STATEMENT

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university and to the best of my knowledge and belief, the thesis contains no material previously published or written by any other person, except where due reference is made in the text of the thesis.

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CONTENTS

ABSTRACT

The 22 races isolated from 299 *Uromyces appendiculatus* collections made during a four-year period in Eastern Australia formed a series in which most races differed from at least one other in pathogenicity on one of eight differentials. Most races were virulent for genes used by Australian breeders during the past two decades.

Genetic analyses of ten accessions of *Phaseolus vulgaris*, *viz.* Actopan/Sanilac Selection 37, Aurora, Bonita, Brown Beauty, Cornell 49-242, Gallaroy Genotypes I and II, NEP 2, PR 5 and Redlands Greenleaf B, indicated a minimum of 13 and a maximum of 15 genes for seedling resistance. Three loci with genes *Ur-1*, *Ur-2* or *Ur-2*^{2} and *Ur-3* were established and two others were postulated. Seven genes, *viz.* *Ur-1* and *Ur-2*, given permanent designations and *Ur-C*, *Ur-D*, *Ur-F*, *Ur-J* and *Ur-Red* which retained their temporary symbols gave resistance to certain Australian races, whereas eight others, *viz.* *Ur-2*^{2}, *Ur-3* , *Ur-G*, *Ur-H*, *Ur-I*, *Ur-K*, *Ur-L* and *Ur-N* were effective against all Australian races. Except for *Ur-L*, all genes effective against local races in greenhouse tests conferred a high degree of protection in the field. There was no linkage between genes controlling rust reaction and those conditioning presence or absence of seed coat colour or habit. Stocks with single genes for resistance were derived from accessions possessing resistance genes in combination. These should be of value in applying results of this study to other geographic areas where Sanilac is susceptible and may form the basis of a new set of tester genotypes, suitable both for local and international surveys. Genetic analyses of some testers, and infection types conferred by others, with a range of races, indicated that five differentials carry seven genes for rust resistance and that some have genes in common. Hypothetical phenotypes were allocated to three differentials for which no genetic data were available.

One-hundred-and-seventy-eight of 534 accessions were resistant to all Australian races and 165 were susceptible to all races. Hypothetical phenotypes

for rust reaction were assigned to the remainder on the basis of reaction patterns and infection types. Seedling resistance effective against all Australian races was present in approximately half of the dry beans from Africa, Central America and South America, but was rare in dry beans of the types commercially grown in Australia, and in the fleshy-podded beans with bush habit.

Resistance expressed in the field as slower and more limited disease development on cultivars susceptible as seedlings to the prevalent races, was studied in trials in which one or more races were released onto spreader rows. This type of resistance was present in many bush fleshy-podded cultivars but in few of the dry beans types commercially grown in Australia.

The inheritance of this form of resistance was studied in inbred lines derived from three two-way crosses between the resistant cultivars Apollo and California White Kidney and the susceptible Sanilac. The resistances were highly heritable but were not controlled by a gene at a single locus in either cultivar. In the cross between the resistant cultivars, transgressive segregation produced some lines with superior resistance and others with greater degrees of susceptibility than either parent. The high degree of resistance of some of the lines, the high correlations between ratings in two seasons, and the similar or identical ratings of replicates of most lines suggest that this form of resistance could be easily used by breeders.

The application of these results to improvement of the stability and degree of protection which may be obtained by breeding is discussed.

1. INTRODUCTION

Rust, caused by *Uromyces appendiculatus* (Pers.) Unger, on common bean, *Phaseolus vulgaris* L., occurs widely throughout the world. It is particularly destructive in tropical countries and other areas where beans are either grown for several months or are continually cropped. Severe infections on certain groups of cultivars have been reported from many geographic areas (Ballantyne 1974a). Yield reductions of 25 *per cent* were estimated on 56 *per cent* of the production area surveyed in Colombia (Anon. 1976a). Yield reductions of approximately 75 and 50 *per cent* in the cultivars Gallaroy and Kerman, respectively, in South-East Queensland were largely attributed to the effects of rust (Gallagher 1978). While the use of resistant cultivars is often the only feasible means of control, resistance has not been fully exploited because the pathogen occurs as many races differing in ability to cause damage on a range of cultivars. Until recently, little concentrated attention has been given to this problem.

Some forms of *P. vulgaris* are cultivated as a grain legume whereas others are grown for the fleshy pods used as a vegetable. The former, dry beans, are a staple food in Central America and South America, the regions considered to be primary and secondary centres of origin of the bean. The species has eleven pairs of chromosomes (2n=22) and is normally self-pollinating.

More attention is now being given to bean rust investigation. At a Bean Rust Workshop held at Centro Internationale de Agricultura Tropicale (CIAT), Cali, Colombia in 1974, papers were presented on the organization of international bean rust nurseries (Meiners 1974), resistance breeding strategies (Coyne and Schuster 1975), an infection type rating system (Augustin 1974), and the establishment of a set of international differential genotypes and a standard nomenclature of races (Ballantyne 1974b). CIAT began a bean improvement programme in 1973 in which selection for resistance to rust and other diseases are major objectives.

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The following strategies in breeding for rust resistance have been suggested (Vieira 1972, Ballantyne 1974a, Coyne and Schuster 1975, Evans 1975):-

- Combinations of genes for specific resistance. There is evidence with bean rust, as with many other diseases, that single genes for resistance in the host have not provided an adequate stability of protection.
- Improvement of resistance expressed as slow rusting.
- Use of specific resistances in slow rusting backgrounds.
- Multilines.

There has been insufficient information on the extent and nature of variability in the pathogen and on the genetic bases of resistance in the host to indicate which approach is most likely to be successful in the long term.

This thesis examines variation in pathogenicity of *U. appendiculatus* and the genetic bases of resistance in several *P. vulgaris* accessions in order to provide some information which should be useful in improving the stability and degree of protection obtainable by breeding.

Two approaches were adopted. Firstly, seedling studies were conducted in the greenhouse to (i) differentiate the pathogenic races and (ii) genetically analyse the seedling resistances of certain accessions in both crosses with a standard susceptible parent and in intercrosses among resistant accessions. This enabled genotypes to be allocated to host stocks and phenotypes to pathogen cultures. Using these host stocks as standards and the different races as testers, a large array of host germplasm was classified.

The second approach was to investigate the response of a wide range of host germplasm to various pathogenic races in the field, and to study the mode of inheritance of slow rusting shown by some accessions.

These studies attempt to apply to the bean rust situation the principles of the genetics of host pathogen interactions determined for several other disease systems (Gallegly and Niederhauser 1959, Watson 1970a, Flor 1971, Loegering *et al.* 1971, Day 1974).

1.1. Terminology

Terms used in this thesis to describe the host pathogen interaction are those defined by Loegering and Powers (1962). "Infection type" (IT) describes the lesions formed by the interaction of the bean and the rust fungus. When a

necrotic or chlorotic fleck, or a small pustule is formed, the IT is said to be "low", the reaction of the bean, "resistant" and the pathogenicity of the fungus, "avirulent" on that particular host. Similarly, when a large pustule is formed the IT is "high", the host, "susceptible" and the pathogen, "virulent".

The term "seedling resistance" is used to describe resistance expressed as a fleck or small pustule on seedlings in greenhouse tests. It does not include other forms of seedling or post-seedling resistance which may be expressed as slight reductions in spore mass produced, or numbers of pustules formed, as were described by Johnson and Bowyer (1974) and Priestley and Doling (1976), respectively. All the seedling resistances studied in this thesis are known to be specific on the basis of reactions obtained in Australia or overseas.

"Slow rusting" is used to describe resistance expressed as slower and more limited disease development in the field on some cultivars relative to others when both are of similar maturity and are equally susceptible as seedlings. The use of other terms will be restricted to quotations from published reports.

A "race" of the pathogen gives a characteristic array of reactions on a chosen group of host accessions known as a differential set. A "collection" represents a sample of rust-affected leaves or pods and may yield more than one race of the pathogen. A "culture" is a clone of the pathogen derived from a single pustule.

The host "differential set" usually consists of one accession susceptible to all races in the particular geographic area and several accessions which respond with resistant or susceptible reactions following infection. In addition, most investigators include a further group that has been consistently resistant throughout the particular area, since such a group is likely to include currently resistant cultivars and sources of resistance for breeders.

2. LITERATURE REVIEW

2.1. The pathogen

Uromyces appendiculatus is autoecious, but in some tropical areas it survives solely by means of urediniospores on a succession of host plants. Telia which form at the end of the season, or under cool conditions (Ogle and Johnson 1974), are considered to be the means of survival in at least some temperate regions (Milbrath 1944, Zaumeyer and Thomas 1957). According to Harter *et al.*(1935), some races never produce telia whereas others form them readily. Reports of aecia are few and are confined to temperate regions, but since the aecia are small, white and inconspicuous, they may be more common than the records indicate (Ballantyne 1974b).

Surveys of the bean rust pathogen populations in the Americas, Australia, East Africa, Europe and Hawaii have indicated considerable variability. However, the value of some surveys has been limited by at least three factors. Firstly, different host accessions bearing the same name may vary in genotype, *e.g.* 643 (Meiners personal communication) and 780 (Ogle and Johnson 1974). Secondly, while most workers adopted at least some of the host differential testers established by Harter and Zaumeyer (1941), others chose differentials that were limited to particular regions and for which seed was not generally available. Thirdly, most surveys were not done in conjunction with resistance breeding programmes and the testers did not include possible sources of resistance.

Thus results obtained at different times and places are difficult to interpret and are often of little relevance to breeding. Ballantyne (1974b) reviewed the international differential and race survey situation to 1974. More recent results are given by Coelho and Chaves (1975), Pereira and Chaves (1977) and Groth and Shrum (1977).

In the earliest Australian surveys conducted from 1942 to 1953, Waterhouse (1954) detected three races, two of which were avirulent on the widely grown "Wonder" cultivars, including Brown Beauty, whereas the third, 17A, isolated in 1948, was virulent. By 1953, race 17A had become widespread and had apparently spread to New Zealand (Brien and Jacks 1954) whereas the other two

races were comparatively rare. One of the early races avirulent on Brown Beauty produced a rating of 8 on a 0 - 10 infection type scale on the differential accession designated 643. However, Waterhouse's accessions of these differentials were not maintained and it is impossible to establish their relationships with current accessions bearing the same names.

In studies from 1954 to 1970, Johnson distinguished eight races, all virulent on Brown Beauty (Ogle and Johnson 1974). One race, recorded in 1970, was virulent on 643. Johnson established a set of five differentials including 643 and Golden Gate Wax, derived from the North American sets of Harter and Zaumeyer (1941) and Fisher (1952), respectively, plus CCGB 44, Redlands Greenleaf A and Epicure. He also determined the reactions of five other accessions used as race differentials by Harter and Zaumeyer (1941) plus 94 additional accessions, to the eight races.

2.2. The host

2.2.1. RUST RESISTANCE

Specific resistance has been reported by Harter *et al.* (1935), Hagedorn and Wade (1974), Ogle and Johnson (1974), Madriz and Vargas (1975), Rodrigues 1955, Rodriguez *et al.* (1977) and Groth and Shrum (1977). In Eastern Australia the Redlands series of cultivars, Gallaroy and others were bred for rust resistance, but new pathogen variants with virulence on them appeared either during seed increase or within four years of their release.

The differential 643, resistant to all Australian races detected from 1954 until 1970, was crossed with Brown Beauty and other cultivars to produce the Redlands series of green bean cultivars (Ogle and Johnson 1974), in one programme. In another Queensland breeding project 643 was crossed with Sanilac to obtain the dry (Navy) bean cultivars Burnia, Gallaroy and Kerman (Groszmann and Gallagher 1966, Gallagher 1968). The parentages of these cultivars and the sequence of new races suggested that two, or more genes in 643 were separated during selection of the new cultivars.

Instances of resistance where specificity has not been established have

also been reported. Vieira (1972) noted that several cultivars showed considerable "horizontal" resistance to rust under Brasilian conditions. Ballantyne (1974a) reported on trials with 158 cultivars in two seasons under conditions of natural infection. Differences in disease severity occurred; some cultivars susceptible as seedlings to the common races developed little rust either in field trials or in commercial crops where the same races were present.

Former studies on inheritance of specific resistance (Wingard 1933, Zaumeyer and Harter 1946, Yen and Brien 1960, Hikida 1961, Augustin *et al.* 1972, Anon 1976a) are of limited value to geneticists and breeders because in most instances the conclusions were drawn from testing only F2 populations. Futhermore, there is no indication that the particular host accessions and pathogen cultures used were maintained, that single-gene lines were extracted from multiple-gene combinations, or that intercrosses between resistant lines were tested. Hence comparisons between the results of work carried out at different times and places are impossible.

However, reports on the three cultivars *viz.* PR 5, Great Northern US 1140 and Westralia are relevant to this study. In naturally-infected field plots, results with F2 populations suggested that the resistance of PR 5 was simply inherited and dominant (Anon. 1976a). Until 1975, PR 5 was resistant to all known races of bean rust (Vakili personal communication), but as the International Bean Rust Nursery was more widely grown, this cultivar was found to be susceptible at several locations (Anon. 1977). A single dominant gene, R_{B11} was detected in US 1140 on the basis of testing F2 populations with Brasilian race 11 (Augustin *et al.* 1972). Australian accessions of US 1140 behave identically to one of the genotypes of Gallaroy analysed in this thesis. Yen and Brien (1960) detected a single gene in Westralia in tests on F2 populations and F3 lines with an unspecified New Zealand race(s). Westralia and its derivative Mangere Pole have remained resistant in Australia and New Zealand.

2.2.2. GENETIC MARKERS

Inheritance studies on growth habit and seed coat colour, characters used

as genetic markers in this thesis, have indicated varying degrees of complexity. However, there is general agreement that coloured seed depends on the presence of the dominant gene *P* at one locus and complementary genes at other loci. Either the absence of *P* or, less frequently, a complementary factor, results in white-seededness and absence of purple or brown stem pigment in seedlings (Moh 1971, Yarnell 1965). Similarly, while plant architecture generally may not be simply controlled, a single dominant gene has been found to govern indeterminate growth in all instances studied. The corresponding recessive allele results in determinate growth (Yarnell 1965).

2.3. Classification of disease resistance

A number of criteria have been used to classify disease resistance. The most commonly used are the relative effectiveness of resistance against different genotypes of the pathogen, the complexity of inheritance and the growth stage at which resistance is expressed. Hence various contrasting terms have been adopted to describe resistance; for example, vertical resistance and horizontal resistance (Van der Plank 1968, Robinson 1969, 1971, 1973), specific and non-specific (Watson 1970b), specific and general or generalized (Caldwell 1968), differential and uniform (Zadoks 1972), monogenic (or oligogenic) and polygenic (or multigenic), major gene and minor gene, seedling resistance and adult plant resistance (Anon. 1973b). Johnson and Law (1975) used the term durable resistance to describe resistance which had remained effective in the field for a relatively long period of time. The terms "slow rusting" (Mackenzie 1976) and "slow mildewing" (Shaner 1973) are becoming more widely used. These describe the host response but avoid possible connotations of specificity or durability.

Until recently, it was commonly believed that resistance expressed as slow disease development was effective against all variants of the pathogen and therefore was permanent in effect. Since data from several disease systems (Simons 1972, Parlevliet 1976) suggested that several genes were involved, the occurrence of new races of the pathogen able to overcome such resistance was thought unlikely. However, the persistent adult plant resistance of the wheat (*Triticum aestivum* L.,) cv. Hope and its derivatives, reported to have non-specific

resistance to the stem rust fungus† (Watson and Luig 1968b), was found by Hare (1976) to be simply inherited. This resistance was not readily detected in greenhouse seedling tests, and thus had some features in common with the bean rust resistance reported by Ballantyne (1974a) in the Redlands series and the Tendercrop group of green beans and certain dry beans such as the Red and White Kidney groups.

Breeders have been encouraged to use resistance expressed as slow disease development in preference to other forms (Robinson and Chiarappa 1975, Robinson 1976). However, there has been little critical experimental evidence available to indicate its long-term effectiveness, stability and mode of inheritance. Recent reports indicate that such resistance may be specific and thus ephemeral in at least some instances (Johnson and Taylor 1972, Clifford 1975, Ellingboe 1975, Johnson 1976, Habgood 1976, Martin and Ellingboe 1976, Parlevliet 1975b). Ellingboe (1975) stated that 'careful analysis in several laboratories has continued to accumulate evidence that horizontal resistance . . . is controlled by the same kinds of genes and the same kinds of genetic interactions as those controlling infection type'.

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† *Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.

3. GENERAL MATERIALS AND METHODS

3.1. Materials

3.1.1. THE HOST

As each host genotype was received, an accession number, prefixed by the letter B, was allocated. This number, rather than the name, is the basic reference for a particular host genotype. For example, the differential stock 643 from Johnson is B965, that from Meiners, B1595, and that from Oliveira, B1665. In addition, seed stocks from "Single Typical Plants" were established to ensure genetic homogeneity of host differentials and other accessions of interest. Pedigrees follow the system proposed by Purdy *et al.* (1968).

Sanilac, a well-adapted and widely grown Small White (Navy) bean cultivar, was used as the contrasting female parent in crosses between resistant and susceptible accessions. It was chosen because it was susceptible to all Australian races of *U. appendiculatus* (Ogle and Johnson 1974), has small seeds, thus increasing the likelihood of many seeds per plant and is a commercial cultivar. Thus the results may be applied to improvement of Small White beans. The cultivar Michelite from which Sanilac was bred (Minges 1972), and Small White beans generally, are susceptible to many overseas races (Harter and Zaumeyer 1941, Howland and Storey 1962, Netto *et al.* 1969). However, Michelite showed resistance to some Brasilian races (Netto *et al.* 1969) and Sanilac was resistant to at least one North American race (Zaumeyer 1960, Groth and Shrum 1977). Therefore genetic segregants and single gene lines derived from the present study should be useful in all areas where Sanilac is susceptible or where virulent races are available.

3.2. Methods

3.2.1. GREENHOUSE METHODS

Seedlings for inoculation were raised in the greenhouse in an unsterilized sand/mushroom compost mixture and were provided with supplementary feeding. Apical shoots were removed from plants seven to nine days after inoculation to improve light penetration to infected leaves and to facilitate handling.

3.2.1.1. Storage of pathogen cultures

Short-term storage of urediniospores was at 10 - 15°C and 50 *per cent*

relative humidity for periods of less than two months. For long-term storage, urediniospores were placed in aluminium foil packets and kept in a liquid nitrogen refrigerator. Immediately after removal from liquid nitrogen a heat shock of 40°C was applied for four minutes. All cultures used in genetic analyses and in rust reaction surveys were stored for future reference. These procedures are identical to those used by Australian cereal rust workers.

3.2.1.2. Inoculation and incubation

For seedling studies, primary leaves were inoculated when 1/3 to 2/3 expanded. Plants were counted before inoculation. This monitored germination rate and enabled later-germinating seedlings, or "escapes", to be distinguished from resistant plants showing few or no signs of infection.

