, wet-weather diseases such as Ascochyta blight. This may lead to the selection of disease escapes that do not have true resistance. As a result, the lines were evaluated for agronomic performance rather than disease severity. This is rather unfortunate as, noted above, poor glasshouse evaluation for Ascochyta blight was obtained. It is possible that the resistance tested in the glasshouse was inadequate.

Several cultivars from a CIAT Ascochyta blight nursery were tested for Ascochyta blight resistance at Cedara. Seed of ASC 4, BAT 795 and VRA 81018 was obtained for further testing. In addition, a cross between BAT 795 and NC 19 (a line developed for Ascochyta blight resistance from the multiple disease resistance programme) was also tested. Poor resistance was noted at the University, but the conditions were very favourable for disease spread with daily over-head sprinkler irrigation. Another possibility for the poor resistance is that most overseas work appears to have been conducted with P.exigua var. diverspora. The causal organism of Ascochyta blight in South Africa was reported as P.exigua var. exigua (Trench et al., 1986). Hanson et al. (1993) reported moderate levels of Ascochyta blight (P.e. var. diverspora) resistance within P.vulgaris germplasm with low to moderate levels of narrow-sense heritability (0.19 - 0.64). They expressed a concern that the resistance may be insufficient to prevent significant yield loss. An increase in Ascochyta blight susceptibility after flowering, as found in this study, was also noted.

As a greater control of the glasshouse environment can be achieved, progress can be made independently of field conditions. Plants can be transferred to conditions most favourable for inoculation and symptom development of each disease. At least three generations of beans per year can be raised in the glasshouse compared to two in the field. With Pietermaritzburg, the second generation would have to be grown somewhere such as the Makhatini Flats, Natal, where it is frost-free, thus causing greater expense.

It is difficult to rate many of the agronomic characteristics of beans under glasshouse conditions. A bean cultivar may be perfect in every respect, but would be of no use if, for example, the yield was shattered over the ground. Ideally, a cross between the disease-resistant line and the desired cultivar would be tested in the glasshouse for disease resistance. Further progenies would then be tested under field conditions close to normal growing conditions. Field evaluations of disease resistance can also be made. The test plants can be inoculated, but ideally a natural infection, with or without the aid of spreader rows, should be used.

A possible difficulty that may arise with glasshouse evaluation is the age at which the plant should be inoculated. In the field, the plant is

continually challenged by the pathogen assuming conditions are adequate for infection. The inoculation of the pathogens in this project occurred before flowering and mostly in the seedling stage. Fortunately, the seedling angular leaf spot and rust resistances were maintained in field trials conducted by Pannar Seed. The BCMV, halo blight and anthracnose resistances used in this project have not been reported as having different seedling and adult plant reactions. The common and Ascochyta blight resistances used, do have higher levels of resistance before flowering but these were the best resistances that were available at the start of the project.

Use of vertical disease resistance is advocated in this system as it is far easier to evaluate under glasshouse conditions and rapid progress can be made due to its simple Mendelian inheritance. But, by definition, it is assumed that vertical resistance can break down. With anthracnose, for example, the Are-gene would be useless in South America due to presence of races that can overcome it. Hence a cultivar bred from the multiple-resistant line would be of little use in South America. But at this stage no race is known in southern Africa that can overcome it and its use is still justified. The definition of individual races may also become important. The single-pustule isolates of rust M1B and U6A gave similar responses in the rust differential cultivars (although the inoculations were not quantitative) and only differed in the response on the local cv. Teebus. Is the SPI U6A a variant of the SPI M1B, or vice versa, or a separate race? Ideally, a differential series should consist of lines with single resistance genes, but this has not been attained with beans and bean rust.

With pathogens that exist as many races and can be rapidly dispersed such as the rusts, the breakdown of resistance is expected far sooner. In these cases individual races must be stored for testing against possible resistance sources. After preliminary tests with rust from Potchefstroom, Bethal and the Pietermaritzburg area only, at least eight races of rust were identified and probably more await discovery. With rust, if the resistance of Carioca 80 was overcome by a new race, then a new resistance source must be found. The new resistance would then be tested against all prevailing races and if it confers resistance, it could be used alone. But if some prevailing races were to overcome the new resistance it would have to be used with that of the Carioca 80 resistance, which would confer resistance

to prevailing races. This does not exclude the possibility of the formation of a "super" race which may progressively develop as different resistances are used. The rust pathogen can bear unnecessary virulence genes (Alexander et al., 1985) and recessive virulence genes can be masked in a heterozygous state (McCain et al., 1990). As a result, even if vertical resistance genes were to be "rested" it is possible that a super race could still develop. This assumes that linkage of certain virulence genes does not lead to a reduction in reproductive fitness of the race. This possibility (the ABC-XYZ system of Vanderplank (1982)) may have been found by Alexander et al. (1985). The durability and efficacy of such an ABC-XYZ system would, however, probably be only known in retrospect.

If resources do not allow the storage of every race then the cultivar must be tested with rust from at least three localities. Rust from Potchefstroom proved to be more virulent than that from Delmas (comparing glasshouse tests of cultivar susceptibility of a sample of rust from one season to international bean rust nursery trial results in the previous season). Testing for resistance with Potchefstroom rust may be a better test than with that from Delmas, but Delmas is a very important centre of bean production. Besides rust from Potchefstroom and/or Delmas, tests should be conducted with rust from Natal, perhaps Cedara or Ukulinga, and the Low Veld. The Low Veld testing could easily be done as part of a winter-increase nursery. This should challenge the cultivar with a range of races as found in South Africa, but the National Cultivar Trials, with trials throughout the major bean-growing areas in South Africa, will give an even better test before cultivar release.

Beans are an important, but not a major crop and the scale of cultivation in South Africa is not as large that one might expect the advent of new pathogen races on a large scale. Vertical resistance may prove to be a viable option if steps are taken to prolong its usefulness. Isolines of a cultivar could be developed, but instead of planting a mixture of isolines, only one isoline is planted per period (one to three seasons) and followed by the next. The seed company must be able to sell each isoline as the same cultivar (perhaps with a number suffix) to avoid grower rejection, and the grower need not necessarily be made aware of changes to the "cultivar". With the development of new genetic techniques it may soon be possible to

introduce genes, such as vertical resistance genes, without labourious back-crossing. The main failing of such a system would be a grower failing to replace the isoline. His crop may not be heavily infected, but it could act as an inoculum source for surrounding areas, where conditions may be more favourable for an epidemic.

As beans are a self-pollinating crop, growers can maintain a cultivar from farm-grown seed after an initial purchase. At present, many growers buy certified disease-free seed every second year and alternate crops are planted with farm-grown seed. If resistant cultivars are used, the increased seed contamination which prevents further use of farm-grown seed would fall away. But a seed company producing a new cultivar requires an adequate return on its investment and by producing a multiple-disease resistant cultivar it may be cutting its profits in the long term.

5.2 The slightly incompatible reaction in response to rust infection

Ring necrosis was found in the cv. Epicure after a natural rust infection. It was studied as a possible source of horizontal resistance but the cv. Epicure was fully susceptible to rust from Empangeni. The reaction, as indicated below, is a form of partial vertical resistance.

The ring necrosis reaction after rust infection was first reported by Wei (1937) and called it a mesothetic reaction. Wei (1937) found 13 out of 44 cultivars tested responded with a mesothetic reaction. Ballantyne (1978) reported ring necrosis in the cv. Brown Beauty and progeny of crosses between a few other cultivars and susceptible cultivars. Xiang-Sheng and Deverall (1989b) studied the ring reaction in the cv. Redlands Greenleaf C and labelled it a slightly incompatible reaction. In this study the ring reaction was found in the following cultivars, or after crosses with them: Carioca 80, Epicure, KW 780, Mkuze (A 286), Mountain White Half Runner, PI 165426, PVA 2280 and Teebus. It seems as if the ring reaction is uncommon but not very rare.

The ring reaction of Epicure and PVA 2280 was conditioned by a single dominant gene. The reaction of Carioca 80 was conditioned by two genes with

epistatic interaction between the dominant alleles after a cross between Carioca 80 and a susceptible cultivar. But a cross between a susceptible cultivar and plants that had ring symptoms derived from the Carioca 80 cross indicated in that instance the ring reaction was conditioned by a single dominant gene. Further complications arose when differences in severity of the ring reaction of plants of the F_3 of an Epicure cross were noted. Perhaps modifying genes can condition a less severe reaction.

Xiang-Sheng and Deverall (1989b) worked with a necrotic ring reaction in Redlands Greenleaf C and compared it to a susceptible reaction in Epicure. No ring necrosis was found in Redlands Greenleaf C or after a cross between it and a susceptible cultivar in this study. Ballantyne (1978) reported the ring reaction of Brown Beauty and Redlands Greenleaf B to be conditioned by the gene Ur-C. She also reported the presence of this gene in Redlands Greenleaf C. Brown Beauty was fully susceptible to all single pustule isolates it was tested with. Hence the ring necrosis found in this study is unlikely to be that conditioned by Ur-C.

A cross between Epicure and PI 165426 gave no susceptible progeny and the gene conditioning ring necrosis in both cultivars is probably the same. A cross between ring symptom-bearing plants of RC 53 (an Epicure cross) and NC 16 (a Carioca 80 cross) also gave no susceptible segregants. Presumably the ring reaction in these cultivars, Carioca 80, Epicure and PI 165426, is conditioned by the same gene.