Seedlings were inoculated by spraying a suspension of urediniospores in Odourless Mineral Spirit† above them with a pressure pack unit using Freon^{R} (dichorodifluoromethane) as propellant. Sufficient spores were added to the spirit to produce a faint brown colour.

Incubation was in a greenhouse with under-bench misting nozzles, for 16-22 hours.

3.2.1.3. Reaction assessments

Reactions were assessed on the basis of IT 12 to 14 days after inoculation. Sanilac was used as the reference susceptible genotype. The IT rating system used was a modification of the Davison and Vaughan(1963)system, incorporating the ";" to denote the necrotic or chlorotic fleck, as used by cereal rust workers. The range of pustule sizes was also extended, such that the six categories were:

0 No signs of infection

; Necrotic or chlorotic flecking

1 Pustules smaller than 300mμ

2 Pustules 301 - 499 mμ

3 Pustules 500 - 799 mμ

4 Pustules larger than 800 mμ

Pustule sizes close to the lower limit for each category were indicated by

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† available from Mobil Oil Company.

"-" and those approaching the upper limit by "+". Necrosis was indicated by "N" for a small necrotic area and "N+", "N++" and "N+++" for increasingly extensive degrees of necrosis. A definite necrotic ring surrounding the lesion was indicated by "NR", a purple area by "PA" and a white area by "WA".

Assessments of reaction were normally made on the lower leaf surface. Where reactions on upper and lower surfaces differed, the IT of the upper surface was recorded before a "/" and that of the lower after the slash. Where a range of ITs occurred on a single plant, the most frequent were recorded before the least frequent. For example, 2+3- indicates that most pustules were 2+ and others 3-. Where individual plants of one accession differed in reaction, the ITs were separated by ",". For example, ;1, 2+3- indicates that some plants of the accession produced IT ;1 and the others 2+3-.

Generally, ITs 0, ;, 1 and 2 were classified as low and interpreted as resistant reactions under greenhouse conditions; ITs 2+2 and 2+3- were classified as intermediate and similarly were interpreted as resistant reactions and ITs 3, 3+ and 4 were classified as high and regarded as susceptible reactions.

3.2.1.4. Race nomenclature

Each host differential was assigned a code letter and each race was named by the code letter, or letters, of the differential(s) on which it was virulent. As all races were virulent on differential b (Sanilac), this letter was omitted. In instances where three IT classes, high, intermediate and low were observed, "()" was used to indicate the intermediate level. Hence each race designation is an abbreviated avirulence/virulence formula appropriate to situations where a constant group of differentials is used. In such instances, statement of only the avirulence of the virulence attributes permits deduction of the total formula.

The occurrence of three of more IT classes on a single host inoculated with a range of cultures may indicate the presence of two or more genes in the host, heterozygosity and incomplete dominance of virulence or avirulence in the dikaryotic pathogen, or more complex progressive increases in virulence in the pathogen with respect to a single host gene (Watson and Luig 1968a).

3.2.1.5. Cultures used

Cultures used in greenhouse studies and released for field testing, together with their race designations and collection sites are listed in Table 1. Throughout this thesis race designations rather than culture numbers will be used.

3.2.2. FIELD STUDIES

3.2.2.1. Inoculation

Rust was established on spreader rows of Sanilac, either by spraying with a spore suspension, or by placing pots of infected plants in the row. When the weather was dry, plots were lightly spray-irrigated during late afternoon to provid favourable conditions for infection. Sanilac and other cultivars known to be severely affected were interspersed through the experimental plantings.

3.2.2.2. Disease assessments

Visual numerical assessments based on the four categories used by Ballantyne (1974a) were adopted, but the slight, moderate and severe categories were subdivided and a further class, "very severe", was added. The classes were as follows:

0 Resistant: Rust either absent or present only on senescent leaves as small pustules.

1 - 3 Slightly affected: Few to many leaves affected; pustules generally few, small and without a yellow halo; the few such haloes present occurred around the largest pustules. The plots or plants which showed least rust in this category were placed in class 1 and those with most rust in 3.

4 - 6 Moderately affected: Many leaves affected; pustules usually more abundant and larger than in the previous category. Within this category plots or plants with the least rust were rated 4 and those with most rust as 6.

7 - 9 Severely affected: All or most leaves affected; pustules more abundant and larger than in classes 1 - 6. Plots or plants showing least rust were placed in class 7 and those with the most rust in 9.

10 Very severely affected: All leaves affected; pustules larger and more numerous than in other groups; premature defoliation.

Table 1. Race designations and collection sites of cultures used to inoculate a range of host germplasm and in genetic studies

Culture no.	Race designation†	Collection Site
73.2	dh	Copmanhurst, N.S.W.
73.16	a(d)h(i)	Rydalmere, N.S.W.
73.17	gh	Rydalmere, N.S.W.
73.18	ch	Rydalmere, N.S.W.
73.34	fg	Castle Hill, N.S.W.
73.35	eg	Castle Hill, N.S.W.
74.15	egh	Castle Hill, N.S.W.
74.27	h	Rydalmere, N.S.W.
74.63	cfh	Rydalmere, N.S.W.
74.73	(a)dfhi	Valla, N.S.W.
74.75	(a)dhi	Valla, N.S.W.
75.80	adehi	Clare, Qld.
75.82	efg	Rydalmere, N.S.W.
75.87	(a)(d)h(i)	Gin Gin, N.S.W.
75.88	af	Clare, Qld.
75.92	adhi	Home Hill, Qld.
76.14	(a)c(d)h(i)	Lindenow, Vic.

† The designation is indicated by the code letters of the host differentials on which a particular race is virulent. The key to the symbols is in Table 3.

4. SURVEY OF *UROMYCES APPENDICULATUS* POPULATIONS

4.1. Introduction

The aims of this survey were to establish a set of differential host testers, to examine variation in the pathogen and to obtain a collection of cultures for use in genetic investigations and for breeding.

4.2. Materials and methods

Collections of bean rust were taken from commercial crops, home gardens and experimental plantings in three regions of Eastern Australia as shown in Figure 1.

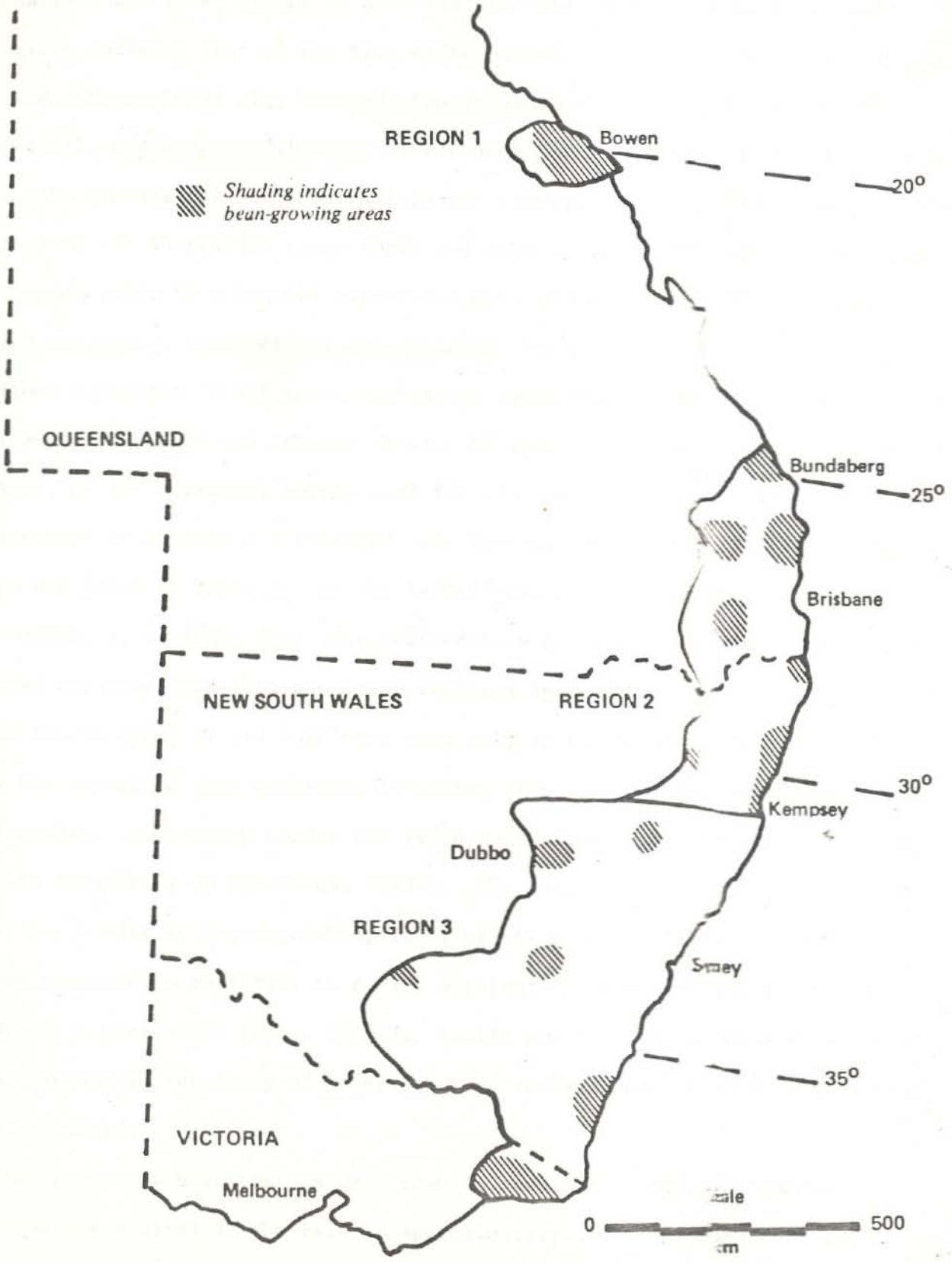
Region 1 includes the East Central Coast of Queensland, a tropical area where seed crops of a wide range of fleshy-podded and dry types are grown from April to September and where the rust fungus may persist on volunteer plants at other times of the year.

Region 2 embraces areas in two states and includes the Redlands Bay and Gympie districts of Queensland and the North Coast of New South Wales, areas where beans are likely to be cropped throughout the year. Apollo, Gallatin 50 and the Redlands cultivars predominated for most of the survey period. It also includes two areas where beans are grown only during summer, *viz.* the Kingaroy area in Queensland where Gallaroy and Kerman are used and the Northern Tablelands of New South Wales where limited production of a range of cultivars is carried out.

Region 3 includes other coastal and inland areas of New South Wales and Victoria. During the survey period, only fleshy-podded types, principally Apollo, Canyon and Gallatin 50 were grown in coastal areas, but a wide range of dry, as well as the above fleshy-podded, beans were produced inland.

Each rust collection was given a number, indicating year collected and order within the year. Since a collection represents a bean rust sample from the field, it may contain more than one race of the pathogen. On the other hand, a culture is derived from a single pustule and represents a single clone. Two cultures having the same virulence and avirulence attributes with respect to a set of host testers and therefore carrying the same race designation are not necessarily identical. Hence culture number is the basic reference while race designation

Figure 1. Main bean-growing areas of Eastern Mainland Australia showing the three regions described in the race survey



attempts to place sets of cultures into similar phenotypic groups. The collection number was used as a culture number in instances where only one race was preserved for future studies; another number was allocated where more than one race was chosen from a single collection.

Sanilac and three groups of host testers were used in the survey. The first group included four of the five differentials used routinely by Johnson (Ogle and Johnson 1974) plus Redlands Greenleaf B substituting for the fifth differential, Redlands Greenleaf A. The second group of three comprised Veracruz 1A6, one of the Australian differentials not routinely used by Johnson but reported resistant to all Australian races (Ogle and Johnson 1974), Brown Beauty which is grown commercially to a limited extent and used in the Queensland green bean breeding programme, and Redlands Greenleaf C. The third group included six accessions resistant to all known Australian races (Ballantyne 1974a and unpublished data, Ogle and Johnson 1974). As indicated in Table 2, some of these have been, or are currently being, used for similar purposes by overseas workers. Some descriptive details and allotted code letters for the set of differential testers are given in Table 3. In the latter stages of the project an additional differential, j, a single gene line derived from Sanilac/NEP 2 was added. Initially bulk seed was used, but this was later replaced by seed derived from single plants.

A fourth group of six cultivars used only in the preliminary stages of the survey was discarded when reactions were shown to be similar to the designated differentials. Hawkesbury Wonder and Tweed Wonder reacted identically to Brown Beauty in the survey of Waterhouse (1954). The parentage of Gallaroy suggested that it may assist in distinguishing races of the bean rust fungus. Ormiston had been reported as resistant to a race virulent on differentials a (643) and i (Redlands Greenleaf C) (Anon. 1973?). Apollo and California White Kidney were used as parents in the study of inheritance of resistance expressed as slow rusting in the field.

Two groups of host testers were sown in each 9.5 cm pot. The spore suspension was applied at the rate of approximately 1.5 ml to each host set in a mobile inoculation chamber. Precautions were taken to minimize contamination.

Table 2. Differentials and other testers used in race surveys of the bean rust fungus throughout the world

Differential	Surveys in which used[†]
3	1,2,3,4,5,6,7,8,9,11,13,15,16,17,18,20
181	1,2,4,6,11,13,18
643	1,2,3,4,5,6,7,8,11,13,15,16,17,18,19,20
650	1,2,3,4,5,8,11,13,17,18
765	1,2,3,4,5,6,7,8,11,13,15,16,18
780	1,2,3,4,5,6,7,8,11,13,17,18
814	1,2,3,4,5,6,7,8,9,11,13,15,16,17,18
Golden Gate Wax	2,3,4,5,6,7,8,15,16,17,18,19,20
Z4	2
Dade	7
Bayo Camana	9,14
California Small White[‡]	9
Canario 101	9,11,12
Cornell 49-242	9,20
Diacol Nutibara	9
Kentucky Wonder White[§]	9
Redlands Greenleaf C	9,20
Turrialba 4	9
Mulatinho	11
Cuva 168-N	11
Aguascalientes 13	12
Guerrero 6	12
Guerrero 9	12
Guanajuato 10A-5	12
Mexico 6	12
Mexico 12	12
Veracruz	12
Negro 150	12
Bayo	14
Caraotas	14
Panamito Mejorado	14
Panamito Sanilac	14
Canario LM	14
Canario Divex 8120	14

Table 2. Differentials and other testers used in race surveys of the bean rust fungus throughout the world (cont.)

Differential	Surveys in which used
Cocaho	14
Tengeru Sel. 8¶	15,16
Feijao castanho (423 EAN)	17
Idaho Pinto	17
Pinto US 1	17
Pinto US 5	17
Pinto US 14	17
"Wonder var." *e.g.* Hawkesbury Wonder or Brown Beauty	18 20
Redlands Greenleaf A††	19
Redlands Greenleaf B††	20
Epicure	19,20
CCGB 44	19,20
Veracruz 1A6	20
Actopan/Sanilac Selection 37	20
Aurora	20
Bonita	20
NEP 2	20
PR 5	20
Sanilac	20

†Key

U.S.A.

1 Harter & Zaumeyer 1941
2 Fisher 1952
3 Sappenfield 1954
4 Zaumeyer 1960
5 Hikida 1961
6 Goode 1961
7 McMillan 1972

BRASIL

Minas Gerais
8 Netto *et al.* 1969
9 Pereira & Chaves 1977

Rio Grand do Sol
10 Dias & da Costa 1968
11 Augustin & da Costa 1971 a & b

MEXICO

12 Crispin & Dongo 1972

COSTA RICA

13 Christen & Echandi 1967

PERU

14 Dongo pers. comm.

EAST AFRICA

15 Howland & Storey 1962
16 Howland & Macartney 1966

PORTUGAL

17 Rodrigues 1955

Table 2. Differentials and other testers used in race surveys of the bean rust fungus throughout the world (cont.)

Key (cont.)

AUSTRALIA
18 Waterhouse 1954
19 Ogle & Johnson 1974
20 This Survey

† may be similar or identical to 643.

§ may be similar or identical to 3.

¶ Ecuador 66 is reported to be a synonym (D. Allen personal communication).

†† Redlands Greenleaf strains A and B have reacted identically with a range of Australian cultures.

Table 3. Details of genotypes used in race survey and in genetic analyses

Code Letter	Number	Accession Name	Use in Australia	Type	Habit	Seed coat colour
Australian Universal Suscept						
b	B1555	Sanilac	Breeding	Dry	Bush	White
Differentials						
a	B1554	643	Breeding	Dry	Vine	White
c	B1556	Golden Gate Wax	Experimental			
d	B1557	†Redlands Greenleaf B	Commercial	Fleshy-podded	Bush	Brown
			Breeding			
e	B1558	CCGB 44	Experimental			
f	B1559	Veracruz 1A6	Experimental			
g	B1560	Epicure	Home garden			
h	B1561	†Brown Beauty	Commercial	Fleshy-podded	Bush	Brown
i	B1562	Redlands Greenleaf C	Commercial	Fleshy-podded	Bush	Brown
j	B2053	Sanilac/NEP 2 Selection	Experimental			
Accessions resistant to all Australian races						
	B1564	†Actopan/Sanilac Selection 37	Breeding	Dry	Bush	White
	B1568	†Aurora	Experimental	Dry	Vine	White
	B1626	†Bonita	Experimental	Dry	Vine	White
	B1672	†Cornell 49-242	Experimental	Dry	Vine	Black
	B1666	†NEP 2	Experimental	Dry	Vine	White
	B1667	†PR 5	Experimental	Dry	Vine	Black
Accessions susceptible to all or some Australian races and used in preliminary part of survey						
	B617	Apollo	Commercial	Fleshy-podded	Bush	White
	B1210	California White Kidney	Experimental	Dry	Bush	White
	B750	†Gallaroy	Commercial	Dry	Bush	White
	B1263	Hawkesbury Wonder	Commercial	Fleshy-podded		
	B1355	Ormiston	Experimental	Fleshy-podded		
	B1254	Tweed Wonder	Commercial	Fleshy-podded		

† accessions subjected to genetic analysis.

The plants in the chamber were almost saturated with a fine mist of water immediately after inoculation and the mist was left to settle for at least one minute. After the pots were removed, the chamber was washed by an internally mounted sprinkler system. The pressure pack spraying unit was sterilized with methylated spirits for at least one minute and washed under running water.

After incubation, all sets inoculated at a particular time were kept in one greenhouse for six to seven days, to provide similar environmental conditions before removal to separate greenhouse cubicles for isolation.

Where mixed reactions occurred on a differential, single pustules were isolated, increased on Sanilac, or the tester on which the large pustules occurred, and the resultant cultures reinoculated onto host sets.

4.3 Results

4.3.1. REACTION PATTERNS

The eight accessions coded as a, c, d, e, f, g, h and i (Table 3) reacted differentially, whereas Actopan/Sanilac Selection 37, Aurora, Bonita, Cornell 49-242, NEP 2 and PR 5 were resistant to all collections. Brown Beauty B1258, used initially as differential h, proved heterogeneous when inoculated with certain races, having most plants resistant and a few susceptible. B1258 was replaced with B1561 which was derived from a single resistant plant.

Of the accessions included only in preliminary studies, four were homogeneous and two heterogeneous. The homogeneous accessions were firstly, California White Kidney and Tweed Wonder which were susceptible to all collections, and secondly, Apollo and Tendercrop which reacted similarly to differential h. The heterogeneous lines were Gallaroy and Ormiston. While Gallaroy was heterogeneous when tested with most races, it was homogeneous when tested with the (a)dhi and adhi groups of races, although the IT with (a)dhi was different to that produced in tests with adhi:-

	Race		
	(a)dhi group	adhi group	Others e.g. h
Callaroy Genotype I	2+2	3+4	2+2
Callaroy Genotype II	2+2	3+4	;
643 (differential a)	2+2	3+4	;
Sanilac	3+4	3+4	3+4

In this table, the two types of Gallaroy are named Genotypes I and II. The "(a)dhi group" includes races (a)cdhi, (a)dfhi and (a)dhi and the "adhi group" includes adehi and adhi.

Some plants of Ormiston responded in a similar manner to differential h, whereas others reacted in a similar way to differential d :-

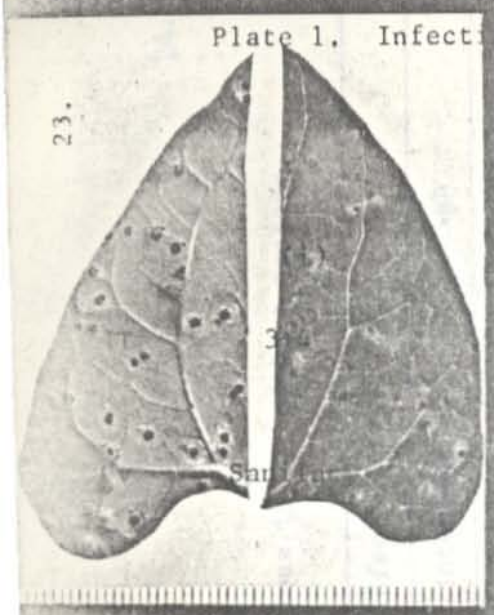
	Race			
	dh group	(d)h(i) group	Other h groups	Groups avirulent on h
Ormiston Genotype I	3+4	3+4	3+4	22-NR22-
Ormiston Genotype II	3+4	22+3-3	;	;
Brown Beauty or (differential h)	3+4	3+4	3+4	22-NR22-
Redlands Greenleaf B (differential d)	3+4	22+3-3	;	;
Sanilac	3+4	3+4	3+4	3+4

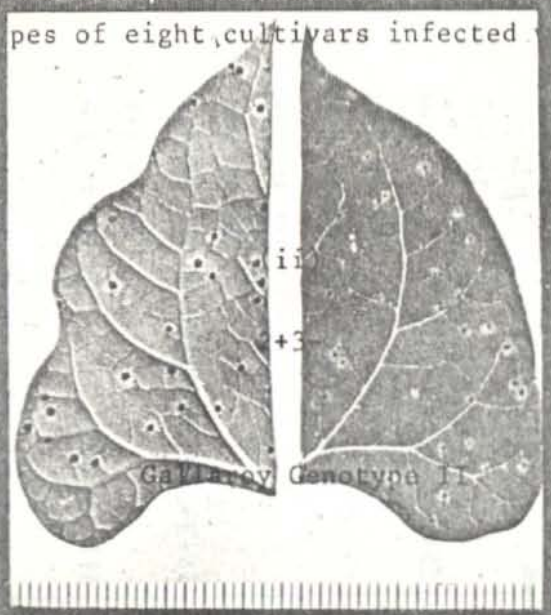
4.3.2. REACTIONS OF INDIVIDUAL ACCESSIONS

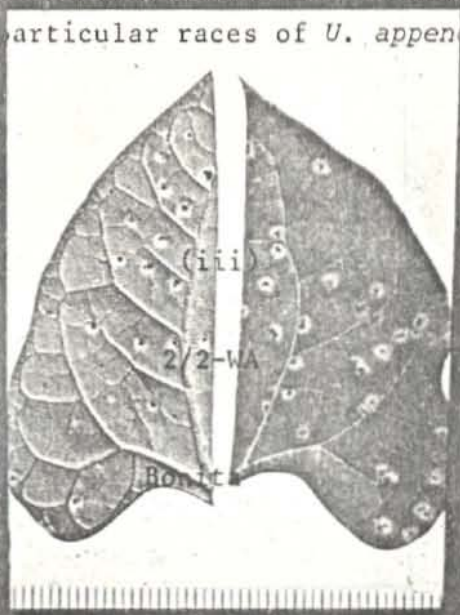
In susceptible reactions, pustules occurring as white blisters at five to seven days after inoculation began to erupt at seven to nine days and became fully expanded at 12 - 14 days. A secondary ring of smaller pustules sometimes developed around the large primary pustule (Plate 1, i)

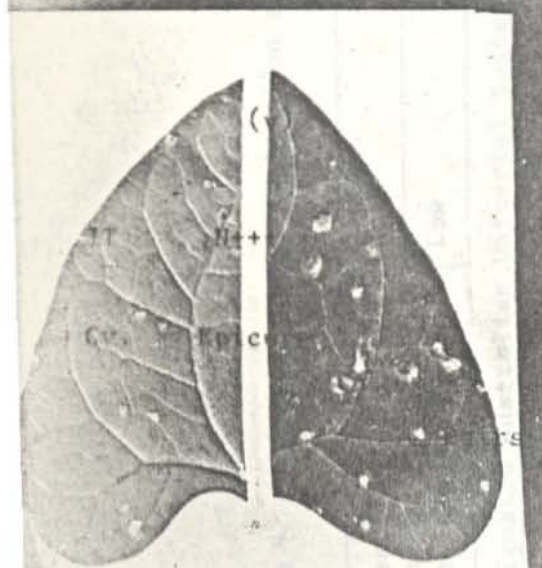
In resistant reactions a wide range of ITs was produced. These are described in Table 4 and some are illustrated in Plate 1 (ii) to (viii). Some ITs were characteristic. These included g (Epicure) with ITs ;N+++ 1N+++ 2N+++ (v)

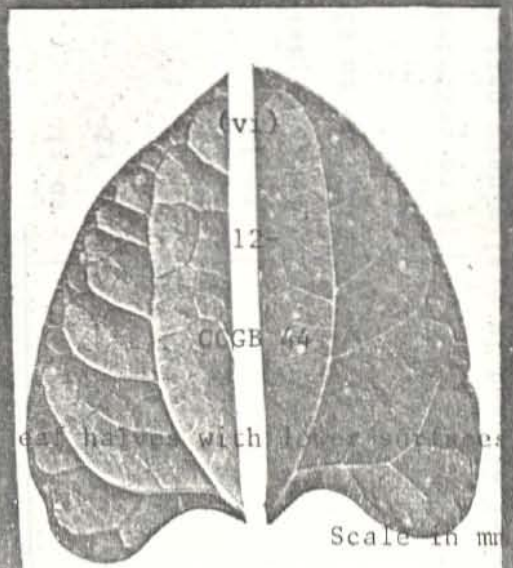
Plate 1. Infecti… …pes of eight cultivars infected … …articular races of *U. appen*… …*tus*

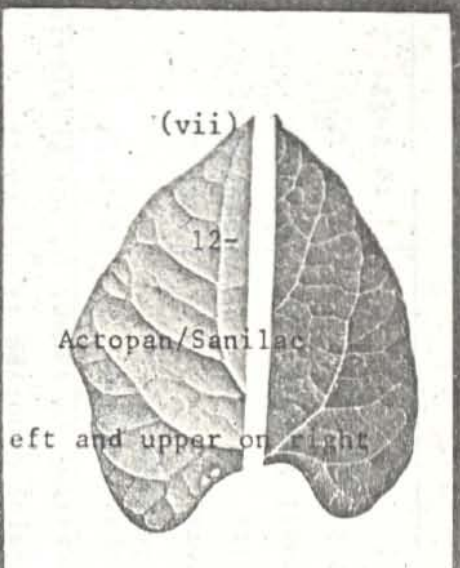












…leaf halves with … …eft and upper on right

Table 4. Infection types produced on accessions in set of host testers

Accession name on code letter	Infection Types		
	Low	Intermediate	High
Designated Australian Universal Suscept			
b			3+ to 3+4
Designated Differentials			
a	; to ;1	2+2 to 2+3-	3+ to 3+4
c	; to 12-		3+ to 3+4
d	;	22+3- to 2+3-33+	3+ to 3+4
e	; to 12-		3+ to 3+4
f	; to 12-		3+ to 3+4
g	12N+++ to ;N+++		3+ to 3+4
h	22-NR22- to 22+NR22+		3+ to 3+4
i	;1	22+3- to 2+3-33+	3+ to 3+4
j	;1 to 2-22+3-3NR		3+ to 3+4
Accessions used in preliminary part of survey			
Gallaroy Genotype I	2+2 to 2+3-		3+ to 3+4
Gallaroy Genotype II	; to ;1	2+2 to 2+3-	3+ to 3+4
Accessions resistant to all Australian races			
Actopan/Sanilac Selection 37	; to ;1		
Aurora	; to ;N		
Bonita	2/2-WA to 2+/2WA		
Cornell 49-242	; to ;1		
NEP 2	; to ;N		
PR 5	; to 2-22+		

Gallaroy and differential a giving IT 22+ or 2+3- (ii) with certain races, Aurora and NEP 2 with IT ;N (viii) and Bonita with a white area around the pustule on the lower leaf surface and IT 2/2-WA to 2+2/WA (iii).

As indicated in Table 4 five testers used throughout the survey produced either a low or a high IT, but three showed low, intermediate and high ITs. Two differentials, d and i, showed identical intermediate ITs, which were different from that seen on differential a and Gallaroy.

4.3.3. INFLUENCE OF ENVIRONMENT ON IT

The reaction of h was influenced by temperature. Under warm to hot conditions (>25°C), races avirulent on this cultivar produced a small pustule, usually with a necrotic ring, but under cool conditions (<20°C), a much larger pustule without necrosis was formed. This latter reaction could not be reliably distinguished from susceptibility. Pustules produced by races virulent on differential h were sometimes ringed by a faint purple line which was characteristic of this host and other accessions with the same spectrum of reactions. The pigmentation usually developed under hot conditions.

4.3.4. RACES IDENTIFIED

The 346 identifications made from the 299 collections examined in this survey indicated twenty-two races. Their frequencies in the 213 samples from commercial crops, home gardens and naturally-infected experimental plantings plus three races found only in inoculated plantings are presented in Table 5. Frequencies of the three races recorded only by Johnson are also given in this Table. Results for field plantings in which previous collections were the source of inoculum will be reported in later sections.

Four races which occurred in more than 15 *per cent* of collections, *viz.* adhi, (a)dhi, gh and (a)(d)h(i), were classified as abundant, 12 which occurred in two *per cent* or fewer samples were considered rare, and four, *viz.*ch, egh, a(d)h(i) and fg, were in an intermediate category (Table 6). Two races with virulence on only one or two differentials, h and dh, respectively, were of unknown prevalence as they may have been present, undetected, in mixtures with more complex races virulent on more differentials.

Table 5. Numbers of collections from non-inoculated plantings in which *U. appendiculatus* races were recorded in Eastern Australia 1954-1977

Race	Number of collections					
	This Survey					Ogle & Johnson 1974
	1973-74	1975	1976	1977	Total Number	
adhi	11	52	30	3	96	5
(a)dhi	12	12	39	5	68	0
gh	28	9	17	4	58	8
(a)(d)h(i)	1	6	23	0	30	0
ch	10	4	1	1	16	59
dh	5	9	1	3	18	45
egh	9	1	8	0	18	0
a(d)h(i)	1	7	2	0	10	0
fg	5	0	3	1	9	0
(a)dfhi	1	1	2	0	4	0
h	1	3	1	0	5	14
af	0	2	0	0	2	0
cfh	2	0	0	0	2	0
eg	2	0	0	0	2	0
eh†	0	1	0	0	1	0
efg‡	0	1	0	0	1	0
a	0	1	0	0	1	0
dfh	0	0	1	0	1	0
adehi	0	1	0	0	1	0
(a)c(d)h(i)	0	0	1	0	1	0
(a)c(d)eh(i)†	0	0	0	1	1	0
(a)cdhi	0	0	0	1	1	0
ceh	0	0	0	0	0	14
deh	0	0	0	0	0	11
cdh	0	0	0	0	0	3
Totals	88	110	129	19	346	159

† found only in inoculated greenhouse experiments.

‡ found only in inoculated field planting.

One-hundred-and-twenty-two naturally-infected samples yielded one race , 71 yielded two, 15 were of three races, five contained four, one yielded five and two were of six races. Thus more than half the collections contained more than one race, presumably providing opportunities for hyphal anastomoses and recombination between different races.

4.3.5. GROUPING OF RACES

The grouping of races according to prevalence and virulence formulae presente in Table 6 shows that:-

- All the abundant races were virulent on h and avirulent on both the e (CCGB 44) and f (Veracruz 1A6) differentials.
- By contrast, the rare races and one (egh) of the intermediate group were virulent on one or more of the c (Golden Gate Wax), e or f differentials or avirulent on h. In addition, three races, af, fg and efg, were virulent on e and f, or both, and avirulent on h. These also occurred in mixtures with more prevalent races with which they had some virulence genes in common. For example, race cfh was present in certain collections with the simpler race ch, of the intermediate category; race (a)c(d)h(i) was recorded only once from a planting in which (a)(d)h(i), of intermediate prevalence, was widespread; three races, eg (rare), fg and egh (intermediate) occurred in mixtures with gh.

Two races were detected only in greenhouse inoculation tests with pure cultures. Race eh occurred as isolated pustules on F3 lines of Sanilac/NEP 2 screened with race h. Race (a)c(d)eh(i) was detected on differential e inoculated with race (a)c(d)h(i). They presumably resulted from mutations within the inoculated cultures.

Nine of the rare races differed from the abundant and intermediate races in pathogenicity on single differentials and three differed in pathogenicity on two differentials. One such rare race, (a)c(d)eh(i), differed only with respect to differential e, from another rare race from which it was isolated in the greenhouse. The extent of such differences is important in considering the patterns of variation and the possible mechanisms whereby changes in pathogenicity occur.

Table 6. A grouping of races according to prevalence and virulence formulae

Abundant	Unknown abundance	Intermediate	Rare	
			Differed from most similar race in reaction on	
			one differential	two differentials
	h	ch ceh†	eh cfh	
gh		egh fg	eg	efg
	dh	cdh† deh†	dfh	
		a(d)h(i)	(a)c(d)h(i)	(a)c(d)eh(i)
(a)dhi			(a)dfhi (a)cdhi	
a(d)h(i)				
adhi			a adehi	af

† denotes races recorded only by Ogle & Johnson 1974.

4.3.6. COMPARISONS WITH RESULTS OF PREVIOUS AUSTRALIAN SURVEYS

A continuing knowledge of the races present and an understanding of the patterns of pathogen change are relevant to breeding programmes aimed at rust resistance.

Some comparisons with the survey reported by Ogle and Johnson (1974) are relevant since seed of all differentials, except h, was derived from Johnson's stocks. The number of rust collections examined by Johnson was not reported, but is estimated at approximately 100. Thus, combining his results with those of the present study, a total of about 400 collections were examined from 1954 to 1977 (Table 5).

Fourteen previously undescribed races were detected in the current survey. Six of these are virulent on differential f which was not routinely used by Johnson. On the other hand, three of his races (cdh, ceh and deh) were not found. Thus if these races found only by Johnson are also considered, the 25 races reported in Eastern Australia within the past two decades form a series in which there may be increases or decreases in the number of virulence factors, usually in single factor steps.

Only limited comparisons may be made with the results of Waterhouse, as neither his seed stocks nor cultures were maintained. His two earliest races were avirulent on Brown Beauty, Hawkesbury Wonder and Tweed Wonder. In the present survey, Brown Beauty was found to be heterogeneous, with some plants susceptible to all races, and others resistant to certain races. The races avirulent on this latter genotype of Brown Beauty were also avirulent on Hawkesbury Wonder but were virulent on Tweed Wonder. One explanation is that the Tweed Wonder stocks used in the two surveys carry different genes. However, no change in commercial seed stocks during this period is known. The second explanation, that the races detected in the two surveys carried different genes for virulence on Brown Beauty, is supported by two facts. Firstly, Waterhouse made no mention of any temperature sensitivity of the resistant reaction of Brown Beauty and Hawkesbury Wonder, yet such an effect was obvious in these studies. Secondly,

the IT of Brown Beauty with the early races was 2 on a 0 - 10 scale, which may be lower than the 22-NR on a 0 - 4 scale produced by races a, af, eg, fg and efg in this survey. Waterhouse did not report heterogeneity in Brown Beauty.

While the resistance of Brown Beauty, Hawkesbury Wonder and Tweed Wonder was effective against all early races, there is no information regarding the field effectiveness of the gene(s) governing the IT 22-NR seen in this survey.

The ways in which race changes may occur will be discussed in Section 8.1.

4.3.7. INFLUENCE OF TYPE OF PLANTING AND HOST GENOTYPE ON RACE DISTRIBUTION

Table 7 shows the distribution of the collections according to the types of planting from which they were sampled. Of the 213 collections from naturally-infected plantings, only about one-third were from commercial crops. The small number sampled restricts the conclusions that can be made.

Forty-seven fleshy-podded and 36 dry bean cultivars were sampled while 21 collections were from unknown cultivars.

Some races were isolated from leaves of host cultivars on which they were avirulent in the seedling stage. For example, races fg and gh which both gave IT ; on Redlands Greenleaf C were isolated from one collection of this cultivar. This was attributed to senescence of the host permitting some sporulation by races avirulent in the seedling stage.

4.3.8. FREQUENCIES OF RACES VIRULENT ON INDIVIDUAL DIFFERENTIALS

The isolation frequencies of individual races in non-inoculated plantings were totalled and the frequencies of races giving high and intermediate reactions on particular differentials were calculated (Table 8). These results will be considered in more detail (Section 6.2.9., p132) after the genotypes or phenotypes of differentials and commercial cultivars have been assigned. However, it is obvious that most races were virulent on differential h while lower proportions were virulent on other differentials. The frequencies of races virulent on differentials with genes present in commercial cultivars (differentials a, d and i) were generally higher than on those differentials with genes not present in such cultivars. One exception was Epicure (g) which is widely grown in home gardens.

Table 7. Distribution of bean rust collections, 1973-1977, according to type of planting

Type of planting	Year				Totals
	1973-74	1975	1976	1977	
Non-inoculated					
Commercial crops	18	43	30	3	93
Breeders plots in production areas	2	22	47	-	71
Other experimental crops	11	4	7	2	24
Home gardens	17	2	4	3	25
Subtotals	48	71	86	8	213
Inoculated					
Field and greenhouse experiments	38	8†	21†	19†	86
Totals	86	79	107	27	299

† new races detected in this group of collections.