Xiang-Sheng and Deverall (1989a) compared a resistance reaction of Epicure to a susceptible reaction of Redlands Greenleaf C. In a co-infection of a virulent and non-virulent race on Epicure, the susceptible and resistant reactions were modified by the proximity (<1 mm) of the two. With the resistant reaction, instead of a necrotic lesion, an incomplete necrotic ring formed on the side of the susceptible reaction. The susceptible reaction gave a lower infection type. Ballantyne (1978) reported the resistance of Epicure as governed by the Ur-Epi gene and presumably this conditions the ring necrosis found in this study.

The reaction of Epicure and its crosses varies from a large necrotic lesion forming three to four dpi to near normal pustules with ring necrosis forming

only at pustule eruption. The range of response may suggest a borderline ability to recognize an avirulent race, which is strongly influenced in some cases by environmental factors such as temperature. Full recognition response would presumably lead to pinpoint lesions or pinpoint uredia. The gene conditioning ring necrosis (Ur-epi or Ur-C) should be isolated in a line with no other resistance genes. This line should then be tested with as many races as possible to examine the range of responses.

Alternatively, the range of reactions is the full response of a resistance gene, but under the influence of environmental factors. Similar necrotic ring reactions have also been obtained after inoculating beans with alfalfa mosaic, tobacco mosaic (TMV), tobacco necrosis and tobacco ring spot viruses with prior rust infection. Bean plants would normally respond with a non-invasive local necrotic lesion (Yarwood, 1951). With TMV, the greater the period between the inoculation of rust and of the virus, the greater the ring formation; the reaction was also temperature-sensitive (Wilson, 1958). Yarwood (1977) also found that inoculation of cucumber downy mildew (Pseudoperonospora cubensis (Berk and Curt.)) with a prior infection of rust gave a necrotic ring reaction around isolated rust pustules. In addition, he found that rust predisposed beans to infection by powdery mildew (Sphaerotheca fuliginea (Schlecht.) Poll.). No necrosis was observed, but with widely spread rust pustules, the mildew extended no more than 1 mm beyond the pustule (Yarwood, 1965). The mildews are normally unable to infect beans and in all cases, including the virus infections, the rust infection was debilitated.

Mendgen (1978) found that the infection sequence of Epicure, which led to a large necrotic lesion, was identical to a susceptible reaction up to 28 hours post inoculation (duration of the experiment). In addition, ring necrosis occurred in a band at a distinct distance from the pustule, suggesting a mechanism that induces susceptibility. Xiang-Sheng and Deverall (1989b) reported that the ring necrosis occurred ahead of the fungal mycelium. In the present study, a greater spread of necrosis occurred where a ring lesion abutted a vein. Development of a lesion was not static and further slight development occurred at the outer periphery. This suggests an elicitor was formed by the fungal-host interaction and transmitted a short distance. With the TMV-infection, the rust infection

induced a state of susceptibility near a pustule and a resistance mechanism responded similarly, if not identically, to that induced in the rust-conditioned ring reaction of Epicure, away from the pustule.

Xiang-Sheng and Deverall (1989b) found that, by four to five days post inoculation, ultrastructural differences were visible in plants with ring symptoms compared to fully susceptible plants. Material was found deposited against cell walls at penetration sites and the width of extra-haustorial matrices was greater around older haustoria. Greater width of the extra-haustorial matrix around older haustoria is possibly an indication of a resistance reaction rather than the cause, as younger haustoria were similar in both types of reaction. Perhaps the matrix differences should be further studied with the high-pressure freezing technique of Knauf *et al*. (1989). Li and Heath (1990) found reduced resistance with increased incidence of haustoria in beans after application of abscisic acid and gibberellic acid following a cowpea rust infection. Indole-3-acetic acid slightly increased haustorium formation but did not affect silica deposition and kinetin had no effect. It was suggested the hormone-induced responses were host-mediated. As the rust-conditioned ring necrosis is inherited as a single gene, the possibility of growth hormone-induced susceptibility seems unlikely. They may have a role in altering the reaction either in response to environmental factors and/or by modifying genes.

The ring necrosis response appears to be an intermediate response between full susceptibility and full resistance. It may be the full response of a resistance gene, or be due to incomplete recognition of an avirulent race. The ring reaction can be conditioned by one of two genes (*Ur*-C and *Ur*-Epi) depending on host/race/environmental factors, and may be altered by modifying genes. It appears that a state of induced susceptibility occurs within the fungal colony and an elicitor induces ring formation beyond the colony. Perhaps this indicates that susceptibility is an induced state and resistance would be the norm in resistance and susceptibility of plants.

5.3 Breeding for horizontal resistance to rust

The 23 cultivars used in the recurrent selection programme were basically

chosen at random regarding their levels of resistance to the single pustule isolate M1B and very highly significant differences between their resistances were found. Levels of resistance were consistent over three field trials although there was a genotype x environment interaction. A similar situation was found with the cultivar yields and a negative correlation of 0.678 between rust ratings and yield was found. There was, however, no genotype x environment interaction. Ideally, yield comparisons should have been made between rust-infected and rust-free trials. In these trials, better adaptation to other agronomic conditions may have been overlooked due to poor rust resistance, or vice versa. The cvs GLP X 1132 and 358 AFR 290, for example, had higher yields than cultivars of equivalent resistance. Simons (1975) found correlations of up to -0.69 between yield reduction and disease severity working with oat crown rust of oats.

Forty-five selections were passed onto Pannar Seed for commercial evaluation before cultivar release. Several selections do not have the desired red speckled sugar colour but were retained for their high yield. These could be used as parents in further crossing programmes. It is interesting that the pedigrees of the selections include all the parent cultivars except KID 16 (crosses with KID 16 were deliberately discontinued) and 380 HAL 10. Several cultivars would not have been included if their dismal results in the cultivar trials had been known in advance, perhaps to the detriment of the selection programme.

There is a possibility that the partial resistance against the single pustule isolate M1B is vertical as opposed to horizontal resistance. The cultivars were tested with three other SPI's. Two of these tests were conducted under glasshouse conditions not so favourable for infection due to the onset of cold weather. In addition, an SPI used in the glasshouse trials was very closely related to M1B. The cultivars did, however, appear to have the same relative levels of infection. The third test was conducted in the field and problems with the source of infection led to the initial epidemic being uneven. By the end of the trial, however, the same relative levels of infection compared to the SPI M1B trials were noted except a few susceptible cultivars which had lower levels than expected.

Several second cross lines were planted in field trials by Mr A. Jarvie at

Greytown and Delmas and rated for rust after natural infection. Of the 33 lines planted at both localities, seven were resistant at one locality but not the other. If the partial resistance was horizontal then there should be no difference. Of the other lines, 12 were susceptible at both localities, these selections were probably retained for desirable seed characteristics. Selections with the different reactions may have poor horizontal resistance and the low levels of infection is due to the presence of vertical resistance to some of the prevailing races. At this stage it cannot be stated if the partial resistance is vertical or horizontal. It was rather unfortunate that the 1994 summer trials with the horizontal rust resistance material was decimated by hail.

Possible mechanisms of the cause of the partial resistance to the SPI M1B were examined. Differences in pustule size of the 23 cultivars in the first field trial were minimal. The response of the cultivars was negligibly affected by proximity to a susceptible cultivar. Presumably the resistance places little reliance on the reduction of inoculum potential.

Latent periods were investigated in the unifoliate leaves of the eight diallel cultivars. The highly resistant 1266 and highly susceptible 1273 had the shortest latent period and the highly susceptible PC 222-5-6-P2 the longest latent period. Differences were in the order of one to two days. Similar latent period responses in the unifoliate leaves of the eight cultivars was also found in the F_1 of crosses between the cultivars and RC 53 plants bearing ring necrosis. Increased latent period in the unifoliate leaf of PC 222-5-6-P2 is probably conditioned by dominant gene(s).

There was closer correlation between latent periods in the fourth trifoliate leaf and resistance. One of the most susceptible cultivars (PC 222-5-6-P2) and one of the most resistant (1266) cultivars had the shortest and longest latent periods, respectively. This is a reverse of the latent period trend in the unifoliate leaf. KID 16 (as susceptible as PC 222-5-6-P2) and 259 AND 621 (as resistant as 1266), however, had similar latent periods of an intermediate length. Increased latent periods may be a characteristic of the true resistance mechanism or an additional resistance mechanism. The latent periods of 1273 and 1266 were similar up to the second trifoliate stage and a difference was only noted by the fourth trifoliate stage. It

is unlikely that latent period would account for the large differences in resistance between the two, observed as early as 39 days after planting. The plants had just set pods at the time of inoculation in the fourth-trifoliate-leaf test. Perhaps the difference in latent periods between 1266 and 1273 is due to differences brought on by flowering and/or pod set.

The number of adaxial and abaxial acicular leaf hairs of trifoliate leaves was examined. Adaxial leaf hair numbers on the first trifoliate leaf of the eight diallel cultivars were highly variable and there were significant differences. These differences were not correlated with resistance. Abaxial leaf hairs were found in all cultivars. In some cultivars the leaf hairs were only on the veins, these were generally more susceptible to the SPI M1B. The cultivars with inter-veinal hairs included the most resistant cultivars, but there were important exceptions. The highly susceptible cvs 1271, 1273 and AFR 241 also had numerous inter-veinal abaxial hairs. PC 235-4-E3 was found to have a few inter-veinal leaf hairs on the first trifoliate, but by the third trifoliate it had a much higher number of leaf hairs. PC 223-4-1-D2 was glabrous on the first and third trifoliate leaves but had a similar level of resistance to PC 235-4-E3.

A closer examination of the abaxial leaf hairs on the first trifoliate leaves indicated that the highly susceptible 1273 was found to have an equal number or more than the highly resistant 1266. It appears unlikely that abaxial leaf hairs greatly affect the partial resistance to the SPI M1B. On the other hand ICA 15522 had *ca*. half the number of abaxial leaf hairs of 1266. Perhaps this may contribute to the moderate resistance of ICA 15522 compared to the high resistance of 1266. Perhaps size and/or alignment may affect resistance conferred by leaf hairs.

The number of adaxial and abaxial leaf hairs counted in this study were in the order of a third of the numbers counted by Shaik (1985). Mmbaga and Steadman (1992b) rated pubescence on a scale of 1 to 10, where 10 was equal to a density of greater than 1000 per cm^2 (10 mm^{-2}) which appears to be out by a factor of 10. If it is assumed that a count of 100 per mm^2 is the intended count, then their pubescent cultivars had 40 - 55 hairs per mm^2 or approximately double the number in this study. Care was taken in this study to avoid hairs along major veins where a greater density occurred and

perhaps this may account for some discrepancy.

No relationship was found between the number of adaxial stomata and partial resistance to the SPI M1B. A cursory examination of the abaxial stomata also indicated no relationship but further examination is required. No attempt was made to look for differences in stomatal structures such as the guard cell lip. Differences in stomatal structure between cultivars may provide a better thigmotropic stimulus for appressorium formation in some cultivars than in others.

It would appear that the numbers of epidermal structures do not affect the degree of partial resistance to the SPI M1B. If one assumes that there are no major differences in surface topography, then the main mechanism of the partial resistance to the SPI M1B resides in the process of infection from appressorium formation to pustule formation. It does not inhibit the formation of pustules regarding size or latent period. Hence the resistance probably acts via early or late colony abortion. Quantitative inoculation of plants and counting of pustules will show if there are differences in ability to form an infection. Microscopic investigation will indicate if there is early or late colony abortion. If the partial resistance proves to be horizontal, it would be interesting to see if there are cytological or histological differences when compared to vertical resistance.

The lack of resistance conferred by leaf hairs conflicts with the work of several other workers. Perhaps the pubescent cultivars that are highly susceptible have an additional mechanism that over-rides the resistance provided by the leaf hairs, but this seems highly unlikely. The other possibility is that leaf hairs themselves do not confer resistance *per se* but are linked to another mechanism that does. For the highly susceptible cultivars that are pubescent this linkage has been broken. It is unlikely that by mere chance that, with a few exceptions, the highly resistant cultivars are pubescent and the highly susceptible cultivars are glabrous. Mmbaga and Steadman (1991b) found a race-nonspecific "lower cellular susceptibility" (presumably there is less ramification of rust mycelium) of the trifoliate leaves of pubescent cultivars. Interestingly, Chérif *et al*. (1992) found that when cucumber plants were grown in silicon-enriched media, silicon accumulated in cells surrounding leaf hair bases (silicon has been

implicated in some cases of host resistance). The transport of the silicon did not appear to be an active process, rather that it was carried along in the transpiration stream. Another possibility is a pleiotropic action of the gene(s) for pubescence that modify other structures. Perhaps the presence of leaf hairs modifies stomata, making them less identifiable for appressorium formation. With the pubescent and susceptible cultivars other genes over-ride the modification caused by the leaf hairs. The diallel results do not appear to support such a possibility, but further work would be required. Rubiales and Niks (1992) found a reduction in appressorium formation after inoculation of several lines of Hordeum chilense (Roem. & Schult.) with several species of cereal rust fungi. This avoidance mechanism was due to poor stomatal recognition and unrelated to number or length of hairs or stomatal frequency or size. They suggested that an ultrastructural examination of stomata was required.

Mmbaga and Steadman (1990 and 1991a) found a reduction in uredial size in pubescent cultivars supposedly due to a delay in infection. This delay would cause a reduced uredial size only if the weakened infection structure was unable to use the host's nutrition to the same extent as a normal infection. The initial growth may be slowed sufficiently to allow some degree of active host resistance to occur, perhaps the initiation of a state of susceptibility is interfered with. If it is assumed that infection can only occur during one "night" (darkness and humidity favourable for infection), as is widely believed to be the case (F.H.J.Rijkenberg, Pers. Comm., 1992), is the difference in timing of infection between the two significant? As noted above, no differences in pustule size in a field trial were noted, but a delay in infection may aid colony abortion. Recent reports by Mmbaga and Steadman (1992b and 1992c) indicated the existence of a glabrous line that has the same type of resistance as pubescent types. They suggested the resistance of the pubescent cultivars was characterized by having low sporulating capacity, reduced uredial size and density and low infection rate. In the field trials reported here, the effects of low sporulation capacity and reduced uredial size (both contribute to reduced inoculum potential) were negated by the proximity of susceptible cultivars to resistant cultivars. In addition, to their own work, Mmbaga and Steadman (1992b) reported a personal communication from Zang (sic) and J.R.Steadman. The latter two studied the relationship of leaf hair density and length,

stomatal density and stomatal ridge height to low infection rate in four cultivars in the glasshouse. Significant differences in leaf hair density and length and density of abaxial stomata were observed, but these were not correlated with infection rate.

Wilcoxson (1981) listed five major post-infection characteristics of slow-rusting of the cereals. Of these, latent period and pustule size do not have a bearing on the partial resistance investigated in the present study. In addition, duration of sporulation and mass of spores produced probably contribute little to the initial resistance mechanism. The remaining factor of Wilcoxson was infection frequency. The nature of the inheritance of the partial resistance, however, is in closer agreement with that of the cereals.

The partial resistance of the eight diallel cultivars in this study was partially dominant and mainly due to general combining ability with specific combining ability also being significant. Reciprocal effects were not significant over the two seasons or in interaction with seasons. There were significant interactions between GCA and SCA with seasons. The significant interactions with seasons indicate a genotype x environment interaction.

Yield results showed similar trends, but with increased importance of SCA and reciprocal effects. Reciprocal effects over both seasons were not significant, but in interaction with seasons it was significant, showing the possible importance of maternal effects on yield. The effect of SCA on the inheritance of yield improvement during a rust epidemic was greater than that of resistance over seasons, but in interaction with seasons it was not significant.

The greater proportion of inheritance of resistance and yield over both seasons and in interaction with seasons was due to GCA indicating the importance of additive gene action. Hence the high level of narrow sense heritability estimates of 0.899 ± 0.056 for the 1990/91 trial and 0.603 ± 0.065 for the 1992 trial for rust resistance. Weather conditions were hotter and drier in the second trial, reducing the heritability estimate.

The importance of GCA and high levels of heritability are often

characteristic of slow-rusting of other crops. Caldwell et al. (1957) found certain slow-rusting wheat cultivars had high glasshouse susceptibilities to leaf rust. The cultivars were bred from a combination of resistance sources and they suggested that there was interaction between resistance genes or there was an additive effect. Das et al. (1992) reported the slow-rusting of spring wheat with leaf rust was predominantly due to additive genetic effects with additive x additive effects also playing a role. Estimates of narrow sense heritability varied from 45 - 92 %. Gavinlertvatana and Wilcoxson (1978) obtained similar estimates and suggested 3 - 21 genes were involved. Broers and Jacobs (1989) found wheat leaf rust partial resistance to be inherited recessively by the additive interaction of two to three genes, depending on the cultivar, with the possibility of transgressive segregation.

Chen and Line (1991a and 1991b) studied adult-plant resistance of wheat to stripe rust. They found the resistance to be partially dominant or recessive with additive gene action conditioning the major part of the resistance. Dominance and non-allelic interactions were also significant in some cases. The number of genes involved was two to three genes with the genes differing between cultivars and those conferring seedling resistance. Estimates of broad- and narrow-sense heritabilities varied from 75.6 - 99.8 %. Skovmand et al. (1978) in studying slow-stem-rusting of wheat found inheritance of resistance to be predominantly due to additive effects with some epistasis. Transgressive segregation occurred in all crosses, sometimes favouring resistance and other times not. Estimates of narrow-sense heritability of about 80 % were obtained and the number of segregating gene pairs was between 2 and 12 pairs.

Luke et al. (1975) found the horizontal resistance of oats to crown rust to be controlled by a few genes with slight partial dominance for susceptibility. Wilkins (1975) reported the inheritance of slow-rusting of ryegrass with Puccinia coronata f.sp. lolii (Cord.) to be largely additive and without dominance or epistasis. Narrow-sense heritability was calculated to be 22 %. Sharma (1977) suggested that additive effects were more important than non-additive effects, although both were significant, in the inheritance of slow-rusting of maize infected with P.sorghi. Epistasis was absent and transgressive segregation was common. Kim and

Brewbaker (1977) also found similar inheritance patterns for slow-rusting of maize with P.sorghi and a narrow-sense heritability estimate of 47 %.

In their review of rust resistance in groundnut, Subrahmanyam et al. (1993) suggested the resistance was characterized by increased latent periods and reductions in infection frequency, pustule size, spore production and viability. The inheritance was mostly due to additive, additive x additive and additive x dominance gene effects. In addition, it was suggested that the resistance does not fit typical vertical or horizontal patterns and was postulated as an intermediate type. Perhaps, the resistance observed is a combination of partial vertical and horizontal resistances.