Table 8. Percentages of races giving high or intermediate ITs on eight differentials during 1973-1977 in three regions

Region	Year	% of races giving high or intermediate ITs on differentials with resistance genes										
		present in commercial cultivars							not present in commercial cultivars				Total frequency
		h	d	i	a	(a)	(d)	(i)	g†	e	c	f	
1	all	95	71	71	81	14	17	17	5	5	0	3	17
2	all	96	68	60	32	31	4	4	25	9	3	5	37
3	all	95	37	32	14	36	17	17	34	14	10	5	45
Greenhouse	all	100	0	0	0	50	50	50	0	100	50	0	1
all	1973-4	92	32	27	14	16	2	2	50	13	14	7	25
all	1975	96	69	60	56	17	12	12	10	4	4	4	32
all	1976	98	57	55	25	50	20	20	26	26	2	5	37
all	1977	95	58	42	16	37	5	5	26	5	11	5	6
all	all	95	54	49	31	36	12	12	25	6	5	5	100(346)

† grown in home gardens.

The frequency of races avirulent on h may be underestimated as all such races, except a, were isolated as single-pustule cultures virulent on the e or f differentials. Race a was isolated as a single-pustule culture from differential a. Consideration should be given in future surveys to retesting of collections in which races other than the common ones occurred, particularly where these races are avirulent on h. The culturing and testing of a range of single-pustule isolates from Sanilac and possibly certain other differentials may permit additional races to be isolated from such collections.

4.3.9. COMBINATIONS OF VIRULENCE AND AVIRULENCE FACTORS

While three races were isolated in which virulence on f occurred in combination with virulence on h, *viz.* cfh, dfh and (a)dfhi, no races were detected in which virulence on f and g occurred with virulence on h. While the number of samples examined was obviously small, race fg was recorded in nine separate collections from relatively isolated areas. In contrast, both races eg and egh were found although egh was more common than eg. If no race of designation fgh is detected in more extensive surveys, the combination of the corresponding resistance genes may be useful in breeding programmes.

4.3.10. MEANS OF OVERWINTERING

The means of overwintering may influence race distribution. Since beans are grown throughout the year in coastal areas of Region 2, a succession of crops enables the fungus to survive on the living host. Beans are not cultivated throughout the year in coastal areas of Region 3. However, rust-affected beans survive into, and possibly through, the winter, as shown by the collection made in July at Rydalmere in the Sydney Metropolitan Area. The first plantings of beans in home gardens in this area normally occur in August. Survival may occur on volunteer plants in sheltered coastal locations of Region 3 and may provide inoculum for spring plantings on the coast, as well as for inland areas where more severe and widespread frosts should prevent the survival of beans. Consequently, the more prevalent races have a greater chance of overwintering

than the less prevalent ones. Nevertheless with small populations, survival is a chance occurrence and races rare in one year may occur with high frequencies in the following season. Although Region 1 was too far from Sydney for close inspection it is assumed that survival occurs on volunteer plants during the summer when beans are not commercially grown.

4.3.11. PRODUCTION OF TELIA

No specific experiments were undertaken to investigate telial formation. However, records were kept of races which formed telia when infected plants were kept for longer than normal, for collection of spores and maintenance of cultures. Only races dh, (a)(d)h(i), (a)dhi, (a)dfhi, adhi and adehi formed telia in the greenhouse. This observation is not necessarily evidence that this group represents an evolutionary sequence.

5. GENETIC ANALYSES

5.1. Introduction

The objectives were to obtain data on the mode of inheritance of seedling resistance, and where more than one gene was involved, to derive lines carrying single genes for resistance. In race surveys, single gene lines permit more precise resolution of races and give more accurate estimations of the frequencies of races virulent for particular host genes. In addition, single gene lines assist in the application of results of race surveys and genetic studies made in one geographic area to other areas.

5.2. Materials and methods

5.2.1. CROSSING

In most instances, parental plants were individually designated and harvested separately to provide reference stocks of the particular accessions analysed. This overcame problems associated with genetic heterogeneity within cultivars subjected to genetic analysis. Detailed family results will be presented for the first cross in order to illustrate the methods of analysis, but for subsequent crosses, homogeneous data will be pooled. Crosses of Sanilac with Gallaroy and Redlands Greenleaf B were made with undesignated plants whose progenies were not kept.

Wherever possible within a particular cross, the F1 seeds grown to establish F2, F3 and F4 populations were taken from the same pod or had a pollen parent in common. While records and reference stocks of the Sanilac parent used as female parents were kept, these were not considered further as Sanilac proved susceptible to all Australian races. Stocks derived from Single Typical Plants were used in intercrosses between cultivars and single gene lines.

5.2.2 NOTATION SYSTEMS

Each family group derived from the same pollen parent was given the same number and each family group derived from a single F1 seed from this parent was given a letter. Thus family groups 1A, 1B and 1C were all derived from a single pollen parent, but 1A was derived from a distinctive F1 seed. Family groups 1A,

2A and 3A were derived from different pollen parents.

Each F2 plant was given two numbers, one which was common to all plants derived from a single F1 seed, *e.g.* 194 and another to indicate individual plants, *e.g.* 194.1. The F3 plants were denoted by three groups of numbers based on the F3 number, *e.g.* 194.1.1, and likewise F4 plants were given four groups of numbers.

5.2.3. GENE SYMBOLS

Each gene was named using the symbol *Ur* approved by Dr. D.H. Wallace, Convenor of the Bean Gene Symbols Committee, and a distinguishing suffix. Initially all genes were assigned temporary symbols of *Ur-* and a letter, *e.g. Ur-A, Ur-B, etc.* Subsequently, where sufficient data were available on location and identity, certain genes were given permanent symbols, with a number replacing the letter and a superscript to indicate allelism, *e.g. Ur-1, Ur-2, Ur-2^2, etc.* according to the rules advised by Dr. Wallace. These rules are similar to those adopted by workers with Cucurbitaceae (Robinson *et al.* 1976).

The corresponding genes for pathogenicity in the pathogen were designated by the symbols *P* and *p*, for avirulence and virulence, respectively, plus the letter or number symbol of the host resistance gene, *viz. P-A, p-A, etc.*

5.2.4. OTHER SYMBOLS AND ABBREVIATIONS IN TABLES

* significant deviation from expectation at P = 0.05.

** significant deviation from expectation at P = 0.01.

The following abbreviations were used

d.f.	=	Degrees of freedom
Res.	=	Resistant
Sus.	=	Susceptible
Hom. Res.	=	Homozygous resistant
Hom. Sus.	=	Homozygous susceptible
Seg.	=	Segregating

5.2.5. RAISING PLANTS

Most F1 plants, and seedlings tested in the greenhouse and grown on for progeny-testing, were raised in the greenhouse. Other plants were raised in field rows using 30 cm spacings to facilitate note-taking and harvesting. All plants were labelled ; those with vine habit were staked and tied. Details of field rust reactions, and habits of F2 plants were recorded. F2 plants classified for rust reaction in the greenhouse and transplanted into the field produced few seeds because of inadequate recovery from transplanting. Consequently, F3 tests were based mainly on untested random F2 plants.

5.2.6. DERIVATION OF SINGLE GENE STOCKS

In deriving single gene stocks, F4 populations raised from 20 seeds of selected F3 lines segregating monogenically were tested with a minimum of three widely differing avirulent races and with one virulent race if available. One homozygous resistant F4 line with superior agronomic features was chosen as the single gene line. In two instances where no such F4 line was recovered, a segregating line was used to generate F5 lines on which similar tests were made.

5.2.7. HARVESTING AND THRESHING

During fine weather, pods were harvested every two or three days from plants standing in the field. However, during periods of prolonged wet weather, plants were pulled and cured under cover. All single plant progenies were hand-shelled.

5.2.8. POPULATION SIZE

Sixteen to 20 seedlings of each F3 line were inoculated with a selected race in the first instance. This number ensured that errors in distinguishing lines segregating for contrasting alleles at a single locus from homozygous resistant lines would occur with a probability of less than 0.01. When more complex genetic situations arose, larger populations were tested. For example, 46 seedlings enabled the distinction of a two-locus segregation ratio from homozygosity at $P = 0.01$. Additionally, this number exceeds 35, which at $P = 0.05$ is needed to distinguish lines segregating at one locus from those segregating at two genetically independent loci (Mather 1963).

Unfortunately, seed numbers were limited in some lines, especially those from crosses involving large-seeded cultivars, such as Brown Beauty and Redlands Greenleaf B.

5.2.9. INOCULATION

In most experiments where a spore suspension in Odourless Mineral Spirit was used, the rate was approximately eight to ten ml to 50 pots situated in an area of 0.5 m^2. In instances where simultaneous infection of individual F2 plants with two different races was desired, the two primary leaves of each plant were inoculated with different races. The tip of the leaf inoculated with one particular race was cut off to distinguish the races. In such instances one or two drops of a spore suspension in Triton B1958^R (0.001%) were spread on each leaf with a Pasteur pipette.

5.2.10. TESTING STRATEGY

5.2.10.1. Crosses with Sanilac

In crosses with Sanilac, relatively little attention was given to F2 segregations while more emphasis was placed on the study of F3 lines.

The races used varied with the particular cross under investigation. For example, in the cross with Brown Beauty, F2 populations and F3 lines were tested with two races avirulent on this cultivar, in two separate experiments.

With cultivars susceptible to at least some Australian races, F2 populations and F3 lines were tested with the simplest race, h. Where more than one gene was detected or predicted, lines apparently segregating monogenically were inoculated with further races specifically selected on the suspicion that they might possess corresponding genes for virulence. These decisions were based on consideration of survey data, ITs and parentages.

Where parental accessions were resistant to all Australian races, approximately 70 F3 lines were tested with race h. Where results were consistent with single-gene segregation, further lines and 20 tested previously, were inoculated with efg, the race most dissimilar to h. This latter group of 20 comprised

five homozygous resistant, five homozygous susceptible and ten lines known to be segregating from the earlier study using race h. If there was complete agreement in reaction to races h and efg, a group or groups of 20 lines were further tested with two or three widely differing races.

Where two or more genes were detected, all lines were screened with race h. Lines apparently segregating monogenically were tested with efg, and subsequently, with other races having one or more virulences in common with this race. The remaining lines were then tested with the race(s) having virulence on seedlings with one or more of these genes.

5.2.10.2. Intercrosses

Testing of F2 populations was adequate for most intercrosses, because data on the responses of seedlings with particular genes were available. These results included ITs, similarity to genes in certain differentials, and where more than one gene was isolated from a cultivar, whether these segregated independently or were linked in coupling.

However, F3 lines of two intercrosses involving cultivars with more than one gene were tested in separate experiments with a range of races virulent for one or more of the host genes.

Crosses between accessions possessing single genes were tested with race h. Crosses between accessions with more than one gene were tested with a race, or races, to which the least number of segregating host genes was predicted from results of the respective crosses with Sanilac.

5.2.11. INTERPRETATION OF RESULTS

The criteria used in interpreting breeding behaviour were firstly ITs and reaction patterns with a range of races, and secondly the closeness of fit of the observed results to models for different types of genetic segregations.

The responses of the F1 hybrid seedlings were compared to those of the parents to determine if the resistance was dominant or intermediate.

Where more than one gene was present and some ITs were clearly different,

incomplete classification was made on the basis of the ITs. However, complete classification was only possible where tests were made with an appropriate range of races which rendered different genes ineffective. This is because the gene with the lowest IT is epistatic to those determining higher ITs. The ITs seen in tests made at different times were not always identical, emphasising the need for inclusion of the parents and possibly other reference cultivars in all tests.

Reaction patterns with a range of races enabled identification of the genes involved in some crosses. For example, where races efg, egh and adehi gave a susceptible reaction and races h, gh, cfh and adhi gave a resistant reaction on certain lines, similarity to a gene(s) in differential e was established.

For single-gene segregations the expected ratio for the F2 segregation is 1:2:1 where the gene lacks dominance or is incompletely dominant. Similarly, the predicted ratio of F3 lines which are homozygous resistant, segregating and homozygous susceptible is 1:2:1. Within the segregating lines, the expectation for pooled numbers of seedlings in IT groups is the same as in the F2 populations.

Where two dominant genes are segregating independently, the expected F2 ratio is 12:3:1 where ITs are clearly different and 15:1 where ITs are similar. The line classification of the F3 lines into disease reaction classes, *viz.* homozygous resistant, segregating and homozygous susceptible was tested for conformity to a ratio appropriate to the classification which the races and ITs permitted. For example, 1:2:1:2:4:2:1:2:1 for complete classification or 4:8:1:2:1 for less complete classification.

Where three dominant genes are segregating independently the expected ratio for the F2 population is 63:1 and where four genes are involved, 255:1.

Where joint segregation ratios differed significantly from expectation and where the linkage component of χ^2 accounted for most of the discrepancy, the recombination value was calculated by the method of maximum likelihood using Tables compiled by Allard (1956).

Where the IT was the same and the classification of F3 lines into disease reaction classes was identical with all races, operation of the same gene was presumed.

5.2.12. FIELD TESTING OF SELECTED F3 LINES

Field experiments were conducted to see if independent sources of adult plant resistance could be found. This was necessary because 13 of the F2 Sanilac/Cornell 49-242 and 14 of the F2 Sanilac/PR 5 plants, which were recorded as resistant in the field during the 1974 - 75 season gave susceptible progenies in greenhouse tests. Twenty seeds of each of six such lines of Sanilac/Cornell 49-242 and seven F3 lines of Sanilac/PR 5 were sown in the 1975 - 76 season and six seeds of the remaining F3 lines of the two crosses were planted in the 1976 - 77 season. The parents were sown as reference cultivars and Sanilac was interspersed through the planting.

Field reactions of selected F3 lines of Sanilac/Bonita were examined to determine if the slow rusting behaviour of Bonita was associated with the factor(s) governing seedling resistance.

5.2.13. DISEASE ASSESSMENTS

Seedling IT ratings were as used in the race survey. Field reactions were recorded on the 0 - 10 system described in Section 3.2.2.2. (p. 12).

5.3. Results

5.3.1. CROSSES WITH SANILAC

5.3.1.1. Accessions susceptible to some Australian races

5.3.1.1.1. Gallaroy

Gallaroy B750 was found to be heterogeneous for rust reaction (Section 4.3.1. p. 21). The two components, designated Genotypes I and II were distinguished by reactions with three groups of races:-

Host	Race		
	h	(a)dhi	adhi
Gallaroy Genotype I	2+2	2+2	3+4
Gallaroy Genotype II	;	2+2	3+4
Sanilac	3+4	3+4	3+4

The two Genotypes were subjected separately to genetic analysis.

Gallaroy Genotype I

The ITs of parents and F1 plants and the frequencies of F2 seedlings in IT classes are shown in Table 9. The phenotypes of F1 plants suggested that the intermediate IT of Gallaroy Genotype I was incompletely dominant and the F2 segregation suggested allelic differences at a single locus. The distribution of F3 lines in reaction classes (Table 10) confirmed that resistance in Gallaroy Genotype I was controlled by a single gene. However, when the pooled segregation ratios for segregating F3 lines within family groups were tested for conformity with 1:2:1 ratios, results for two family groups showed a significant ($P < 0.01$) deviation from expectation (Table 11). This deviation persisted when segregation for all family groups were totalled. However, when IT 2+2 and IT 3- classes were pooled and the resultant frequencies tested for conformity with 3:1 ratios, there was a satisfactory fit. Hence the significant deviations appeared to be associated with difficulty in distinguishing homozygotes and heterozygotes on a single plant basis especially in family group 1.

The gene conferring resistance in Gallaroy Genotype I was tentatively designated *Ur-A*. A line, B1627, derived from a Single Typical Plant of this genotype was selected as a host standard for *Ur-A*. It produced the same IT (2+2 or 2+3-) with races h and (a)dhi and the distribution of reaction classes presented in Table 12 indicates that the same gene was effective against both races. Hence in 643, or differential a, *Ur-A* appeared to be the critical gene on which (a) races were distinguished from a races.

Callaroy Genotype II

Infection types produced by the Gallaroy Genotype II and Sanilac parents, and F1, F2 and F3 offspring are given in Table 13. Six distinctive ITs were observed when F3 plants were inoculated with race h, whereas only three were seen in tests with (a)dhi. With race h, the F1 phenotype indicated dominance of the Callaroy Genotype II resistance whereas with (a)dhi, the F1 phenotype was intermediate. An intermediate IT seen on some F2 seedlings tested with race (a)dhi

Table 9. Seedling frequencies in IT classes for parents, Fls, and F2 populations of Sanilac/Gallaroy Genotype I, infected with race h

Host	IT			χ^2 1:2:1
	22+	2+2	3+4	
Sanilac			6	
Gallaroy Genotype I	6			
F1		2		
F2 1A	10	19	18	4.44
1B	12	15	10	1.54
				5.98
F2 Totals	22	34	28	3.9
Heterogeneity χ^2 (2 d.f.)				2.08

Table 10. Distribution of F3 lines in reaction classes in Sanilac/Gallaroy Genotype I, tested with race h

Family group	Reaction classes			χ^2 1:2:1
	Homozygous resistant	Segregating	Homozygous susceptible	
1A	4	20	5	4.25
2A	5	15	6	0.70
3A	10	12	14	4.89
4A	4	7	0	3.73
Totals	23	54	25	13.57 0.34
Heterogeneity (6 d.f.)				13.23

Table 11. Pooled seedling frequencies in IT classes in segregating F3 lines of Sanilac/Gallaroy Genotype I when tested with race h

Family group	No. of lines	IT classes			X^2 1:2:1	$X^{2\dagger}$ 3:1
		2+2	3-	3-4		
1	20	123	167	105	11.17**	0.67
2	15	91	138	92	6.31*	2.29
3	12	60	104	69	3.37	2.64
4	7	29	68	13	2.73	2.17
					23.58	7.77
	54	303	477	279	12.90**	2.53
Heterogeneity (8 d.f.)					10.68	
Heterogeneity (3 d.f.)						5.24

† IT classes 2+2 and 3- were pooled.

Table 12. Distribution of reaction classes of twenty lines of Sanilac/Gallaroy Genotype I when tested with two races of *C. appendiculatus*

Race (a)dhi	Race h		
	Homozygous resistant	Segregating	Homozygous susceptible
Homozygous resistant	5		
Segregating		10	
Homozygous susceptible			5

Table 13. Infection types of Sanilac, Gallaroy Genotype II and the progeny of crosses between them when inoculated with three races of *U. appendiculatus*

Race	Parents		F1	F2	F3
	Sanilac	Gallaroy Genotype II			
h	3+4	;1	;1	;1,2+2,3-3,3+4	;1,12-,2+2, 2-2+3-3,3-,3+4
(a)dhi (i)	3+4	2+2	3-3	2+2,3-3,3+4	2+2, 3-, 3+4
(ii)	3+4	2+2	-	2+2, 3+4	
adhi	3+4	3+4			

in one test (i) was not observed when another sample (ii) was screened.