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APPENDIX 1: Cultivars used in this project

Cultivar/line	Size[1]	Colour[2]	Growth habit[3]	Origin[4]	Use[5]
Ab 136	*[6]	*	*	Mexico	AnD
AFR 241	L	1,6	I	*	HRR
Aiguille Vert	S	3	*	France	AnD
AND 340	L	6,5	I	*	HRR
AND 366	L	*	*	*	HRR
ASC 4	S	*	IV	*	AsR
Aurora	S	1	IV	*	RD
Actopan x Sanilac 37	S	1	IV	*	RD
BAT 795	S	8	IV	*	AsR
BAT 1427	L	6,7	I	*	RR
Bonus	L	1,6	III	RSA	SC
Broadacres	M	2,4	IV	*	RN
Brown Beauty	L	4	I	USA	RD
Californian SW 643	S	1	I	USA	RD
Canadian Wonder	L	7	I	Canada	HBD
Carioca 80	S	2,4	III	*	RR/AlR
Coco a la Creme	L	1	*	France	AnD
Compuesto NC	S	8	II	*	RD
Cornell 49242	S	8	II	USA	AnD/AnR
COS 10	L	1,6	I	*	HRR
Early Galatin	L	1	I	Australia	RD
Ecuador 299	S	5	III	Ecuador	RD
Edmund	S	1	II	UK	HBD
Epicure	M	4	IV	Holland	RN/HRR
Evolutie	M	1	*	Holland	AnD
Golden Gate Wax	L	4	IV	USA	RD/HRR
GLP X 1125	M	*	*	Kenya	HRR
GLP X 1132	M	2,4	IV	Kenya	HRR
GN No. 27 Sel. 1	M	1	IV	USA	CBR/HBR
GN Tara	M	1	IV	USA	CBR/HBR
ICA 15116	L	6,5	I	Colombia	HRR
ICA 15521	L	6,5	I	Colombia	HRR
ICA 15522	L	6,1	I	Colombia	HRR
ICA 15489	L	*	*	Colombia	HRR
Kentucky Wonder 765	M	4	IV	USA	RD
Kentucky Wonder 780	M	1	IV	USA	RD
Kentucky Wonder 814	M	4	IV	USA	RD
KID 16	L	6	I	USA	HRR
KID 20	L	6	I	USA	HRR
Mexico 235	M	5	IV	Mexico	RD
Mexico 309	M	8	III	Mexico	RD
Mexique 222	*	*	*	Mexico	AnD
Michelite	S	1	*	USA	AnD
Michigan DRK	L	6	I	USA	AnD
Mkuze (A286)	S	2,4	III	*	RR/RN
Mountain WHR	M	1	IV	USA	RD
Natal Speckled Sugar	M	1,6	I	RSA	RD(L)
Nep 2	S	1	II	*	RD
Olathe	M	2,4	III	USA	RD
PAI 6	S	6,1	III	*	AsR/AlR
PAI 127	S	6,1	III	*	AsR/AlR

PC 222-5-6P2	L	1,6	I	RSA	HRR
PC 223-4-1-D1	L	1,6	III	RSA	HRR
PC 232-1-D2	L	1,6	III	RSA	SS
PC 235-4-E1	L	1,6	III	RSA	SS
PC 235-4-E3	L	1,6	III	RSA	HRR
PC 252-D2	L	1,6	III	RSA	SS
PC 256-D1 (Kranskop)	L	1,6	II	RSA	SS
PC 349-1	L	3	I	RSA	HRR
PC 351-19	L	3	I	RSA	HRR
Perry Marrow	L	1	*	USA	AnD
PI 165426	S	4	IV	Mexico	AnD/RN
PI 167399	*	*	*	Colombia	AnD
Pinto 650	M	2,4	III	USA	RD
PVA 2280	L	6,5	I	*	HRR
PVA 992	L	5,6	I	*	HRR
Red Mexican UI 3	M	6	I	*	HBD
Redlands Greenleaf C	L	4	I	Australia	RD
Redlands Pioneer	L	4	I	Australia	RD
Sanilac	S	1	*	USA	AnD
Teebus	S	1	I	RSA	RD(L)/HRR
Tendergreen	L	2,7	I	*	HBD
Umlazi	L	1,6	I	RSA	HRR
United States No. 3	M	1	IV	USA	RD
VRA 81018	S	8	IV	*	AnR
XAN 159	M	2,4	I	*	CBR
ZAA 49	L	7	I	*	RR
1266	L	1,8	I	*	HRR
1271	L	1,6	I	*	HRR
1273	L	1,6	I	*	HRR
1274	L	1,6	I	*	HRR
1312	L	1,6	IV	*	HRR
259 AND 621	L	5,6	I	*	HRR
269 AND 631	M	5,6	IV	*	HRR
312 AND 689	L	5,6	I	*	HRR
339 AFR 290	L	7,1	I	*	HRR
358 AFR 290	L	5	I	*	HRR
380 HAL 10	L	6,7	I	*	HRR
50151	S	8	I	Costa Rica	RD

1: Seed sizes are small (< 25 g/100 seeds), medium (>25 g, < 40 g) and large (>40 g).

2. For seed colour, a single number indicates a monochrome, a second number indicates the colour of the mottle or stripe on the background colour (first number).

1 = white 2 = cream 3 = yellow 4 = brown

5 = pink 6 = red 7 = purple 8 = black

3: Growth habits are:

I = determinate bush II = Indeterminate bush

III = indeterminate prostrate viney bush
IV = indeterminate with climbing habit

(The first three scales are based on Van Schoonhoven and Pastor-Corrales, 1987)

4: Countries of origin.

5: The cultivars were primarily used for the following purposes:
AlR = Source of angular leaf spot resistance
AnD = Anthracnose differential cultivar
AnR = Source of anthracnose resistance
AsR = Source of Ascochyta blight resistance
CBR = Source of common blight resistance
HBD = Halo blight differential cultivar
HBR = Source of halo blight resistance
HRR = Horizontal rust resistance programme
RD = Rust differential cultivar
RD(L) = Rust differential cultivar included for local interest
RN = Study of ring necrosis
RR = Source of rust resistance
SC = Susceptible to the seven diseases covered in this project
SS = Source of speckled sugar seed characteristics and BCMV resistance

6: * = information not recorded

The following crosses were made during this study, in addition to first (RC), second (SC) and third (TC) rust crosses and diallel crosses (DC).

Cross No.	Parents
NC 1	GN No. 27 Selection 1 x PC 252-D2
NC 2	GN No. 27 Selection 1 x PC 256-D1
NC 3	GN Tara x PC 252-D2
NC 4	GN Tara x PC 256-D1
NC 5	GN Tara x Charlevoix
NC 6	Charlevoix x GN Tara
NC 7	NC 1 x NC 4
NC 8	NC 2 x NC 3
NC 9	Cornell 49242 x BAT 1427
NC 10	Cornell 49242 x ZAA 49
NC 11	PC 232-1-D2 x PAI 6
NC 12	PC 232-1-D2 x PAI 127
NC 13	PC 232-1-D2 x Carioca 80
NC 14	PC 235-4-E1 x PAI 6
NC 15	PC 235-4-E1 x PAI 127
NC 16	PC 235-4-E1 x Carioca 80
NC 17	NC 9 x NC 10
NC 18	Broadacres x Mkuze
NC 19	NC 11 x NC 15 & NC 14 x NC 12
NC 20	NC 13 x NC 15 & NC 16 x NC 12
NC 21	PVA 2280 x PC 235-4-E3
NC 22	NC 7 & NC 8 x NC 17
NC 23	NC 19 x NC 20
NC 24	Mkuze x Carioca 80
NC 25	NC 18 ring[1] x NC 16 ring
NC 26	Carioca 80 x PAI 127
NC 27	NC 16 ring x PC 222-5-6-P2
NC 28	Epicure x Redlands Greenleaf C
NC 29	RC 53 ring x PC 222-5-6-P2
NC 30	RC 53 ring x 1266
NC 31	RC 53 ring x 1273
NC 32	RC 53 ring x ICA 15522
NC 33	RC 53 ring x KID 16

NC 34	RC 53 ring x GLP X 1132
NC 35	RC 53 ring x 259 AND 621
NC 36	RC 53 ring x 269 AND 631
NC 37	Epicure x PI 165426
NC 38	NC 22 x NC 23
NC 39	NC 19 x BAT 795
NC 40	RC 53 mild ring x PC 222-5-6-P2
NC 41	RC 53 ring x PC 222-5-6-P2
NC 42	RC 53 ring x NC 16 ring

Also of interest for work with the slightly incompatible reaction.

RC 10	Epicure x PC 222-5-6-P2
RC 53	Epicure x PC 235-4-E3
RC 73	Epicure x 1266

1 = Plants bearing ring symptoms after infection with rust. Mild ring symptoms developed at pustule eruption otherwise ring symptoms developed three to four dpi.

APPENDIX 3: Details of field trials

Ukulinga is situated at 29° 40′ south and 30° 24′ east and an altitude of 726 m above sea level. The average rainfall is 705.5 mm per year and average daily temperatures vary from 18 to 23°C. The weather details experienced during the field trials are given in Figure 14. The soil is classified as a Mispah form of the Mispah series with a depth of class 3. The trial was on slope of a gradient of 1:20.