F2 tests. Of 55 F2 seedlings infected with race h, five produced IT 3+4, whereas 50 showed one of the alternative ITs. This ratio suggested segregation at two genetically independent loci ($\chi^2_{15:1} = 0.76$, $P > 0.3$). When a further F2 sample was tested with race (a)dhi, the segregation of 14 IT 2+2 : 24 IT 3-3 : 21 IT 3+4 indicated variation at a single locus ($\chi^2_{1:2:1} = 3.74$, $P > 0.1$).

Thus (a)dhi appeared to be virulent for one of the two genes detected with race h. If it does share an avirulence gene with race h, then on the basis of similarity of ITs produced when (a)dhi is used to infect both Gallaroy Genotype I and Gallaroy Genotype II, (text table p. 40) this gene is likely to correspond with *Ur-A*.

F3 tests. F3 lines were separately inoculated with races h and (a)dhi.

Race (a)dhi: As indicated in Table 14, the pooled breeding behaviour of 128 F3 lines infected with race (a)dhi did not conform with expectation for single gene segregation. Family group 2B was deficient in the number of homozygous susceptible lines. However, the pooled segregation ratios for segregating lines within family groups conformed closely with expectation for 3:1 ratios after pooling the IT 2+2 and IT 2+3-3 groups (Table 15). In one experiment with family group 1A, only two

Table 14. Distribution of reaction classes in F3 lines of Sanilac/Gallaroy Genotype II when tested with race (a)dhi

Family group	Reaction			χ^2 1:2:1
	Homozygous resistant	Segregating	Homozygous susceptible	
1A	17	31	13	0.54
2A	11	10	5	4.16
2B	18	15	8	8.86*
				13.56
Total	46	56	26	8.25*
Heterogeneity (4 d.f.)				5.31

Table 15. Frequencies of resistant and susceptible plants in F3 segregating lines in Sanilac/Gallaroy Genotype II when tested with race (a)dhi

Family group	IT			χ^2 1:2:1	χ^2 3:1 †
	2+2	2+3-3	3+		
1A	63	81	50	7.03*	0.07
2A	39	70	36	0.3	00
2B	81	133	78	2.36	0.42
				9.69	
	183	284	164	7.48*	0.33
1A	218		71		0.03
					0.85
	685		235		0.15
Heterogeneity (4 d.f.)				2.21	
Heterogeneity (5 d.f.)					0.70

† IT classes 2+2 and 2+3-3 pooled.

IT classes were distinguishable. Similar pooling of IT groups was necessary in the analysis of Gallaroy Genotype I. Thus, despite the discrepancy from 1:2:1 ratios for line distribution, the close fit to a 3:1 ratio within the segregating lines and in the F2 population suggests that a single gene was involved. On the basis of similarity of ITs, segregation and pedigree this gene appears to be *Ur-A*.

Race h: The breeding behaviour of the 128 lines is summarized in Table 16. On the basis of the types of segregation it was clear that a second gene, designated *Ur-B*, was segregating. Moreover, since *Ur-B* conferred a lower IT than *Ur-A*, it could be classified, even in the presence of *Ur-A*. Hence the frequencies of all nine genotypes with respect to the two loci could be determined. The ratio *Ur-B Ur-B* : *Ur-B ur-B* : *ur-B ur-B* conformed with expectation for segregation at one locus.

In most experiments, *Ur-B* was completely dominant, producing ITs ;1 or 12-. However, in one experiment, a range of pustule sizes, IT 2-22+3-3, occurred on individual plants in lines segregating only for *Ur-B*. Progeny tests of four such plants showed that each was heterozygous, whereas four plants with ITs ;1 and 12- were homozygous for *Ur-B*.

Joint segregation. The χ^2 value for independent segregation of the two loci was highly significant (Table 16). Most of the discrepancy was accounted for by the linkage component. Further data supporting the hypothesis of two segregating genes are shown at the bottom of Table 16. The poor agreements with 15:1 and 12:3:1 ratios resulted from excesses of susceptible seedlings which would be expected with linkage in coupling.

In order to estimate recombination (r) between *Ur-A* and *Ur-B*, the F3 line distribution was substituted in Equation 4, Table 6 of Allard (1956) which is for repulsion linkage *i.e.*

$$(Ur\text{-}A\ Ur\text{-}A\ Ur\text{-}B\ Ur\text{-}B + ur\text{-}A\ ur\text{-}A\ ur\text{-}B\ ur\text{-}B)\frac{2}{r} + (Ur\text{-}A\ ur\text{-}a\ Ur\text{-}B\ Ur\text{-}B + Ur\text{-}A\ Ur\text{-}A\ Ur\text{-}B\ ur\text{-}B + Ur\text{-}A\ ur\text{-}a\ ur\text{-}B\ ur\text{-}B + ur\text{-}A\ ur\text{-}A\ Ur\text{-}B\ ur\text{-}B)\left[\frac{1-2r}{r(1-4)}\right] + (Ur\text{-}A\ Ur\text{-}A\ ur\text{-}B\ ur\text{-}B + ur\text{-}A\ ur\text{-}A\ Ur\text{-}B\ Ur\text{-}B)\left(\frac{2}{r-1}\right) + (Ur\text{-}A\ ur\text{-}A\ Ur\text{-}B\ ur\text{-}B)\left(\frac{2(2r-1)}{1-2r+2r^2}\right) = 0$$

Table 16. Genotype distribution and ITs of F3 lines of S[illegible]ilac/Gallaroy Genotype II when tested with two races of *U. appendiculatus*

Presumed genotype from tests with (a)dhi	Frequency	ITs observed with race [illegible]	Frequency	Genotype at second locus
Ur-A Ur-A	46	;1,12-	28	*Ur-B Ur-B*
		;1,12-	17	*Ur-B ur-B*
		;1,12-	1	*ur-B ur-B*
Ur-A ur-A	56	;1,12-,2+2	7	*Ur-B Ur-B*
		;1,12-2-22+3-,2+2,3+4	42†	*Ur-B ur-B*
		;1,12-,2-22+3-, 3+4	7	*ur-B ur-B*
ur-A ur-A	26	2+2	1	*Ur-B Ur-B*
		2+2,3-, 3+4	12	*Ur-B ur-B*
		3+4	13	*ur-B ur-B*
	128		128	

Pooled segregation ratios

$\chi^2_{1:2:1}$ *Ur-A Ur-A:Ur-A ur-A: ur-A ur-A* 46:56:26 = 8.26*

$\chi^2_{1:2:1}$ *Ur-B Ur-B:Ur-B ur-B:ur-B ur-B* 36:71:21 = 4.05

χ^2 Heterogeneity (linkage) (4 d.f.) = 65.39**

$\chi^2_{1:2:1:2:4:2:1:2:1}$ Joint segregation 77.70**

† Pooled frequencies of resistant and susceptible seedl[illegible]s

$\chi^2_{12:3:1}$ 536 IT;1,12-,2-22+3-3:125 IT2+2+3-:138 IT3+4 = 166.24**

$\chi^2_{15:1}$ 661 IT;1,12-2-22+3-3,2+2+3-:138 IT3+4 = 207.04**

$$41 \times \frac{2}{0.75} - \frac{43 \times 0.5}{0.75 \times 0.25} - \frac{4}{0.25} + \frac{42 \times 0.5}{1 - 1.5 + 2 \times 0.75 \times 0.75} = 0$$

$r = 0.75$

Coupling linkage r', was estimated by subtraction from unity, *i.e.* $r' = 1 - 0.75 = 0.25$.

The variance (V) of r is given by $\frac{1}{I}$ where I = ni and n = total population size, i = information (Table 8 of Allard 1956).

$I = 128 \times 1.046$

$V = \frac{1}{I} = \frac{1}{133.89}$

$\sqrt{V} = s = \sqrt{\frac{1}{I}} = 0.09$

i.e. $r' = 25 \pm 9\%$

In order to derive a homozygous line possessing only *Ur-B*, 12 F3 plants in one line of genotype *ur-A ur-A Ur-B ur-B* were progeny tested. When inoculated with race h, the ratio of three homozygous resistant: five segregating: five homozygous susceptible lines conformed with a segregation pattern of 1:2:1 ($\chi^2 = 1.31$, $P>0.5$). The pooled frequencies of resistant and susceptible seedlings in the five segregating lines also supported the single gene hypothesis (Table 17). When separate inoculations of 2, 4 and 5 of these lines were made with races dh, cfh and efg, respectively, the lines behaved identically in all instances. The pooled frequencies of resistant and susceptible seedlings again indicated single-gene segregation.

Line 137.16.6 was chosen as a single gene stock for *Ur-B* and accessioned as B2090. Infection types with a range of races (Table 18) suggest that *Ur-B* is the critical gene in differential i (Redlands Greenleaf C) on which races with low, intermediate and high ITs may be distinguished. However, the intermediate ITs produced by the (d)(i) group of races on i indicate the possibility of at least one other gene in this differential. The similarity of ITs of 643 (differential a) and Gallaroy Genotype II in tests with races h, (a)dhi and adhi indicate that

Table 17. Pooled frequencies of resistant and susceptible seedlings in F4 lines segregating for *Ur-B* in Sanilac/Gallaroy Genotype II

Race	No. of lines	Reaction		χ^2 3:1
		Resistant	Susceptible	
h	5	75	22	0.28
dh	2	26	7	0.25
cfh	4	63	21	0.00
efg	5	46	23	2.54
				3.07
		220	73	0.001
χ^2 Heterogeneity (3 d.f.)				3.07

Table 18. Typical ITs of five host genotypes when tested with five races of *U. appendiculatus*

Race	Sanilac	a	Gallaroy Genotype I	Gallaroy Genotype II	B2090†	i
h	3+4	;1	2+3-	;1	;1	;1
(a)dhi	3+4	2+3-	2+3-	2+3-	3+4	3+4
(a)(d)h(i)	3+4	2+3-	2+3-	2+3-	3+4	22+3-3
adhi	3+4	3+4	3+4	3+4	3+4	3+4
a(d)h(i)	3+4	3+4	3+4	3+4	3+4	22+3-3

† Single gene stock for *Ur-B*.

Ur-B is present in 643, a parent of both Gallaroy and Redlands Greenleaf C.

The assumption that Gallaroy Genotype II carried *Ur-A* was based on consideration of the similarity of ITs on resistant segregates of the Sanilac/Gallaroy Genotype II cross when inoculated with races h and (a)dhi. Although the cross between Gallaroy Genotypes I and II was not made, their close relationship and the correlated behaviour of Gallaroy Genotypes I and II in tests with many races strongly support

this contention.

5.3.1.1.2. Brown Beauty

When tested with race efg, F1 plants produced IT 22+2-NR22+2- , which was intermediate between those of Brown Beauty with IT 2-NR2- and Sanilac with IT 3+4. F2 seedlings segregated 8 IT 2-NR 2- : 7 IT 22+2-NR22+2- : 3 IT 3+4 ($\chi^2_{1:2:1}$ = 3.66, P>0.05).

In the experiment with F3 populations, ITs of resistant plants ranged from 22-NR 22- to 2+3-3 but subdivision within this group was not possible. The line distribution of 8 homozygous resistant : 15 segregating : 2 homozygous susceptible indicated segregation at a single locus ($\chi^2_{1:2:1}$ = 3.88, P>0.1). Classifications with races af and efg were in complete agreement indicating that the same host gene, tentatively designated *Ur-C*, conferred resistance to both races. Pooled frequencies of resistant and susceptible seedlings (Table 19) conformed with single-gene segregation only in the tests with race af. Temperature sensitivity of the reaction, as reported in Section 4.3.3. (p. 25), may have caused the excess of susceptible plants in tests with efg. The resistance of some heterozygotes may have been more clearly expressed under warmer conditions when the tests with race af were conducted. However, this does not account for the excess of susceptible seedlings in only two of the three family groups. There were consistent excesses of susceptible plants in most of the segregating lines of family groups 2 and 3 in tests with efg.

There was no indication of linkage between rust reaction and coloured seed coat.

5.3.1.1.3. Redlands Greenleaf B

Races used. The ITs produced by Redlands Greenleaf B and current accessions of the parents as well as the *Ur-A* and *Ur-B* single gene stocks (Table 20) suggest that this cultivar may carry one or more genes derived from 643 and *Ur-C* derived from Brown Beauty. The IT 3+4 produced in tests with (a)dhi, rather than ITs 2+3- or ; indicates that *Ur-A* and *Ur-B*, are not present. The occurrence of races giving

Table 19. Frequencies of resistant and susceptible plants in segregating F3 lines of Sanilac/Brown Beauty, when tested with two races of *U. appendiculatus*

Race	Family group	No of lines	Reaction		χ^2 3:1
			Resistant	Susceptible	
af	1	6	63	33	4.52
	2	4	41	19	1.4
	3	5	61	11	3.63
					9.55
			165	63	0.84
Heterogeneity (4 d.f.)					8.71
efg	1	6	80	31	0.67
	2	4	40	30	11.96**
	3	5	51	36	12.45**
					25.08
			171	97	17.91**
Heterogeneity (4 d.f.)					7.17

Table 20. Typical ITs produced by Redlands Greenleaf B, 643, Brown Beauty, Sanilac and single gene stocks of *Ur-A* and *Ur-B* when tested with eight races of the bean rust fungus

Race	Redlands† Greenleaf B	643‡	Brown Beauty§	*Ur-B*	*Ur-A*	Sanilac
h	;	;	3+4	;1	2+2	3+4
dh	3+4	;	3+4	;1	2+2	3+4
(a)dhi	3+4	2+2	3+4	3+4	2+2	3+4
adhi	3+4	3+4	3+4	3+4	3+4	3+4
(a)(d)h(i)	22+3-3	2+2	3+4	3+4	2+2	3+4
a(d)h(i)	22+3-3	3+4	3+4	3+4	3+4	3+4
af	2-NR2-	3+4	2-NR2	3+4	3+4	3+4
efg	;	;	2-NR2	;1	2+2	3+4

= Redlands Greenleaf B.

† differential d (Brown Beauty/643//Brown Beauty 17A).

‡ differential a.

§ differential h.

an intermediate IT on Redlands Greenleaf B indicates either the presence of an additional gene conferring an intermediate IT in the host, or three phenotypes of the pathogen with respect to one of the genes. The origin of a postulated additional gene with an intermediate IT is not clear.

Race h was used to classify the gene(s) derived from 643. Race (a)(d)h(i) was used to test for the additional gene conferring an intermediate IT. Race af was chosen to test for *Ur-C*. Because of avirulence for *Ur-C* and virulence for the gene(s) derived from 643, complete classification was expected. Race efg, used only on F1 plants and selected F3 lines, was used as an additional tester for *Ur-C*. As it was avirulent for the gene(s) derived from 643, complete classification was expected only for the lines lacking the gene(s) from 643.

Infection types. Infection types produced by the Redlands Greenleaf B and Sanilac parents and their F1, F2 and F3 progenies when inoculated with the four races are given in Table 21. The reactions of F1 plants with races h and efg were dominant, while those with races af and (a)(d)h(i) were intermediate.

F2 tests. When 63 seedlings were infected with race h, 51 produced IT ; and 12 showed IT 3+. This ratio suggested a single gene ($\chi^2_{3:1} = 1.16$, $P>0.2$).

In tests with race (a)(d)h(i), 19 seedlings showed IT 2-22+, 19 IT 2+3-3 and 16 IT 3+. This ratio gave marginal agreement with a 1:2:1 ratio ($\chi^2 = 5.07$, $P<0.05$), but closer agreement to a 3:1 ratio was obtained when the two resistant classes were pooled ($\chi^2_{3:1} = 0.58$, $P>0.3$).

In tests with race af, 24 seedlings produced ITs in the range 2-NR2- to 2NR2 , 16 IT 22+2- and 5 IT 3+. When the numbers in the two resistant classes were pooled, the ratio suggested segregation at two genetically independent loci ($\chi^2_{15:1} = 1.84$, $P>0.1$; $\chi^2_{3:1} = 4.63$, $P<0.05$). This suggested that *Ur-C* was present, together with an additional gene giving an intermediate IT. As race af is virulent on 643, an additional gene not present in current accessions of that stock or in Brown Beauty must be postulated.

Table 21. Infection types produced by Sanilac, Redlands Greenleaf B, their progeny and current accessions of Brown Beauty and 643, when tested with four *U. appendiculatus* races

Race	Parents		F1	F2	F3	Brown Beauty	643
	Sanilac	Redlands Greenleaf B					
h	3+4	;	;	;, 3+	;, ;1, 3+	3+3+4	;
(a)(d)h(i)	3+4	22+3-3	2+3-3	2-22+, 2+3-3, 3+	22+3-3, 2+23-33+, 3+	3+3+4	2+2
af	3+4	2-NR2	22-NR22-	2-NR2-, 2NR2, 22+3-, 3+	2-NR2-, 2-2, 22+3-3, 3+	2-NR2	3+
efg	3+4	;			2-NR2, 2NR2, 3+	2-NR2	;

These results supported the suggestion that Redlands Greenleaf B carries a minimum of two genes, one derived from 643, a second from Brown Beauty, and possibly a third gene of unknown origin.

21 : 54 : 20

F3 tests. Race h. Twenty-one lines were homozygous resistant, 54 segregated and 20 were homozygous susceptible, confirming segregation at a single locus ($\chi^2_{1:2:1}$ = 1.18, P>0.3). The summed frequencies of 761 resistant : 265 susceptible plants in the 54 segregating lines again supported the single-gene hypothesis ($\chi^2_{3:1}$ = 0.38, P>0.5). The dominant allele, tentatively named *Ur-D*, is distinct from *Ur-A* and *Ur-B*, and is also apparently derived from 643.

Race (a)(d)h(i). Although 95 lines had been tested with race h, sufficient seed remained to screen only 84 with race (a)(d)h(i). Twenty were homozygous resistant, 28 segregated and 16 were homozygous susceptible, indicating segregation at a single locus ($\chi^2_{1:2:1}$ = 2.1, P>0.7).

Eighty-one lines reacted similarly with races h and (a)(d)h(i) but three, whose reactions are given in Table 22, behaved differently. The different results with the races h and (a)(d)h(i) suggest linked genes at two loci, one gene, *Ur-D* conferring IT ; and the other, *Ur-Red*, conferring IT 22+3-3.

Race af. The distribution of disease reactions of the three family groups given in Table 23 differed considerably. While family group 1A conformed satisfactorily to both a 1:2:1 ratio, expected for a one-locus segregation and 7:8:1 and 15:1 ratios expected for two-locus segregations, 3A conformed to a 15:1 ratio, but not to either 1:2:1 or 7:8:1 ratios. Group 2A conformed to a 15:1 ratio, but not to 1:2:1 or 7:8:1 ratios. The family groups gave homogeneous results on the expectation of a 1:2:1 ratio, but not when 7:8:1 or 15:1 ratios were considered.

The pooled frequencies of resistant plants in the segregating F3 lines are given in Table 24. Even though the frequencies for group 2A conformed satisfactorily with a single-gene hypothesis, those for groups 1A and 3A and the totals did not conform with this hypothesis.