The Greytown site is at 29° 02′ south and 30° 37′ east and an altitude of 1110 m. The soil is classified as a Hutton form and there is an average rainfall of 867.9 mm per year.

In the breeding trial, the crosses were planted in numerical order in two blocks between three blocks of the parent cultivars with the following randomized order:

Block 1	Block 2	Block 3
KID 20	380 HAL 10	259 AND 621
ICA 15116	AFR 241	339 AFR 290
312 AND 689	Teebus	AND 340
COS 10	AND 340	PC 222-5-6-P2
AFR 241	ICA 15522	ICA 15521
380 HAL 10	312 AND 689	KID 16
PVA 992	1271	COS 10
PC 235-4-E3	KID 20	1266
PC 223-4-1-D2	PC 223-4-1-D2	PC 223-4-1-D2
1266	COS 10	AFR 241
PC 222-5-6-P2	1273	269 AND 631
1273	259 AND 621	312 AND 689
1312	358 AFR 290	ICA 15522
339 AFR 290	ICA 15116	1271
259 AND 621	339 AFR 290	358 AFR 290
358 AFR 290	1312	GLP X 1132
1271	1266	380 HAL 10
KID 16	PVA 992	Teebus
ICA 15521	PC 235-4-E3	PC 235-4-E3

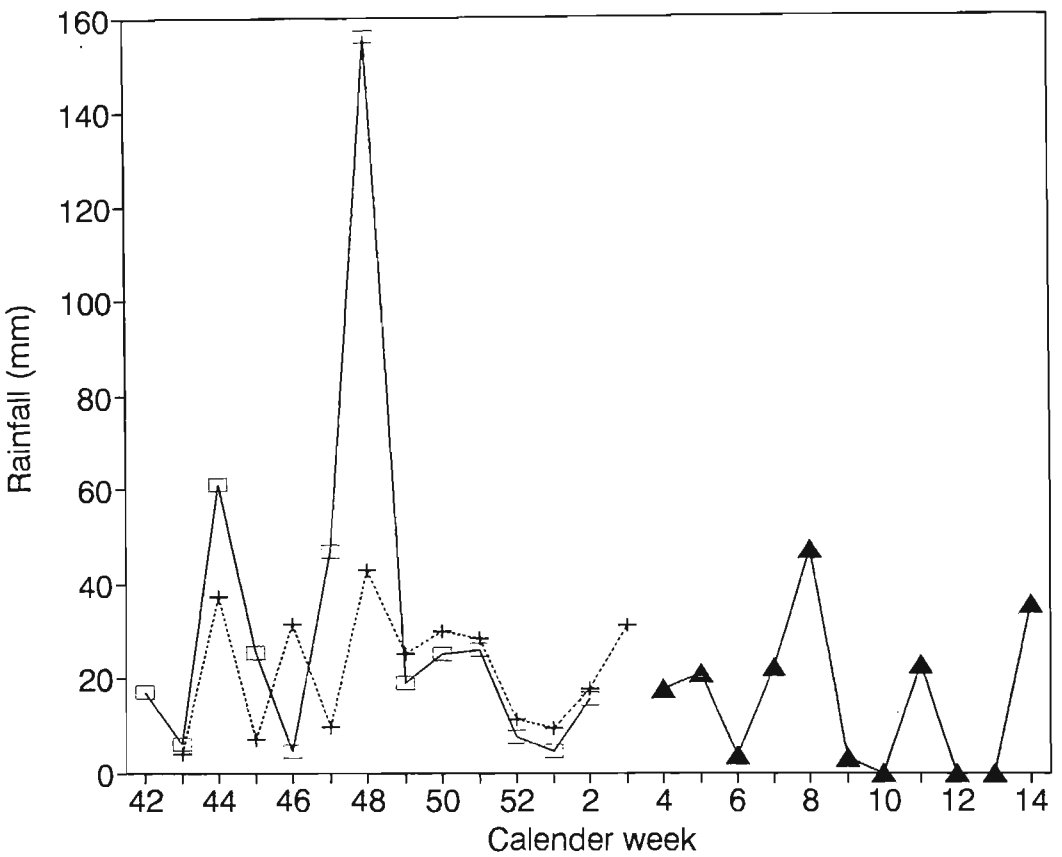
GLP X 1132	KID 16	1312
ICA 15522	ICA 15521	PVA 992
Teebus	GLP X 1132	ICA 15116
AND 340	PC 222-5-6-P2	1273
269 AND 631	269 AND 631	KID 20

In the third trial, Umlazi was planted at the 25th, 22nd and 3rd positions, respectively.

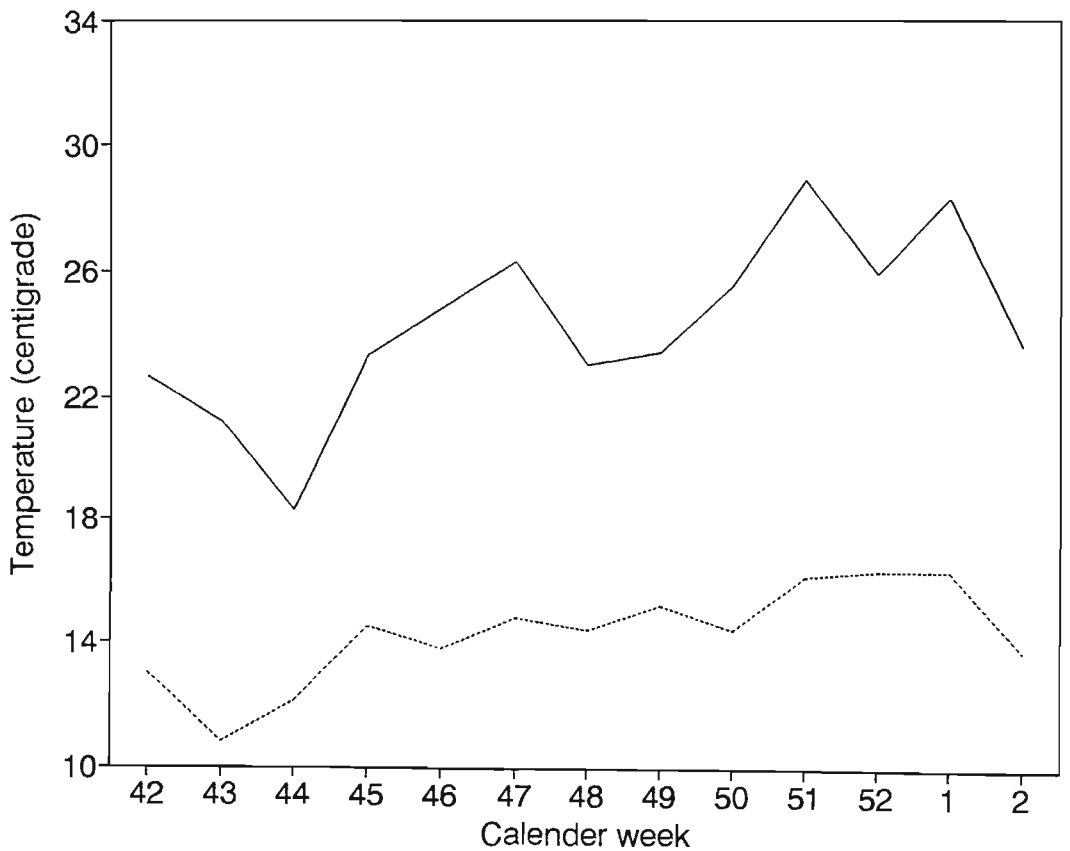
In the diallel test trial, crosses were planted in two blocks, with one replication per block, in a randomized order. The two blocks were planted between three blocks of the parent cultivars in the following randomized order:

Block 1	Block 2	Block 3
1273	269 AND 631	KID 16
259 AND 621	1273	PC 222-5-6-P2
269 AND 631	ICA 15522	1266
PC 222-5-6-P2	GLP X 1132	259 AND 621
KID 16	KID 16	1273
1266	1266	GLP X 1132
ICA 15522	259 AND 621	269 AND 631
GLP X 1132	PC 222-5-6-P2	ICA 15522

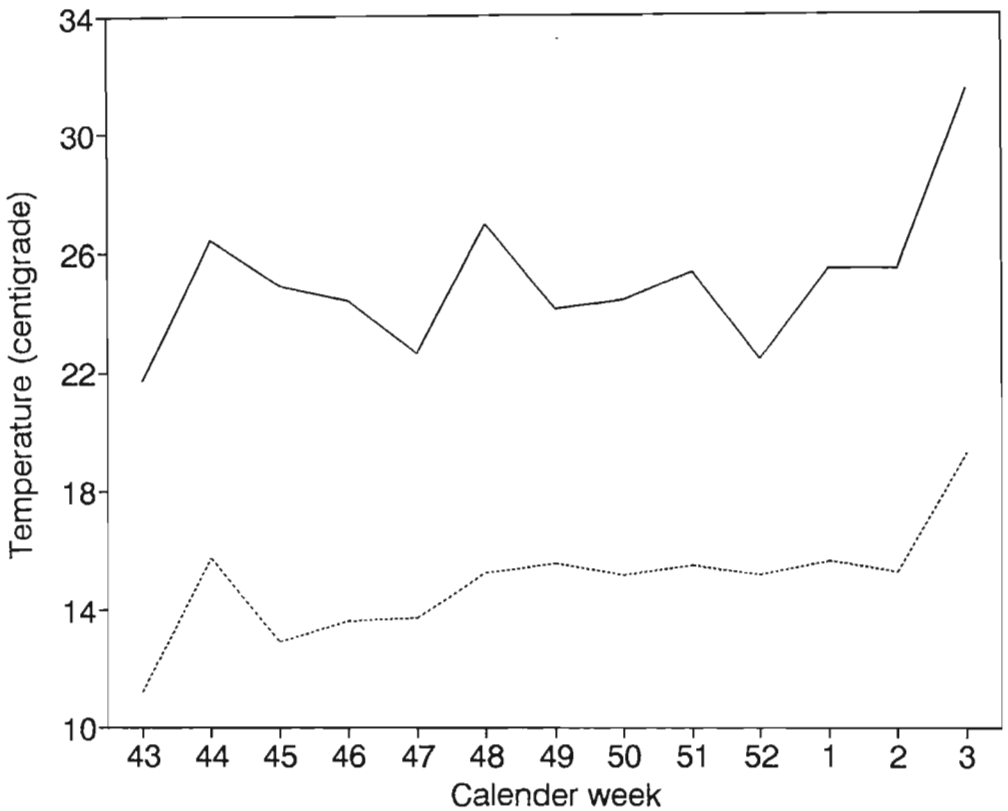
Figure 9. Weather data for the field trials conducted at Ukulinga.
(a). The rainfall for the trials. Open squares are the 1989/90 data, pluses and dashed line are the 1990/91 data and closed triangles are the 1992 data.
(b). Average daily maximum (solid line) and minimum temperatures (dotted line) of the 1989/90 trial.
(c). Average daily maximum (solid line) and minimum (dotted line) temperatures of the 1990/91 trial.
(d). Average daily maximum (solid line) and minimum temperatures (dotted line) of the 1992 trial.
The data is given for the calender week (Monday to Sunday) including the planting of the test rows until the first harvest.


Rainfall (mm)
160
140
120
100
80
60
40
20
0
42
44
46
48
50
52
2
4
6
8
10
12
14
Calender week

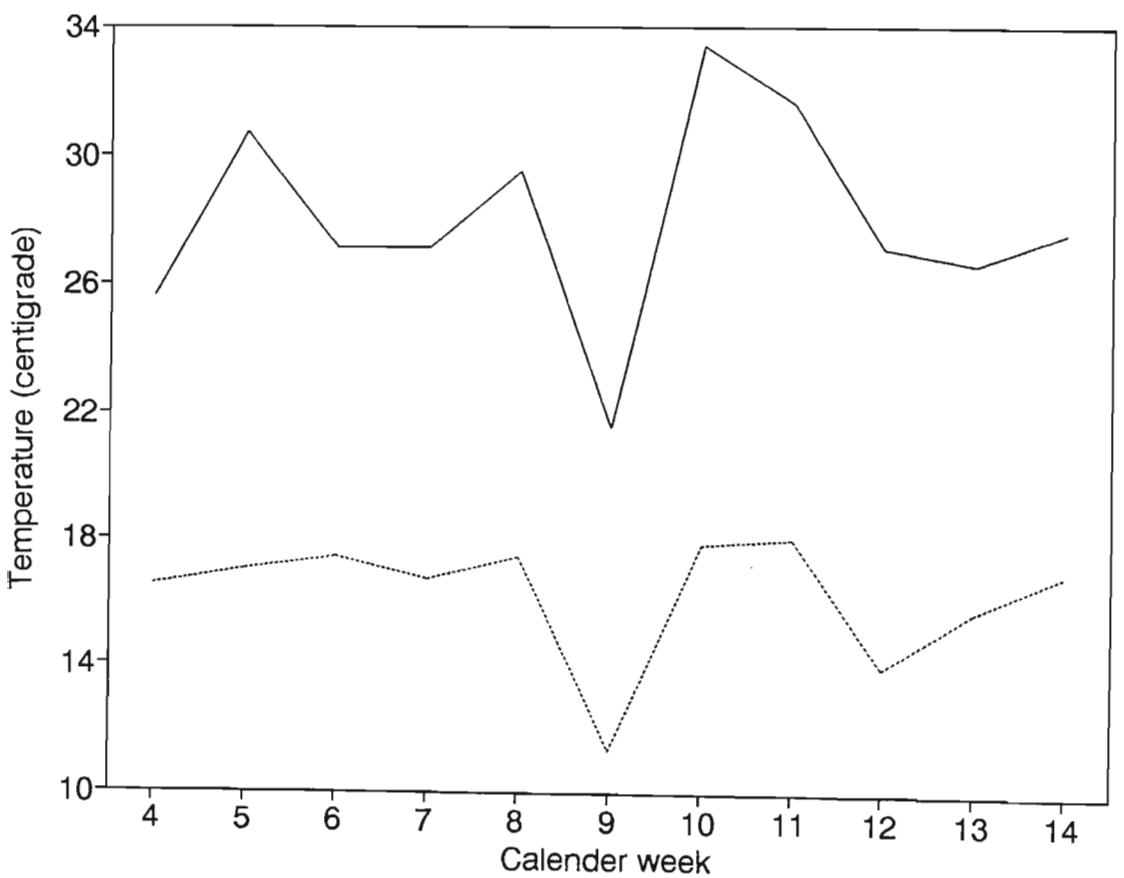
(a).


Temperature (centigrade)
34
30
26
22
18
14
10
42
43
44
45
46
47
48
49
50
51
52
1
2
Calender week

(b).


34
30
26
22
18
14
10
Temperature (centigrade)
43 44 45 46 47 48 49 50 51 52 1 2 3
Calender week

(c).


34
30
26
22
18
14
10
Temperature (centigrade)
4 5 6 7 8 9 10 11 12 13 14
Calender week

(d).

APPENDIX 4: Pedigrees of the TC lines passed on to Pannar Seed

TC Line	Pedigree
TC 6	((PC 222-5-6-P2 x AFR 241) x (1266 x 1312)) x ((ICA 15521 x ICA 15522) x (1312 x AND 340))
TC 8	((PC 222-5-6-P2 x AND 340) x (1266 x 1312)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 13	((PC 222-5-6-P2 x 339 AFR 290) x (1312 x ICA 15522)) x ((1271 x COS 10) x (1312 x ICA 15522))
TC 19	((PC 222-5-6-P2 x 339 AFR 290) x (1312 x ICA 15522)) x ((GLP X 1132 x 259 AND 621) x (1266 x ICA 15522))
TC 23	((PC 223-4-1-D2 x ICA 15522) x (1266 x 1312)) x ((1266 x 269 AND 631) x (1312 x AND 340))
TC 25	((PC 235-4-E3 x ICA 15521) x (1312 x ICA 15522)) x ((1273 x ICA 15116) x (1312 x AND 340))
TC 29	((1266 x 1312) x (AND 340 x 259 AND 621)) x ((ICA 15116 x 259 AND 621) x (1266 x AND 340))
TC 44	((1266 x KID 20) x (1312 x ICA 15522)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 45	((1266 x KID 20) x (1312 x AND 340)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 46	((1266 x AND 340) x (1312 x ICA 15522)) x ((1271 x COS 10) x (1312 x ICA 15522))
TC 51	((1266 x GLP X 1132) x (1312 x 259 AND 621)) x ((1266 x ICA 15116) x (1312 x AND 340))
TC 60	((1266 x 269 AND 631) x (1312 x AND 340)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 61	((1266 x 269 AND 631) x (1312 x AND 340)) x ((ICA 15116 x 259 AND 621) x (1266 x AND 340))
TC 62	((1266 x 269 AND 631) x (1312 x AND 340)) x ((ICA 15521 x ICA 15522) x (1312 x AND 340))
TC 75	((1271 x COS 10) x (1312 x ICA 15522)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 76	((1271 x COS 10) x (1312 x ICA 15522)) x ((ICA 15521 x ICA 15522) x (1312 x AND 340))
TC 81	((1271 x AND 340) x (AND 340 x 259 AND 621)) x ((1266 x ICA 15116) x (1312 x AND 340))
TC 83	((1271 x 339 AFR 290) x (1266 x ICA 15522)) x ((ICA 15116 x COS 10) x (1266 x 1312))
TC 84	((1271 x 339 AFR 290) x (1266 x AND 340)) x ((1273 x ICA 15116) x (1312 x AND 340))
TC 91	((1273 x 1312) x (1266 x ICA 15522)) x ((1266 x 269 AND 631) x (1312 x AND 340))
TC 92	((1273 x ICA 15116) x (1266 x 1312)) x ((1266 x AND 340) x (1312 x ICA 15522)) [2 selections]
TC 93	((1273 x ICA 15116) x (1266 x 1312)) x ((PC 222-5-6-P2 x 339 AFR 290) x (1312 x ICA 15522)) [2 selections]
TC 98	((1273 x GLP X 1132) x (1266 x AND 340)) x ((1271 x COS 10) x (1312 x ICA 15522))
TC 101	((1312 x 339 AFR 290) x (1266 x AND 340)) x ((1266 x KID 20) x (1312 x ICA 15522))
TC 109	((ICA 15116 x COS 10) x (1312 x 259 AND 621)) x ((ICA 15116 x 259 AND 621) x (1266 x AND 340))
TC 114	((ICA 15116 x 259 AND 621) x (1312 x ICA 15522)) x ((ICA 15116 x COS 10) x (1266 x 1312))

TC 115 ((ICA 15116 x 259 AND 621) x (1312 x ICA 15522)) x
((1266 x 269 AND 631) x (1312 x AND 340))