Table 22. Reactions of three F3 lines of Sanilac/Redlands Greenleaf B to four *U. appendiculatus* races, and possible genotypes

Line	Race											
	h			(a)(d)h(i)			af			efg		
	Res. : Sus.		Genotype	Res. : Sus.		Genotype	Res. : Sus.			Res. : Sus.		Genotype
IT	; 3+			22+3-3+ 2+23-33+	3+		2-NR, 2-2+ 22+3-3	3+		2-NR2+ 2NR2	3+	
131.10	13 : 6		*Ur-D ur-D*	(i)§ 19 : 0		*Ur-Red Ur-Red*	(i) 15 : 2			19 : 0		not clear
				(ii) 16 : 0			(ii) 12 : 5					
				35 : 0			27 : 7					
131.16†	0 : 20		*ur-D ur-D*	10 : 0		not clear	22 : 2					*Ur-C ur-C*?
132.8ǂ	16 : 4		*Ur-D ur-D*	0 : 18		" "	10 : 8					*Ur-C ur-C*?

† no residue seed.

ǂ limited seed, grown on for progeny tests.

§ (i) and (ii) results are of tests at different times.

Table 23. Distribution of F3 lines in reaction classes in Sanilac/Redlands Greenleaf B when tested with race af

Family group	Reaction			χ^2 1:2:1	χ^2 7:8:1	χ^2 15:1
	Homozygous resistant	Segregating	Homozygous susceptible			
1A	10	21	3	4.76	3.25	0.38
2A	4	19	0	11.17**	9.97**	1.54
3A	9	19	7	0.49	13.19**	10.81**
				16.42	27.41	12.73
	23	59	10	11.02*	14.21**	3.34
χ^2 Heterogeneity (4 d.f.) =				5.40		
χ^2 Heterogeneity (4 d.f.) =					13.20*	
χ^2 Heterogeneity (2 d.f.) =						9.39**

Table 24. Pooled frequencies of resistant and susceptible plants in segregating F3 lines of Sanilac/Redlands Greenleaf B when tested with race af.

Family group	Reaction		χ^2 3:1
	Resistant	Susceptible	
1A	315	82	4.0*
2A	275	88	0.11
3A	363	90	6.36*
			10.47
	953	260	8.22**
Heterogeneity (2 d.f.)			2.25

The F2 data supported a two-locus hypothesis. However, though not conclusive, the F3 results indicated closer agreement with a single gene.

The IT data presented in Table 21 suggest that *Ur-C*, present in Brown Beauty, is also present in Redlands Greenleaf B. While the IT 22+3-3 suggests that *Ur-Red* is present also, this is not clear. Linkage between *Ur-C* and *Ur-Red* may explain the ITs and ratios.

There was sufficient seed to test only 91 lines with race af, and 84 with both af and (a)(d)h(i). Seventy lines produced similar reactions with all three races, but the 11 F3 and two F4 lines listed in Table 25 and the three lines listed in Table 22 reacted differently. Eight of these were homozygous resistant with both races h and (a)(d)h(i) but segregated in tests with race af. The gene *Ur-C* may have been responsible for the segregation observed in tests with race af. The homozygous resistant reactions in inoculations with (a)(d)h(i) and segregation with af demonstrate that the gene being detected by race af is not *Ur-Red*. This is confirmed by the susceptibility to race af of the other three F3 lines and two F4 lines listed in Table 25. These lines segregated with races h and (a)(d)h(i). The 2:18 ratio of 131.23.2 in tests with race (a)(d)h(i) may be attributed to difficulty in distinguishing plants heterozygous for *Ur-Red* from those with *ur-Red*. Differences between resistant and susceptible seedlings were small and reactions difficult to classify.

The results of line 131.10 (Table 22) with races (a)(d)h(i) (19:0), af (15:2) and efg (19:0) are difficult to explain. The reaction of 19:0 with race efg indicates that *Ur-C* may be present. Hence the segregation with race af cannot be accounted for on the basis of factors previously encountered.

While some evidence, namely IT data, indicates that the phenotype of the culture of race af (75.88) used throughout this study is *P-C P-Red*, the discrepancies between line classification in tests with races af and (a)(d)h(i) suggest that the phenotype is *P-C p-Red*. Either culture 75.84, race a, or 75.89, race af, may assist in resolving this difficulty if either were clearly shown to be *P-C p-Red*.

Table 25. Reactions of 11 F3 lines and two F4 lines with races h, (a)(d)h(i) and af and postulated genotypes

Line	Race											
	h				(a)(d)h(i)				af			
	Hom. Res.	Seg Res:Sus	Hom. Sus.	Genotype	Hom. Res.	Seg Res:Sus	Hom Sus.	Genotype	Hom. Res.	Seg Res:Sus	Hom. Sus.	Genotype
132.9	20			*Ur-D Ur-D*	20			*Ur-Red Ur-Red*		(i)† 15:4 (ii)‡ 19:1		*Ur-C Ur-C?* " "
132.12	19			" "	20			" "		(i) 13:6 (ii) 15:4		" "
132.14	19			" "	20			" "		(i) 9:10		" "
132.16	20			" "	-			" "		(i) 12:8		" "
132.17	20			" "	20			" "		(i) 11:4 (ii) 11:9		" "
133.2	20			" "	20			" "		(i) 13:5 (ii) 15:5		" "
133.10	20			" "	20			" "		(i) 16:4 (ii) 12:7		" "
133.14	19			" "	18			" "		(i) 15:3		" "
317.3		18:2		*Ur-D ur-D*		11:7		*Ur-Red ur-Red*			20	
133.25		17:2		" "		15:4		" "			20	
319.2		15:4		" "		13:4		" "			20	
131.23.1		13:6		" "		17:3		" "			19	
131.23.2		14:4		" "		2:18		" "			16	

†(i) first test.

‡(ii) repeat test.

However, there was insufficient seed for such tests. This illustrates the lack of precision which occurs in race determination when differentials carrying more than one gene are used, particularly when the ITs are similar as with *Ur-C* and *Ur-Red*.

Race efg. Sufficient seed remained to test with race efg only 15 of the 20 lines homozygous susceptible with race h. The results were generally in agreement with predictions made on the basis of tests with race af, except for line 131.10, which segregated with af but was resistant with efg. Six lines were homozygous resistant and nine segregated when infected with efg, a distribution which differed significantly from 1:2:1 ($\chi^2 = 5.4$, $P<0.05$). Linkage in coupling between *Ur-C* and *Ur-Red* would yield a significant excess of homozygous susceptible lines, so their absence was attributed to sampling error.

In order to derive a homozygous line possessing only *Ur-D* or *Ur-D* plus *Ur-Red*, F3 plants of 131.23 were grown on for progeny-testing. Of the six plants which survived to produce seed, three lines segregated and three were homozygous susceptible when tested with race h. This sample is too small to test for agreement with a 1:2:1 ratio. Sixteen plants of one F4 line were progeny-tested. Five were homozygous resistant, five segregated and six were homozygous susceptible, a distribution which conformed with 1:2:1 ($\chi^2 = 2.37$, $P>0.3$). The reactions of 14 lines tested with race efg and of four lines tested with race (a)(d)h(i) agreed with the reactions for race h. In the tests with race (a)(d)h(i), an intermediate IT typical of *Ur-Red* was observed.

Line 131.23.2.4 accessioned as B2113, was chosen as a reference stock for the combination of *Ur-D* and *Ur-Red*.

Thus Redlands Greenleaf B, differential d, carries *Ur-D* which it apparently shares with differential a, *Ur-C* in common with differentials h and d and possibly an additional gene *Ur-Red*, presumably closely linked with *Ur-D*. The intermediate IT on differential i of all the cultures intermediate on d suggests that *Ur-Red* is present also in Redlands Greenleaf C, differential i. Neither *Ur-D* nor *Ur-Red*

was isolated as a single gene line.

The gene *Ur-D* is the critical factor in Redlands Greenleaf B on which races producing IT ; are distinguished from others. As all races virulent for *Ur-B* are also virulent for *Ur-D*, the suggestion that *Ur-D* is present in 643 is based on the parentage of Redlands Greenleaf B. Unless a culture virulent for *Ur-B* and avirulent for *Ur-D* can be isolated, a genetic study of the current accession of 643, B1554, will be necessary to demonstrate the presence of *Ur-D*.

5.3.1.2. Accessions resistant to all known Australian races

5.3.1.2.1. Actopan/Sanilac Selection 37.

When inoculated with race h, Actopan/Sanilac Selection 37 produced IT ;, the F1s of the cross with Sanilac, IT ;1 and the F2s, ITs ;1, 12- and 3+4, indicating near dominance of the low IT. The F2 segregation of 57 resistant: 6 susceptible showed a satisfactory fit to a 15:1 ratio (χ^2 = 1.27, P>0.2). With race efg, the ITs were similar, but the ratio of 30 resistant : 7 susceptible conformed more closely to segregation of alleles at a single locus ($\chi^2_{3:1}$= 0.73, P>0.1) than at two independently inherited loci ($\chi^2_{15:1}$= 10.15, P<0.01).

One-hundred-and-six F3 lines were inoculated separately with the above cultures. With race efg, the line distribution given in Table 26 confirmed that a single gene was segregating.

All lines homozygous resistant with race efg were also homozygous resistant with race h, lines segregating with efg were either segregating or homozygous resistant with race h and lines susceptible with efg were distributed 7 homozygous resistant : 16 segregating : 6 homozygous susceptible with race h ($\chi^2_{1:2:1}$= 0.33, P>0.7). The total segregation ratio of 236 resistant and 78 susceptible plants in the 16 segregating lines when screened with race h, further indicated segregation at a single locus ($\chi^2_{3:1}$= 0.004, P>0.9). However, the pooled frequencies of 730 resistant : 207 susceptible in the 18 lines classified as segregating monogenically with both races h and efg differed marginally from expectation ($\chi^2_{3:1}$= 4.18, P<0.05). The pooled segregation ratio of 764 resistant : 46 susceptible for the 16 lines

Table 26. Genotypic classification of F3 lines of Sanilac/Actopan/Sanilac Selection 37 when tested with races h and efg

Race efg	Reaction classes		No. of lines	Seedling frequencies in segregating lines	χ^2	Postulated genotype
	No. of lines	Race h		Res:Sus		
Hom. res.	27		27			*Ur-E Ur-E*
Seg.	50	not classified	4			*Ur-E Ur-E*
		Hom. res.	12			*Ur-E ur-E* *Ur-F Ur-F*
		Seg. 15:1	16	764:46	0.34	*Ur-E ur-E* *Ur-F ur-F*
		3:1	18	730:207	4.18*	*Ur-E ur-E* *ur-F ur-F*
Hom. sus.	29	Hom. res.	7			*ur-E ur-E* *Ur-F Ur-F*
		Seg. 3:1	16	236:78	0.004	*ur-E ur-E* *Ur-F ur-F*
		Hom. sus.	6			*ur-E ur-E* *ur-F ur-F*

$\chi^2_{2:1}$ †*Ur-E ur-E:ur-E ur-E* 40:29 = 0.48

$\chi^2_{1:2:1}$ *Ur-F ur-F:Ur-F ur-F* 19:32:24 = 2.28

χ^2 Heterogeneity (linkage) (2 d.f.) = 4.36

$\chi^2_{2:4:1:2:1}$ Joint segregation 12:34:7:16:6 = 7.72

† The incomplete classification permitted only certain classes to be included in the analysis.

apparently segregating for both genes fitted a two-locus segregation hypothesis ($\chi^2_{15:1} = 0.34$, $P>0.5$).

These results indicated that race h was virulent for genes tentatively designated *Ur-E* and *Ur-F*, but race efg was avirulent only for *Ur-E*.

The postulated genotypes of the 106 lines are included in Table 26. Since seedlings possessing both *Ur-E* and *Ur-F* produced similar ITs when inoculated with race h, only those lines segregating or susceptible with race efg could be classified genetically for the *Ur-F* locus.

In order to derive homozygous lines possessing only *Ur-E* or *Ur-F*, approximately 20 F3 plants in two *ur-E ur-E Ur-F ur-F* lines and one *Ur-E ur-E ur-F ur-F* line were progeny tested. The results presented in Table 27 indicated that a single gene was involved in each instance.

Ur-E. The distribution of disease reaction among the F4 progeny of the line segregating only for *Ur-E* showed close agreement with a 1:2:1 ratio (Table 27). However, while the individual tests with different races conformed to 3:1 ratios, the totals differed significantly from this ratio at P = 0.05. Misclassification is unlikely to be the cause of the deficiency of susceptible plants as the ITs ;, 12- and 3+4 should have been readily and accurately classified. Except for the test with race gh where there was exact agreement with a 3:1 ratio, there was a consistent deficiency of susceptible seedlings, both in the tests on F4 progeny (Table 27) and in the F3 lines segregating for this gene (Table 26). There was identical line classification with races h, gh, cfh, efg and adehi, indicating that the same gene was detected in all tests.

All plants classified as resistant in the field were classified as *Ur-E Ur-E* or *Ur-E ur-E* in greenhouse tests and all plants classified as susceptible in the field were susceptible in the greenhouse. Thus this gene confers field protection in the Sanilac background.

Ur-F. As shown in Table 27, the line classification and the pooled frequencies of resistant and susceptible seedlings in segregating lines conformed closely with a

Table 27. Distribution of disease reaction classes in F4 lines of Sanilac/ / Actopan/Sanilac Selection 37 and frequencies of resistant and susceptible seedlings in segregating lines when tested with widely differing races

Gene	Line	Race	Reaction			χ^2 1:2:1	Pooled seedling frequencies	χ^2 3:1
			Hom. res.	Seg.	Hom. sus.		Res:Sus	
Ur-E	141.21	h	6	8	5	0.59	127:30	2.91
		gh					57:19	0
		cfh					62:14	1.76
		adehi					137:34	2.39
		Totals					383:107	2.53
Ur-F	140.5	h	4	7	5	0.38	101:44	2.21
		gh						
		cfh						
	140.21	h	5	11	3	0.90	158:55	0.03
		gh						
		cfh						
		Totals	9	18	8	0.09	259:99	1.35

single-gene hypothesis in tests with races h, gh and cfh. There was complete agreement in reaction of all lines.

Line 141.21.5 was chosen as a single gene stock for *Ur-E* and accessioned as B2055. Line 140.5.1 was chosen as the *Ur-F* stock and accessioned as B2054. Plants carrying *Ur-F* were susceptible to races efg, egh and adehi. Thus on the basis of reaction patterns and the ITs produced, this line behaved identically to differential e. This differential, CCGB 44 B1558, was not genetically analysed in this study, but on the basis of these results, appears to possess a single gene for resistance to the cultures avirulent on differential e. Plants possessing *Ur-E* were resistant to the five cultures with which they were tested. As Actopan/ Sanilac Selection 37 was resistant to all rust collections sampled, including e-virulent races, *Ur-E* is presumed to be effective against all Australian

races, and is thus distinguished from *Ur-A*, *Ur-B*, *Ur-C*, *Ur-D*, *Ur-Red* and *Ur-F*.

5.3.1.2.2. PR 5

The F1 plants of Sanilac/PR 5 showed IT ; and individual F2 and F3 segregates, ITs ;, 12-, 2, 2+3- and 3+4, indicating near dominance. The F2 ratio of 54 resistant : 10 susceptible seedlings suggested segregation at a single locus ($\chi^2_{3:1}$ = 3.0, P>0.05). The F3 line behaviour in six family groups is summarized in Table 28. Family group 2A showed a significant deviation from the distribution expected on the basis of variation at a single locus. However, when the segregation ratios for the five segregating lines were pooled and tested for conformity with a 3:1 ratio (Table 28), there was a close fit. Hence the abnormal distribution of lines in 2A was attributed to sampling error.

The identical line classification of family group 1C when tested with races h and efg and of the group of 20 lines when tested with races cfh and adehi suggested that the same gene conferred resistance to all races. The pooled frequencies of resistant and susceptible seedlings in the segregating lines tested with races cfh and adehi again suggested single-gene segregation (Table 28).

The gene in PR 5, tentatively designated *Ur-G*, was distinguished from *Ur-A*, *Ur-B*, *Ur-C*, *Ur-D* and *Ur-F* on the basis of reactions with different races, but could not be distinguished from *Ur-E*.

The seedling-susceptible F3 lines derived from 14 F2 plants recorded as resistant in the field during 1974-75 were susceptible in subsequent field trials. The freedom from rust shown by the 14 plants was attributed to a low level of disease early in the season.

Ur-G was inherited independently of genes conditioning bush habit and stem colour.

5.3.1.2.3. Cornell 49-242

The IT of F1 plants of Sanilac/Cornell 49-242 was the same as that of Cornell 49-242, *i.e.* ;1, when tested with race h, indicating dominance of resistance. The F2 plants produced ITs ;1 and 3+4 and F3 lines screened in another experiment

Table 28. Distribution of disease reaction classes and pooled segregation ratios of F3 lines of Sanilac/PR 5 when tested with a range of races of *U. appendiculatus*

Family group	Race	Reaction classes	$\chi^2_{1:2:1}$	Pooled frequencies in segregating lines	$\chi^2_{3:1}$
		Hom. res. : Seg. : Hom. sus.		Res. : Sus.	
1A	efg	3 : 11 : 5	0.89	156 : 60	0.89
1B	efg	8 : 7 : 4	3.0	107 : 34	0.05
1C	h,efg	5 : 10 : 8	1.18	309 : 87	1.95
1D	h	6 : 16 : 2	4.00	254 : 64	4.02
1E	h	5 : 11 : 8	0.92	167 : 59	0.25
2A	h	4 : 5 : 10	8.05**	86 : 24	0.52
			18.04		
Totals		31 : 60 : 37	1.06		
Various	cfh			149 : 47	0.11
	adehi			141 : 50	0.14
					7.93
				1369 : 425	
Heterogeneity (10 d.f.)			16.89		
Heterogeneity (8 d.f.)					6.29

showed ITs 12-, 2-1 and 3+4.

The F2 ratio of 43 resistant : 16 susceptible plants in tests with race h conformed with expectation for single-gene segregation ($\chi^2_{3:1}$= 0.15, P>0.5). This hypothesis was confirmed by F3 line classification of 32 homozygous resistant : 57 segregating : 29 homozygous susceptible ($\chi^2_{1:2:1}$= 0.23, P>0.8).

When races gh, efg, cfh and adehi were inoculated onto different groups of 20 lines, the results agreed in each instance with those from the prior inoculation with race h. This suggested that the same gene conferred resistance to all races.

Table 29 presents the pooled frequencies of resistant and susceptible seedlings in segregating F3 lines when tested with the various races. The results for race adehi, the subtotal for race h and the grand total showed significant deviations from expectations for 3:1 ratios. However, the data were homogeneous and it was concluded that a single gene tentatively designated *Ur-H* was involved.

Table 29. Pooled frequencies of resistant and susceptible seedlings in segregating F3 lines of Sanilac/Cornell 49-242 when tested with various *U.appendiculatus* races.

Race	Family group	Reaction Resistant:Susceptible	$\chi^2_{3:1}$
h	1A	192: 49	2.80
	2A	112: 32	0.59
	2B	100: 24	2.11
	2C	118: 38	0.04
	2D	126: 28	3.81
	2E	233: 61	1.86
	Subtotals		11.21
		881:232	10.34**
Heterogeneity (5 d.f.)			0.87
gh	several	152: 48	0.11
cfh	several	139: 46	0.001
efg	several	138: 62	3.84
adehi	several	162: 36	4.59*
	Subtotals		8.54
		591:192	0.06
Heterogeneity (3 d.f.)			8.48
Grand total			19.75
		1472:424	7.03**
Heterogeneity (9 d.f.)			12.72

The IT produced by beans carrying *Ur-H* was similar to that shown by plants possessing *Ur-E* and *Ur-G*, which were also effective against all Australian races.

The seedling-susceptible F3 lines derived from 13 F2 plants recorded as resistant in the field during 1974-75 were susceptible in subsequent field trials. The freedom from rust shown by the 13 plants was attributed to a low level of disease early in the season.

No linkage was detected between rust reaction, growth habit and stem colour.

5.3.1.2.4. NEP 2

F1 and F2

The ITs of the parents, F1 and F2 plants inoculated with races h, efg and egh are presented in Table 30. The resistance of F1 plants was dominant and the range of ITs in F2 plants suggested the involvement of more than one gene.

The ratios of resistant and susceptible plants in three double inoculation experiments on F2 populations are given in Table 31, together with the corresponding ratios from testing 130 random F3 lines separately with the three races. In experiments (i) and (ii) races h and egh were applied, one race to each of the two primary leaves as outlined in Section 5.2.7. (p. 36), and in (iii) races h and efg were used. Some seedlings which gave a resistant reaction to race h on one primary leaf produced a susceptible reaction to race efg or egh inoculated onto the other leaf.

The pooled frequencies of resistant and susceptible plants in the tests with race h gave closer agreement with a three-gene than with a four-gene hypothesis. However, the ratio of the F2 tests with this race gave satisfactory agreement with both a three-gene and a four-gene hypothesis. The F2 and F3 tests with race egh and the totals showed closer agreement with a two-gene than a three-gene hypothesis. Similarly, in the tests with race efg there was satisfactory agreement with a two-gene hypothesis. All plants susceptible to race h were also susceptible to races efg or egh. Similarly, where classifications were made by testing F3 lines with the three races, all plants susceptible to race egh were also susceptible

Table 30. Infection types of Sanilac, NEP 2 and the hybrid progeny when inoculated with races h, egh and efgj

Race	Parents		F1	F2
	Sanilac	NEP 2		
h	3+4	;N	;N	(i) ;N, 2PA/2PA, 2-, 3+4 (ii) ;N, 2, 2-, 12-, 3+4 (iii) ;N, 2-/2-WA, 12-, 3+4
egh	3+4	;N	;N	(i) ;N, 2PA/2PA, 22-NR, 2-, 3+4 (ii) ;N, 2, 2-, 22-NR, 3+4
efg	3+4	;N	;N	(iii) ;N, 2-/2-WA, 3+4

Table 31. Frequencies of resistant and susceptible plants in F2 populations of Sanilac/NEP 2, including F2 classifications derived from F3 testing

Generation	Test	Race							
		h			egh			efg	
		Res:Sus.	χ^2 255:1	χ^2 63:1	Res:Sus	χ^2 63:1	χ^2 15:1	Res:Sus	χ^2 15:1
F2	(i)	69: 2			64: 7				
	(ii)	108: 0			104: 4				
	(iii)	103: 1						93:11	
Totals		280: 3	3.29	0.27	168:11	24.77**	0.004	93:11	3.34
F3		127: 3	12.53**	0.56	125: 5	5.0*	1.27	121: 9	0.11
Grand totals		407: 6	12.15**	0.04	293:16	27.53**	0.60	214:20	2.13

to race efg (Table 32). This pattern and the IT data provided limited evidence that the two genes detected by race efg were the same genes detected by races h and egh.

These results indicated that NEP 2 carried a minimum of three genes for resistance. In the tests with race efg, the presence of two different ITs (Table 30) and the satisfactory agreement with a 15:1 ratio suggests that there are genes at two independently segregating loci. The gene conferring IT ;N is tentatively named *Ur-I* and the other gene governing IT 2-/2-WA or 2-/2PA is designated *Ur-K*.

Thus F2 ratios, reaction patterns with a range of races and IT data indicate that NEP 2 possesses genes for rust reaction at four loci. However, the ratio of pooled homozygous resistant and segregating lines to homozygous susceptible lines when inoculated with race h was consistent with an hypothesis of only three segregating genes.

Two explanations for this discrepancy are:-

(i) Races h, egh and efgj were detecting different genes in individual segregating lines. For example, in line 200.13, races h and egh may be detecting different host genes, *Ur-X* and *Ur-Y*, which might be represented in differentials x and y, respectively. Race h (or more precisely, hx) may be avirulent on plants with *Ur-Y* but virulent on those with *Ur-X*, whereas race egh (or more precisely, eghy) may be avirulent on *Ur-X* but virulent on *Ur-Y*.

(ii) Some of the genes, *Ur-F*, *Ur-I*, *Ur-J*, and *Ur-K* were linked.

Progeny-tests of nine selected F3 lines (Table 33) were carried out to examine these hypotheses.

Table 33. Reactions of certain F3 lines of Sanilac/NEP 2 when tested with selected races of *U. appendiculatus*, listed in order of presentation of F4 tests

Line	Race					Gene segregating *Ur-*
	h	egh	efgj	af	fg	
	Res:Sus	Res:Sus	Res:Sus	Res:Sus	Res:Sus	
195.15	15:2	17: 3	15: 5			*K*
202.10	15:5	13: 6	18: 1			*K*
195.3	13:6	0:16	0:20	14:3	16:4	*F*
194.9	13:6	18: 2	0:19			*J*
200.13	15:5	16: 4	0:20	12:6	16:4	*J*
195.19	20:0	19: 0	17: 3			*I*†
194.16	17:1	13: 4	16: 4			*I* & *J*+
195.1	19:1	12: 4	16: 3			*F* & *K*
195.13	20:0	16: 0	13: 0			*I* & *K*

† Homozygous for *Ur-J*.

Table 32. Reaction patterns of certain F3 lines of Sanilac/NEP 2 when inoculated with selected races of *U. appendiculatus*

Line	Race		
	h	egh	efgj
199.16	Hom. sus.	Hom. sus.	Hom. sus.
201.2	Hom. sus.	Hom. sus.	Hom. sus.
202.6	Hom. sus.	Hom. sus.	Hom. sus.
195.3	Segregating	Hom. sus.	Hom. sus.
202.11	Segregating	Hom. sus.	Hom. sus.
194.9	Segregating	Segregating	Hom. sus.
199.5	Segregating	Segregating	Hom. sus.
199.17	Segregating	Segregating	Hom. sus.
200.13	Segregating	Segregating	Hom. sus.

Both were effective against races h, egh and efg.

F3

The second and third groups of lines listed in Table 32 showed differing segregation patterns with races h, efg and egh. Whereas lines 195.3 and 202.11 segregated with race h but were homozygous susceptible with both efg and egh, lines 194.9, 199.5, 199.17 and 200.13 segregated with races h and egh but were homozygous susceptible with race efg. Thus two additional genes may be postulated. One, in lines 195.3 and 202.11, giving similar reaction patterns and ITs to a gene in differential e and to *Ur-F* detected in Section 5.3.1.2.1. (p. 60). The second in 194.9, 199.5, 199.17 and 200.13 is not represented in the differential set and is designated as *Ur-J*. Thus if line 200.13 is nominated as differential j, the race known as efg is more precisely described as efgj, since it is virulent on j as well as differentials e, f and g. Whereas the IT of *Ur-F* was usually ;1, that of *Ur-J* varied between ;1 and 22-NR22-.

F4 and F5

Lines segregating at one locus

Ur-I. Only one F3 line, 200.5, gave results consistent with monogenic segregation for *Ur-I*. No residue seed was available.

Ur-K. The ITs of resistant plants were 2-/2-WA and 2/22+WA in some tests and 2-/2-WA in others. F3 lines 195.15 and 202.10 were progeny-tested.

195.15. There was complete agreement between distribution of reaction classes of all F4 lines when tested with races h, fg, egh, efgj and adehi. The distribution of 5 homozygous resistant : 9 segregating : 4 homozygous susceptible lines indicated variation at a single locus ($\chi^2_{1:2:1} = 0.10$, $P>0.5$). The pooled frequencies of 480 resistant and 137 susceptible seedlings using the five races on all the nine segregating lines supported this hypothesis ($\chi^2_{3:1} = 2.57$, $P>0.1$).

202.10. There was complete agreement for reactions of each family when inoculated with races h, fg and efgj. Seven lines were homozygous resistant, 7 segregated and 4 were homozygous susceptible, a distribution indicative of single-locus segregation ($\chi^2_{1:2:1} = 1.62$, $P>0.3$). In the tests with races h and efgj, the resistant seedlings were separated into two IT classes, 2-/2-WA and 2/22+WA, whereas in the inoculations with fg there was less variation and all seedlings showed IT 2-/2-WA. The summed frequencies of 32 IT 2-/2-WA : 71 IT 2/22+WA : 46 IT 3+3 for the tests with race h gave a satisfactory fit to a 1:2:1 ratio ($\chi^2 = 2.86$, $P>0.2$). The pooled frequencies of 86 resistant : 28 susceptible plants when screened with race fg, closely conformed to a 3:1 ratio ($\chi^2 = 0.01$, $P>0.8$). The frequencies of 39 IT 2-/2-WA : 46 IT 2/22+WA : 31 IT 3+4 in the tests with race efgj gave poor agreement with a 1:2:1 ratio ($\chi^2 = 6.07$, $P<0.02$), but close agreement with a 3:1 ratio when the frequencies for the two low IT groups were pooled ($\chi^2 = 0.18$, $P>0.5$). The poor agreement with a 1:2:1 ratio was attributed to misclassification of homozygous and heterozygous plants. The summed frequencies of 374 resistant : 105 susceptible seedlings in the seven segregating lines for all three races also

supported the single-gene hypothesis ($\chi^2_{3:1}$= 2.42, P>0.1).

The F4 line 195.15.18 was selected as the single gene stock for *Ur-K* and accessioned as B2052. This accession was resistant to all cultures used in the greenhouse and to race adhi in the field.

Ur-F. The ITs observed were 2- and 2 in some experiments and 2-/1 and 2/1 in others. Line 195.3 was progeny-tested. There was complete agreement in reaction of each F4 line with races h, gh and cfh. Three lines were homozygous resistant, 9 segregated and 5 were homozygous susceptible ($\chi^2_{1:2:1}$= 0.53, P>0.7), indicating segregation at a single locus. The frequencies of 273 resistant; 75 susceptible plants in the nine segregating lines, pooled over races also supported this hypothesis ($\chi^2_{3:1}$= 1.4, P>0.3). Races egh, efgj and adehi gave susceptible reactions on 195.3 derivatives, confirming the similarity of reaction with the gene in differential e.

The F4 line 195.3.8 was chosen as the single gene stock for *Ur-F* and was accessioned as B2051.

Ur-J. F3 lines 194.9 and 200.13 were progeny-tested.

194.9. There was complete agreement between distribution of reaction classes for all lines when tested with races h, egh and fg. One line was homozygous resistant, 11 segregated and 7 were homozygous susceptible ($\chi^2_{1:2:1}$= 4.27, P>0.1), a distribution statistically consistent with segregation at a single locus. The pooled ratio of 485 resistant : 147 susceptible plants in the segregating lines tested with the three races supported this hypothesis ($\chi^2_{3:1}$= 1.03, P>0.2).

200.13. The ITs of resistant plants varied between ;1 and 2 in some tests and between 12-NR and 2-22+3-3NR in others. The necrotic ring was present in tests with races egh and adehi, as well as in one experiment with race h, but was not seen in a later study with race h, or with races fg, gh and cfh. Hence the reaction probably varied with the environment. There was complete agreement between reactions in the tests with races h, fg, gh, cfh, egh and adehi. The distribution of 3 homozygous resistant : 11 segregating : 3 homozygous susceptible conformed

with a 1:2:1 ratio (χ^2 = 1.48, P>0.3). The pooled frequencies of 569 resistant : 172 susceptible seedlings in the 11 segregating lines tested with the six races were consistent with single-gene segregation ($\chi^2_{3:1}$ = 1.27, P>0.2). As expected, all F4 lines carrying *Ur-J* were susceptible when infected with race efgj.

The F4 line 194.9.16 was chosen as a single gene stock for *Ur-J* and accessioned as B2053.

Race efgj, the only known Australian race virulent for *Ur-J*, is also virulent for *Ur-F*, thus this combination enabled less precise classification of F3 lines than a culture virulent for *Ur-J* but avirulent for *Ur-F* would have done. Since races fg and efgj have similar virulence formulae, the possibility that fg is virulent for *Ur-J* should be considered. As indicated in Table 32, F3 line 200.13 segregated when inoculated with races h, fg and egh, but was homozygous susceptible with efgj. However, from F3 data, using inoculations on separate samples, lines segregating for *Ur-J* would not be confidently distinguished from those segregating for *Ur-F* and *Ur-J*, if these genes were closely linked in coupling. On the other hand, close linkage is unlikely as two F3 lines segregated only for *Ur-F* while at least two and possibly four segregated monogenically for *Ur-J* (Table 32). The close agreement with the single gene (*Ur-J*) model in the two F3 lines subjected to progeny-testing, therefore enabled rejection of the hypothesis that F3 lines 194.9 and 200.13 carried both *Ur-F* and *Ur-J*, and that race fg is also virulent for *Ur-J*.

Ur-I and *Ur-J*. 195.19. This line was chosen because it may have been segregating for both *Ur-I* and *Ur-J*. In the race h and egh inoculations, all lines were homozygous resistant, showing either IT ;N, typical of *Ur-I* or ;1, consistent with *Ur-J*.

In the tests with race efgj, segregation occurred and the resistant seedlings showed only IT ;N. The distribution of 10 homozygous resistant : 18 segregating : 7 homozygous susceptible lines conformed to a 1:2:1 ratio (χ^2 = 0.54, P>0.7). The pooled frequencies of 245 resistant : 78 susceptible plants in the 18 segregating

segregating lines also indicated a single-gene segregation ($\chi^2_{3:1}$= 0.12, P>0.7).

The occurrence of susceptible seedlings only in tests with race efgj indicates that these lines are homozygous *Ur-J Ur-J*. The IT ;N indicates that *Ur-I* is present. Thus the genotype of F3 line 195.19 is *Ur-I ur-I Ur-J Ur-J*.

Lines segregating at more than one locus

Ur-I and *Ur-J*. 194.16. Infection type ;N, typical of *Ur-I* and IT 2, consistent with either *Ur-F* or *Ur-J* were observed on resistant seedlings in tests with races h, fg and egh. Only IT ;N was seen on resistant seedlings in tests with race efgj.

Limited seed supplies restricted the testing that could be done, especially with race fg. Detailed results given in Table 34 indicate that the 16 lines tested with h and egh reacted similarly. However, there were differences between reactions with fg and those with h and egh. There were also differences when reactions with efgj were compared with h and egh, but the differences here may be attributed to the presence of *Ur-J*. No gene previously distinguished can explain the homozygous resistant reaction given by 194.16 lines 2,3,18,19 and 24 when tested with race fg and the segregation of most of these lines when tested with races h, egh and efgj. All lines deviated in the same direction.

The numbers of seedlings tested per line with races h and fg were too small to permit precise classification of lines as homozygous resistant, segregating for one gene or for two. However, if the three classes of lines are pooled, χ^2 values may be calculated for the ratio of this total to that of the homozygous susceptible class. While in the tests with race h, 17 lines were homozygous resistant and 19 segregated, and in the tests with fg, 23 lines were homozygous resistant and 13 segregated, the total, 36 was the same for both races. Two lines, 194.16.1 and 194.16.4 were susceptible to both cultures. This line distribution is consistent with two-gene segregation ($\chi^2_{15:1}$= 0.16, P>0.7).

Similarly, considering the frequencies of resistant and susceptible seedlings in the segregating lines, the numbers tested were too small to distinguish homozygous

Table 34. Segregation ratios in 194.16.1-41 derived from Sanilac/NEP 2 with races h, fg, egh and efgj

Lines	Races											
	h			egh			fg			efgj		
	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.
194.16.1			20			20			20			20
2		18: 4			15: 5		21				18: 2	
3		20: 1		20			21				18: 1	
4			21			20			20			19
5		20: 1		20				18: 2			16: 3	
6		18: 3			15: 5			18: 3				20
7	18			20			21			19		
8	20			20			21			20		
9	20			20			21			20		
11	20			20			21				17: 3	
12	20			20			20			19		
14	19			20			19			19		
15	21			18			19			19		
16	19						19			17		
17		15: 4		20				19: 1			15: 4	
18		13: 7			18: 2		19				15: 4	
19		19: 1					20				18: 2	
20		13: 7			21: 4			16: 5			16: 4	
21	17					13				11		
22		18: 3						14: 6				13
23		19: 1						18: 2			16: 3	
24		20: 1					21				7: 9	
25		17: 2				20				16		
26		15: 6						16: 4				22
27	20					20				19		
28	20					20					13: 6	
29	19					15						
30		16: 4						17: 2			14: 4	
31	19					19				14		
33		19: 2						12: 4				18
34	20					20						18

Table 34. Segregation ratios in 194.16.1-41 derived from Sanilac/NEP 2 with races h, fg, egh and efgj (cont.)

Lines	Races											
	h			egh			fg			efgj		
	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.	Hom Res.	Seg Res:Sus	Hom Sus.
194.16.35	21						21					20
36		13: 8						16: 4			16: 4	
37		16: 3						15: 5			13: 5	
38		14: 4						14: 6			13: 5	
39	20						21				12: 8	
40	21						20			20		
41		14: 5						11: 6				

resistant from segregating lines. However, since similar numbers were screened with each race, a test of interaction may be made on the pooled frequencies of resistant plants in all lines and susceptible plants in segregating lines to determine if the populations in experiments with races h and fg were different. The method of Steel and Torrie (1960) was used:-

For the table

Race	Reaction		Totals
	Resistant	Susceptible	
h	$n_{11} = 651$	$n_{12} = 66$	$n_{1.} = 717$
fg	$n_{21} = 656$	$n_{22} = 50$	$n_{2.} = 706$
Totals	$n_{.1} = 1307$	$n_{.2} = 116$	$n_{..} = 1423$

$$\text{Adjusted } \chi^2 = \frac{[n_{11}n_{22} - n_{12}n_{21} - n_{..}/2]^2 n_{..}}{n_{1.}n_{2.}n_{.1}n_{.2}}$$

$$= \frac{[(651 \times 50) - (66 \times 656) - (1423/2)]^2 1423}{717 \times 706 \times 1307 \times 116} = 1.87 \qquad P>0.1$$

The adjustment to the χ^2 value is made because only one degree of freedom is involved.

This probability value indicates that the populations are not significantly different.