TC 116 ((ICA 15116 x 312 AND 689) x (1312 x ICA 15522)) x
((1266 x AND 340) x (1312 x ICA 15522))

TC 122 ((ICA 15521 x 259 AND 621) x (1266 x AND 340)) x
((1266 x 269 AND 631) x (1312 x AND 340))

TC 123 ((ICA 15521 x 259 AND 621) x (AND 340 x 259 AND 621)) x
((1266 x 312 AND 689) x (1312 x AND 340))

TC 128 ((ICA 15522 x COS 10) x (1312 x AND 340)) x [2 selections]
((1273 x ICA 15116) x (1312 x AND 340))

TC 129 ((ICA 15522 x COS 10) x (1312 x AND 340)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 132 ((ICA 15522 x 358 AFR 290) x (1266 x 1312)) x
((1266 x 269 AND 631) x (1312 x AND 340))

TC 139 ((PVA 992 x 312 AND 689) x (1312 x AND 340)) x
((1266 x 312 AND 689) x (1312 x AND 340))

TC 140 ((PVA 992 x 312 AND 689) x (1312 x AND 340)) x
((ICA 15116 x COS 10) x (1266 x 1312))

TC 141 ((KID 20 x AFR 241) x (1266 x ICA 15522)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 142 ((KID 20 x AFR 241) x (1266 x ICA 15522)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 152 ((AND 340 x GLP X 1132) x (1266 x 1312)) x
((PVA 992 x 259 AND 621) x (1266 x AND 340))

TC 158 ((AND 340 x 259 AND 621) x (1266 x ICA 15522)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 167 ((339 AFR 290 x 312 AND 689) x (1312 x AND 340)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 168 ((358 AFR 290 x 259 AND 621) x (1312 x AND 340)) x
((1271 x COS 10) x (1312 x ICA 15522))

TC 169 ((358 AFR 290 x 312 AND 689) x (1266 x AND 340)) x
((1266 x 312 AND 689) x (1312 x AND 340))

APPENDIX 5: Results of the second and third diallel field trials.

Results are the means of eight plants unless plants were lost to hoeing, wind damage, etc., insufficient seed was available for planting or plants were selfs. Ratings of % leaf area affected by rust for the 1990/91 trial were done on the 18th (1) and 27th (2) of December and 3rd (3) and 10th (4) of December and for the 1992 trial on 17th (1), 26th (2) and 31st (3) of March and 8th (4) and 16th (5) of April.

Rep = Replication or block No. in trial Y = Yield in grammes per plant

S = Plants senescent by that date * = No data available

	Rep	1990/91 Trial					1992 Trial					
Cv./Cross		1	2	3	4	Y	1	2	3	4	5	Y
PC 222-5-6-P2	1	21	17	17	15	2	11	16	12	S	S	1
	2	14	14	16	16	2	10	14	21	S	S	1
	3	13	12	15	15	*	9	18	22	S	S	*
1266	1	2	1	1	2	15	1	1	4	S	S	9
	2	2	3	3	2	15	1	1	3	S	S	6
	3	2	2	4	4	*	1	2	4	S	S	7
1273	1	12	13	13	13	4	9	7	6	9	S	2
	2	10	12	13	21	3	8	6	10	18	S	1
	3	9	9	13	19	*	8	7	8	12	S	2
ICA 15522	1	6	10	10	8	8	3	4	6	9	S	4
	2	6	9	11	9	8	2	5	8	13	S	3
	3	6	9	12	11	*	2	3	8	8	S	4
KID 16	1	17	16	19	20	3	9	16	17	S	S	1
	2	11	16	21	25	2	4	*	*	*	*	*
	3	11	14	16	21	*	8	18	35	S	S	1
GLP X 1132	1	8	7	7	6	12	8	3	4	8	7	8
	2	7	4	5	5	15	7	5	9	10	5	2
	3	8	5	4	3	*	4	3	5	7	4	6
259 AND 621	1	1	2	2	2	16	1	1	2	S	S	6
	2	2	2	3	3	23	1	1	2	S	S	4
	3	2	2	2	3	*	1	1	3	S	S	8
269 AND 631	1	13	9	9	8	5	7	3	4	7	S	4
	2	9	7	8	9	6	5	5	6	7	S	5
	3	11	7	7	9	*	5	2	6	7	S	4
DC 1	1	5	4	5	4	18	7	4	2	S	S	10
	2	6	8	10	6	20	2	3	6	S	S	6
DC 2	1	14	15	19	20	5	10	12	11	19	S	4
	2	10	10	15	12	6	9	8	10	12	S	3

DC 3	1	12	13	15	14	13	9	7	7	S	S	8
	2	9	9	13	11	13	3	4	5	S	S	8
DC 4	1	13	14	21	18	4	14	12	17	S	S	2
	2	11	15	19	20	4	9	11	16	S	S	2
DC 5	1	11	7	8	6	16	8	6	9	12	S	4
	2	8	7	8	5	17	6	3	5	6	S	5
DC 6	1	5	3	3	6	26	5	3	4	S	S	16
	2	5	5	5	4	25	2	1	3	S	S	10
DC 7	1	11	8	9	11	10	13	5	8	10	S	10
	2	11	7	8	6	9	6	3	7	8	S	9
DC 8	1	7	4	6	8	22	4	2	2	S	S	15
	2	4	5	6	4	28	3	2	4	S	S	9
DC 9	1	5	4	6	6	20	3	3	5	S	S	7
	2	4	4	6	3	16	2	2	5	S	S	7
DC 10	1	6	7	9	10	13	2	3	5	6	S	12
	2	3	3	4	4	18	4	3	6	7	S	7
DC 11	1	8	10	10	10	16	3	2	4	S	S	8
	2	5	5	7	8	15	2	1	4	S	S	5
DC 12	1	5	3	4	3	27	5	3	4	4	6	15
	2	3	2	2	2	22	1	1	2	3	4	16
DC 13	1	4	2	3	3	29	2	2	4	S	S	10
	2	5	2	3	3	16	1	1	3	S	S	11
DC 14	1	5	4	5	4	20	2	3	6	7	4	7
	2	4	4	4	3	17	2	1	4	6	5	10
DC 15	1	15	14	15	16	6	10	9	13	S	S	3
	2	11	10	15	14	7	9	9	10	S	S	3
DC 16	1	6	5	8	6	18	4	4	4	S	S	7
	2	4	4	5	4	18	4	3	6	S	S	3
DC 17	1	9	10	11	11	7	10	8	6	10	S	11
	2	9	9	10	11	5	6	4	7	9	S	4
DC 18	1	15	14	17	20	4	10	8	14	S	S	5
	2	14	12	18	16	7	9	4	10	S	S	5
DC 19	1	10	8	9	9	14	9	5	6	7	8	9
	2	8	5	6	6	15	5	4	3	7	6	4
DC 20	1	7	7	7	7	19	2	2	4	S	S	8
	2	5	3	4	4	24	2	1	3	S	S	7
DC 21	1	11	8	10	12	6	6	4	7	9	S	7
	2	*	*	*	*	*	7	3	6	6	S	4

DC 22	1	11	11	13	11	10	9	9	8	11	S	11
	2	10	12	13	12	12	*	*	*	*	*	*
DC 23	1	7	6	8	6	12	5	4	4	8	S	11
	2	6	9	11	6	12	1	1	3	3	S	8
DC 24	1	11	9	10	10	6	6	6	9	12	S	6
	2	9	11	12	10	9	5	3	6	7	S	4
DC 25	1	9	8	12	13	9	7	7	8	S	S	9
	2	9	11	13	16	10	3	2	5	S	S	8
DC 26	1	7	4	5	4	14	5	3	5	6	5	18
	2	6	2	3	3	16	*	*	*	*	*	*
DC 27	1	2	2	3	3	16	1	2	5	S	S	10
	2	*	*	*	*	*	1	1	2	S	S	7
DC 28	1	8	8	11	9	9	5	3	4	6	6	14
	2	7	4	5	5	12	*	*	*	*	*	*
DC 29	1	12	12	14	17	5	10	8	15	S	S	4
	2	12	11	16	19	6	8	12	10	S	S	1
DC 30	1	8	9	10	11	10	6	4	5	S	S	8
	2	8	8	12	12	13	2	2	4	S	S	7
DC 31	1	11	11	17	19	5	14	10	14	S	S	4
	2	10	8	13	12	6	7	9	12	S	S	2
DC 32	1	7	10	14	13	11	5	6	8	S	S	10
	2	6	7	9	11	10	5	4	9	S	S	6
DC 33	1	10	11	13	9	12	7	5	7	S	S	8
	2	7	6	6	5	15	4	6	10	S	S	4
DC 34	1	7	6	9	10	15	1	2	4	S	S	9
	2	6	6	8	5	17	1	2	4	S	S	6
DC 35	1	9	8	12	12	8	10	4	10	10	S	9
	2	*	*	*	*	*	*	*	*	*	*	*
DC 36	1	8	5	8	5	13	11	5	7	8	S	9
	2	6	5	5	4	18	7	4	6	8	S	7
DC 37	1	6	4	4	3	18	4	3	6	9	S	12
	2	5	4	3	3	11	2	2	4	6	S	10
DC 38	1	8	7	7	10	9	12	6	4	8	8	9
	2	9	5	5	4	10	6	3	4	6	7	10
DC 39	1	5	4	4	3	14	3	4	4	7	5	9
	2	5	4	4	3	12	4	2	6	6	4	10
DC 40	1	10	8	9	9	10	6	6	8	11	S	7
	2	8	6	5	6	16	6	5	7	9	S	7

DC 41	1	2	2	3	1	22	2	1	2	3	S	22
	2	4	2	3	2	18	1	1	3	8	S	14
DC 42	1	8	6	6	7	8	10	4	4	6	5	12
	2	9	5	5	4	11	6	2	6	5	3	8
DC 43	1	5	3	6	4	21	4	2	3	S	S	16
	2	6	7	9	9	15	*	*	*	*	*	*
DC 44	1	4	2	4	3	21	2	2	3	S	S	11
	2	3	2	3	2	19	1	2	4	S	S	10
DC 45	1	8	7	9	8	12	3	2	3	S	S	13
	2	6	5	7	6	13	2	2	5	S	S	10
DC 46	1	4	4	5	4	15	2	4	6	S	S	10
	2	3	2	3	3	17	1	1	3	S	S	9
DC 47	1	8	7	10	10	19	1	4	7	S	S	10
	2	4	6	6	8	22	1	2	3	S	S	3
DC 48	1	2	1	2	1	28	1	1	4	S	S	14
	2	1	1	2	1	29	1	1	2	S	S	11
DC 49	1	4	4	5	5	18	4	1	2	S	S	17
	2	3	2	2	1	17	2	1	2	S	S	11
DC 50	1	9	9	10	10	9	10	5	7	8	S	11
	2	9	8	8	10	10	4	4	5	6	S	8
DC 51	1	5	4	5	4	17	4	2	4	4	S	16
	2	4	3	3	3	17	3	2	4	4	S	12
DC 52	1	10	8	10	10	6	9	6	7	8	7	3
	2	9	6	8	5	12	6	3	6	7	6	5
DC 53	1	7	5	6	5	10	5	4	7	7	S	7
	2	7	4	5	3	12	3	3	6	8	S	8
DC 54	1	10	10	10	9	8	5	5	7	7	S	7
	2	11	10	10	9	12	6	3	6	9	S	3
DC 55	1	9	3	6	4	15	11	6	5	6	8	11
	2	9	5	5	4	17	5	4	7	7	4	10
DC 56	1	3	2	3	2	25	2	1	2	3	S	22
	2	5	3	4	4	13	1	1	2	3	S	14

Results are the means of eight plants unless plants were lost to hoeing, wind damage, etc., insufficient seed was available for planting or plants were selfs. Ratings of % leaf area affected by rust for the second trial were done on the 18th (1) and 27th (2) of December and 3rd (3) and 10th (4) of December and for the third trial on 17th (1), 26th (2) and 31st (3) of

March and 8th (4) and 16th (5) of April.

Rep = Replication or block No. in trial Y = Yield in grammes per plant

S = Plants senescent by that date * = No data available

APPENDIX 6: Genstat 5 Release 2.2 programmes for the diallel analyses

The first programme (rust) calculates the sums of squares by analysing the data as 56 crosses and 8 selfs (ie. the cultivars). The second programme (diallel cross) analyses the data by considering it as an 8 x 8 diallel. The programmes were writen with the aid of Mr. J. Levin.

```
job'rust'
units[256]
factor[levels=2; val=128(1,2)]seasons
factor[levels=2; val=(1,2)128]blocks
factor[levels=64]crosses
open'A:parac';ch=2
read[ch=2]seasons,blocks,crosses,rate
model rate
terms blocks+seasons+crosses+crosses*seasons+crosses*blocks
fit blocks
add seasons
add crosses
add crosses*seasons
add[p=a,e]crosses*blocks
stop

job 'diallel cross'
units[256]
variate sup
fact [lev=2; val = 128(1,2)] block
fact [lev=2; val = (1,2)128] seas
  &  [lev=8] m
  &  [lev=8] f
  &  [lev=29] sca
  &  [lev=28] rec
open'A:para';ch=2
read[ch=2] seas,block,m,f,y
scal mi,fi
for i=2...8
calc g[i] = (i==m)*0.5 + (i==f)*0.5
endfor
calc s[1] = ((m==1).and.(f==2))+((m==2).and.(f==1))
calc r[1] = ((m==1).and.(f==2))-((m==2).and.(f==1))
calc s[2] = ((m==1).and.(f==3))+((m==3).and.(f==1))
```

```
calc r[2] = ((m==1).and.(f==3))-((m==3).and.(f==1))
calc s[3] = ((m==1).and.(f==4))+((m==4).and.(f==1))
calc r[3] = ((m==1).and.(f==4))-((m==4).and.(f==1))
calc s[4] = ((m==1).and.(f==5))+((m==5).and.(f==1))
calc r[4] = ((m==1).and.(f==5))-((m==5).and.(f==1))
calc s[5] = ((m==1).and.(f==6))+((m==6).and.(f==1))
calc r[5] = ((m==1).and.(f==6))-((m==6).and.(f==1))
calc s[6] = ((m==1).and.(f==7))+((m==7).and.(f==1))
calc r[6] = ((m==1).and.(f==7))-((m==7).and.(f==1))
calc s[7] = ((m==1).and.(f==8))+((m==8).and.(f==1))
calc r[7] = ((m==1).and.(f==8))-((m==8).and.(f==1))
calc s[8] = ((m==2).and.(f==3))+((m==3).and.(f==2))
calc r[8] = ((m==2).and.(f==3))-((m==3).and.(f==2))
calc s[9] = ((m==2).and.(f==4))+((m==4).and.(f==2))
calc r[9] = ((m==2).and.(f==4))-((m==4).and.(f==2))
calc s[10] = ((m==2).and.(f==5))+((m==5).and.(f==2))
calc r[10] = ((m==2).and.(f==5))-((m==5).and.(f==2))
calc s[11] = ((m==2).and.(f==6))+((m==6).and.(f==2))
calc r[11] = ((m==2).and.(f==6))-((m==6).and.(f==2))
calc s[12] = ((m==2).and.(f==7))+((m==7).and.(f==2))
calc r[12] = ((m==2).and.(f==7))-((m==7).and.(f==2))
calc s[13] = ((m==2).and.(f==8))+((m==8).and.(f==2))
calc r[13] = ((m==2).and.(f==8))-((m==8).and.(f==2))
calc s[14] = ((m==3).and.(f==4))+((m==4).and.(f==3))
calc r[14] = ((m==3).and.(f==4))-((m==4).and.(f==3))
calc s[15] = ((m==3).and.(f==5))+((m==5).and.(f==3))
calc r[15] = ((m==3).and.(f==5))-((m==5).and.(f==3))
calc s[16] = ((m==3).and.(f==6))+((m==6).and.(f==3))
calc r[16] = ((m==3).and.(f==6))-((m==6).and.(f==3))
calc s[17] = ((m==3).and.(f==7))+((m==7).and.(f==3))
calc r[17] = ((m==3).and.(f==7))-((m==7).and.(f==3))
calc s[18] = ((m==3).and.(f==8))+((m==8).and.(f==3))
calc r[18] = ((m==3).and.(f==8))-((m==8).and.(f==3))
calc s[19] = ((m==4).and.(f==5))+((m==5).and.(f==4))
calc r[19] = ((m==4).and.(f==5))-((m==5).and.(f==4))
calc s[20] = ((m==4).and.(f==6))+((m==6).and.(f==4))
calc r[20] = ((m==4).and.(f==6))-((m==6).and.(f==4))
calc s[21] = ((m==4).and.(f==7))+((m==7).and.(f==4))
calc r[21] = ((m==4).and.(f==7))-((m==7).and.(f==4))
calc s[22] = ((m==4).and.(f==8))+((m==8).and.(f==4))
calc r[22] = ((m==4).and.(f==8))-((m==8).and.(f==4))
```

```
calc s[23] = ((m==5).and.(f==6))+((m==6).and.(f==5))
calc r[23] = ((m==5).and.(f==6))-((m==6).and.(f==5))
calc s[24] = ((m==5).and.(f==7))+((m==7).and.(f==5))
calc r[24] = ((m==5).and.(f==7))-((m==7).and.(f==5))
calc s[25] = ((m==5).and.(f==8))+((m==8).and.(f==5))
calc r[25] = ((m==5).and.(f==8))-((m==8).and.(f==5))
calc s[26] = ((m==6).and.(f==7))+((m==7).and.(f==6))
calc r[26] = ((m==6).and.(f==7))-((m==7).and.(f==6))
calc s[27] = ((m==6).and.(f==8))+((m==8).and.(f==6))
calc r[27] = ((m==6).and.(f==8))-((m==8).and.(f==6))
calc s[28] = ((m==7).and.(f==8))+((m==8).and.(f==7))
calc r[28] = ((m==7).and.(f==8))-((m==8).and.(f==7))
calc sup = 1
for i=1...28
calc sup = sup +i*s[i]
endfor
calc sca = sup
model y
terms block + seas + g[] + sca + g[].seas + sca.seas[1]
fit [p=a] block
add [p=a] seas
add [p=a] g[]
add [p=a] sca
add [p=a] g[].seas
add [p=a] sca.seas
stop
```

1 = For calculation of the reciprocal value the terms statement and following lines are replaced as follows:

```
terms block + r[]
fit [p=a] block
add [p=a] r[]
stop
```

The terms statement can only accommodate a maximum of thirty parameters and hence a r[].seas (reciprocal x season) effect could not be calculated by Genstat.