The lack of agreement in distribution of reaction classes when tested with races h and fg may be further investigated by progeny-tests on seedlings susceptible to race h and resistant to race fg. Such data would enable consideration of the hypothesis that a fifth gene is present in NEP 2. If a fifth gene is isolated, races h, egh and efgj would be virulent for this gene, and fg avirulent. Linkage, possibly with *Ur-I* may be postulated.

Both the distribution of disease reaction classes and the pooled frequencies of resistant and susceptible seedlings within segregating lines supported the single-gene hypotheses suggested in tests with these genes separately (Table 35). There was no evidence of linkage.

Thus the genotype of F3 line 194.16 may be given as *Ur-I ur-I Ur-J ur-J*, subject to tests for a fifth gene. *Ur-I* and *Ur-J* were inherited independently in this line.

In order to derive a stock homozygous resistant only for *Ur-I*, seven F4 plants in one line, 194.16.36, were progeny-tested with races h, fg, cfh, efgj and adehi. The line distribution of 2 homozygous resistant : 4 segregating : 1 homozygous susceptible obtained with each race conformed with a 1:2:1 ratio (χ^2 = 0.43, P>0.8). The pooled frequencies of 372 resistant : 102 susceptible seedlings obtained with the five races in the four segregating lines supported the single-gene hypothesis ($\chi^2_{3:1}$ = 3.06, P>0.05).

The F5 line 194.16.36.7 was chosen as a single gene stock for *Ur-I* and accessioned as B2091. However, if a fifth gene is involved it may also be present in B2091.

Ur-F and *Ur-K*. 195.1. In the tests with race h, the ITs of resistant seedlings ranged between 2-/2-WA and 2/22+WA, typical of *Ur-K* and ;1 consistent with *Ur-F*.

Table 35. Genotypic classification of 194.16.1-41 derived from Sanilac/NEP 2 when tested with races h, egh and efgj

Reaction classes				Seedling frequencies in segregating lines	$\chi^2_{3:1}$	Postulated genotype
Race efgj	No. of lines	Race h	No. of lines	Res:Sus		
Hom. res.	12		12			*Ur-I Ur-I* / *Ur-I Ur-I*
Seg.	16	Hom. res.	3	237: 67	1.42	*Ur-I ur-I Ur-J Ur-J*
		Seg.	13			*Ur-I ur-I Ur-J ur-J*
Hom. sus.	8	Hom res.	2			*ur-I ur-I Ur-J Ur-J*
		Seg.	4	70: 14	2.81	*ur-I ur-I Ur-J ur-J*
		Hom. sus.	2			*ur-I ur-I ur-J ur-J*

$\chi^2_{2:1}$ †	*Ur-I ur-I:ur-I ur-I*	16:8	= 0
$\chi^2_{1:2:1}$	*Ur-J Ur-J:Ur-J ur-J:ur-J ur-J*	5:17:2	= 4.92
Heterogeneity (linkage) (2 d.f.)			= 4.92
$\chi^2_{8:1:2:1}$	Joint segregation	16:2:4:2	= 0

† The incomplete classification permitted only certain classes to be included in the analysis.

However, in tests with races egh and efgj, only the ITs typical of *Ur-K* were seen.

The reaction of some lines in tests with races h and egh confirmed the presence of *Ur-F* and *Ur-K*. Some lines which were homozygous resistant or segregating with race h were homozygous susceptible when inoculated with races egh and efgj. Others, homozygous resistant with race h, segregated with efgj. The genotypic classifications of the 14 lines tested (Table 36) indicated that the

Table 36. Genotypic classification of 195.1.1-14 derived from Sanilac/NEP 2 when tested with races h, egh and efgj

Race efgj	Reaction class: No. of lines	Race h	No. of lines	Postulated genotype
Hom. res.	5	Hom. res.	3	*Ur-K Ur-K Ur-F Ur-F*
		Seg.	1	*Ur-K Ur-K Ur-F ur-F*
		Hom. sus.	1	*Ur-K Ur-K ur-F ur-F*
Seg.	7	Hom. res.	3	*Ur-K ur-K Ur-F Ur-F*
		Seg.	2	*Ur-K ur-K Ur-F ur-F*
		Hom. sus.	2	*Ur-K ur-K ur-F ur-F*
Hom. res.	2	Hom. res.	1	*ur-K ur-K Ur-F Ur-F*
		Seg.	1	*ur-K ur-K Ur-F ur-F*
		Hom. sus.	0	*ur-K ur-K ur-F ur-F*

$\chi^2_{1:2:1}$	*Ur-K Ur-K:Ur-K ur-K:ur-K ur-K*	5:7:2 = 1.28
$\chi^2_{1:2:1}$	*Ur-F Ur-F:Ur-F ur-F:ur-F ur-F*	7:4:3 = 4.85
χ^2 Heterogeneity (6 d.f.)		2.14
$\chi^2_{1:2:1:2:4:2:1:2:1}$	Joint segregation	3:1:1:3:2:2:1:1:0 = 8.17

loci involved segregated independently. The pooled frequencies of 165 resistant : 58 susceptible seedlings in the seven lines which segregated when tested with races egh and efgj gave a close fit to the predicted 3:1 ratio (χ^2 = 0.18, P>0.7). The ITs shown by seedlings with *Ur-K* were clearly different from those with *Ur-J*. Since *Ur-J* was epistatic to *Ur-K* complete classification was possible.

Thus the genotype of 195.1 was *Ur-F ur-F Ur-K ur-K*.

Ur-I and *Ur-K*. 195.13. In the tests with races h, egh and efgj, the ITs of

resistant seedlings were ;N, typical of *Ur-I* and 2-/2-WA indicative of *Ur-K*. All lines reacted identically with the three races. The distribution of reaction classes over the 17 lines (Table 37) was consistent with segregation at two independently inherited loci. Thus the genotype of 195.13 is *Ur-I ur-I Ur-K ur-K*.

Table 37. Genotypic classification of 195.13.1-17 derived from Sanilac/NEP 2 when tested with races h, egh and efgj

Genotype	Expected ratio	Observed frequency
Ur-I Ur-I —— ——	4	8
Ur-I ur-I Ur-K Ur-K	2	0
Ur-I ur-I Ur-K ur-K	4	4
Ur-I ur-I ur-K ur-K	2	3
Ur-I ur-I Ur-K Ur-K	1	0
ur-I ur-I Ur-K ur-K	2	1
ur-I ur-I ur-K ur-K	1	1
	16	17

$\chi^2_{2:1}$ †	*Ur-I ur-I:ur-I ur-I*	7:2	= 0.40
$\chi^2_{1:2:1}$	*Ur-K Ur-K:Ur-K ur-K:ur-K ur-K*	0:5:4	= 3.67
χ^2	Heterogeneity (2 d.f.)		= 0.25
$\chi^2_{2:4:2:1:2:1}$	Joint segregation	0:4:3:0:1:1	4.32

† The incomplete classification permitted only certain classes to be included in the analysis.

Discussion and summary of analysis of NEP 2

Four genes were isolated from NEP 2 and there was limited evidence for a fifth gene for which races h, egh and efgj are virulent and fg avirulent.

In F3, F4 and F5 tests, lines homozygous susceptible with race h were also homozygous susceptible with races egh, efgj and other races used in certain F4 and F5 inoculations. Thus race h is avirulent for all four genes isolated from NEP 2, while egh and other races virulent on e are virulent for *Ur-F* and race efgj is virulent for two genes, *Ur-F* and *Ur-J* (Plate 2). Thus the hypothesis that races h, egh and efgj are detecting different genes in individual segregation lines may be rejected.

The alternative hypothesis is that some genes are linked. The ratios of resistant : susceptible F2 plants in the tests with race efgj and the progeny testing of 195.13 suggest that *Ur-I* and *Ur-K* are inherited independently of each other. The results of progeny-tests of 195.1 with genotype *Ur-K ur-K Ur-F ur-F* and 194.16 with genotype *Ur-I ur-I Ur-J ur-J* did not indicate linkage between *Ur-F* and *Ur-K* or *Ur-I* and *Ur-J*. Other possible linkages are between *Ur-F* and *Ur-I*, *Ur-J* and *Ur-K* or *Ur-F* and *Ur-J*. The simplest method of determining which genes may be linked is to test crosses of the derived single gene stocks.

The ITs of *Ur-I* and *Ur-K* were clearly distinguishable from each other and from other genes effective against all Australian races, *viz. Ur-E, Ur-G* and *Ur-H*.

There was complete agreement between field and greenhouse results with race h, both on F2 and F3 plants, indicating that the four genes conferred protection under field conditions.

5.3.1.2.5. Bonita

When tested with race h, the F1 plants produced IT 22+/2WA, slightly higher than the Bonita parent with IT 22-/2-WA. The F2 plants showed both these ITs, as well as 3+4, but classification into three meaningful categories was not possible. Infection types 2+2/3+4 and 3+3 were observed on some plants in certain F3 lines.

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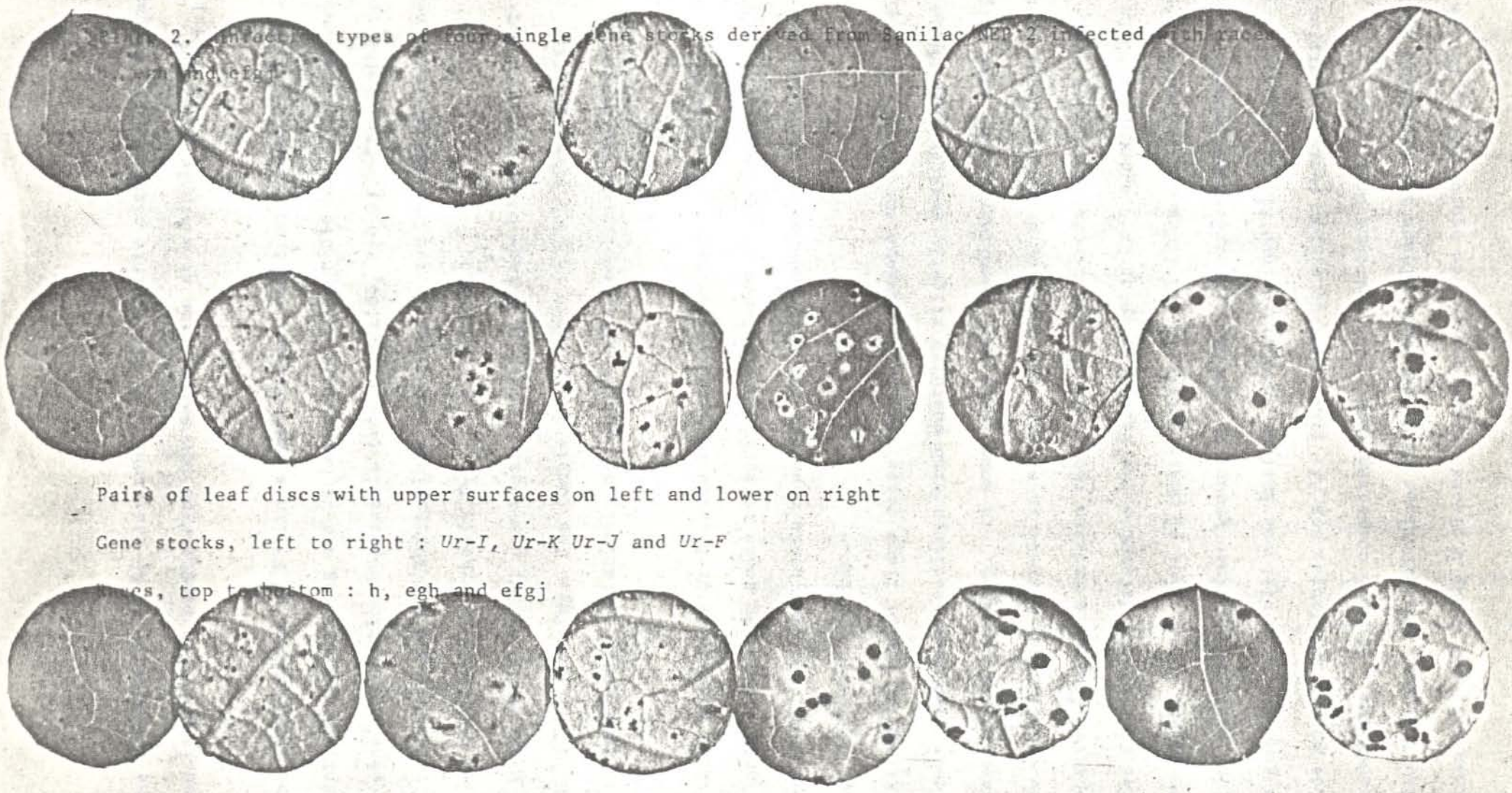
Plate 2. Infection types of four single gene stocks derived from Sanilac/NEP 2 infected with races h, egh and efgj



Pairs of leaf discs with upper surfaces on left and lower on right

Gene stocks, left to right : *Ur-I, Ur-K Ur-J* and *Ur-F*

Races, top to bottom : h, egh and efgj

The frequencies of 48 resistant : 12 susceptible plants in the F2 population were consistent with expectation for segregation at a single locus ($\chi^2_{3:1} = 0.8$, $P>0.7$).

The distribution of F3 lines into disease reaction classes of 27 homozygous resistant, 59 segregating and 21 homozygous susceptible was consistent with the single-gene segregation hypothesis ($\chi^2_{1:2:1} = 1.65$, $P>0.3$). The pooled frequencies of 865 resistant and 312 susceptible seedlings in the 59 segregating lines also conformed with this hypothesis ($\chi^2_{3:1} = 2.7$, $P>0.01$).

A group of 20 lines, previously tested with race h was screened with races gh, cfh, adehi and efgj. The complete agreement in line classification indicated that the same gene, tentatively designated *Ur-L*, conferred resistance to all five races.

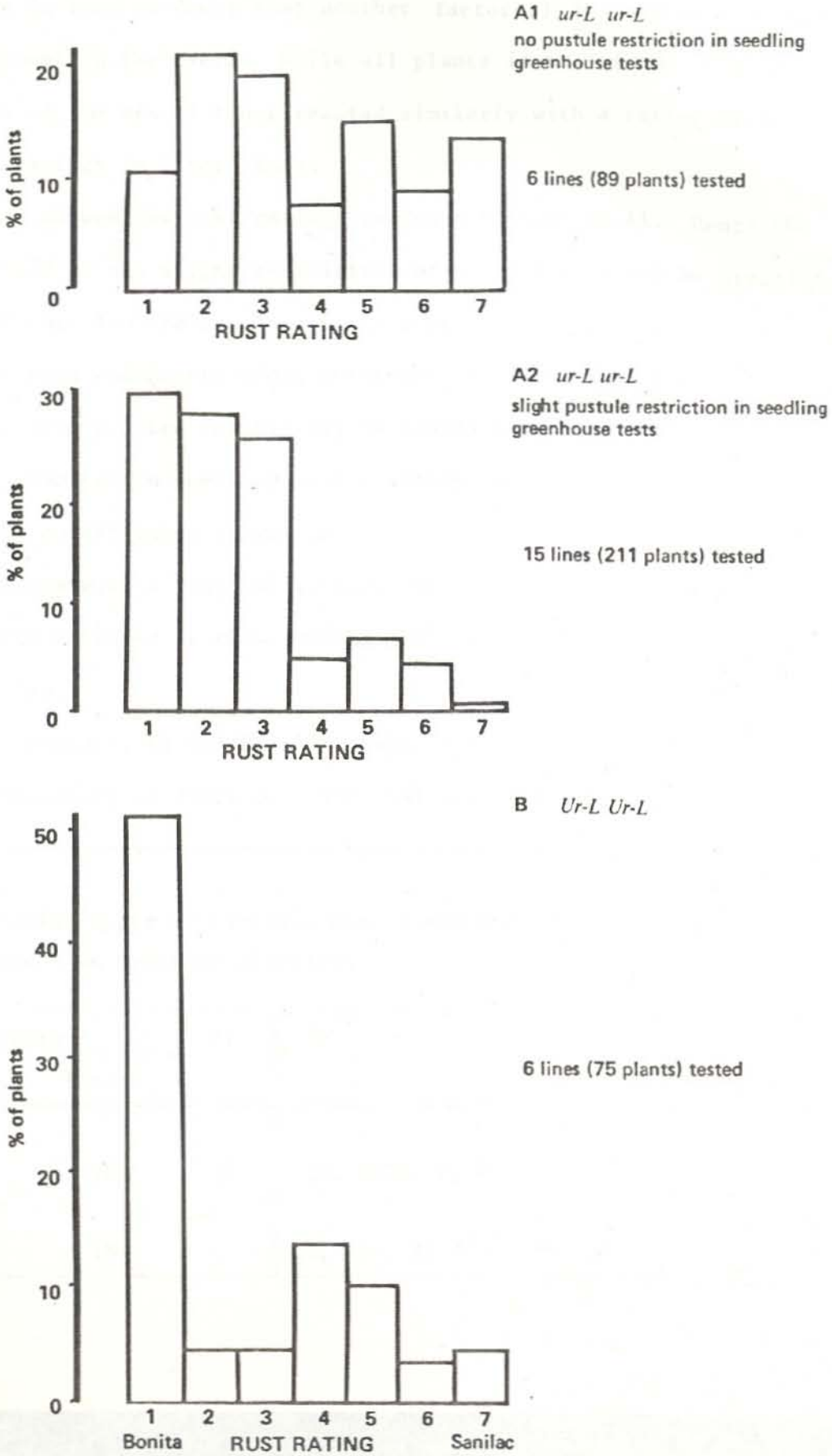
Plants possessing *Ur-L* could be readily distinguished on the basis of IT from plants possessing genes conferring resistance to all races, *Ur-D*, *Ur-G*, *Ur-H* and *Ur-I*. In some tests however, the ITs produced by seedlings carrying *Ur-K* and *Ur-L* were similar.

Fifteen of the 21 lines classified as homozygous susceptible in greenhouse tests contained some seedlings with pustules slightly smaller than those typical of a susceptible reaction. In some lines most seedlings showed some slight pustule restriction, whereas in others, the majority developed large pustules.

Observations were made in the field of rust development (race adhi) on selected F3 lines to determine the relationship of *Ur-L* and the factor(s) governing the slight restriction of pustule size in the lines classified as *ur-L ur-L*. The lines were divided into groups on the basis of greenhouse reactions. Group A with genotype *ur-L ur-L* was further subdivided into A1, lines producing large pustules on all seedlings, and A2, lines showing slight pustule restriction on some seedlings. Group B comprised lines with genotype *Ur-L Ur-L*.

The frequency distributions of field rust ratings are presented in Figure 2. More than half the Group B plants showed resistance equivalent to Bonita (rating 1).

Figure 2. Frequency distributions of field rust ratings of three groups of F3 plants derived from Sanilac/Bonita.



However, some were as susceptible as the Sanilac parent (rating 7), indicating that *Ur-L* is of limited effectiveness in at least some genetic backgrounds. In contrast, approximately 10 and 30 per cent of plants in Groups A1 and A2, respectively, showed field resistance similar to the Bonita parent, despite the absence of *Ur-L*. There is thus no doubt that another factor(s) is involved in restriction of rust development in the field. While all plants in two of the 15 Group A2 lines and three of the six A1 lines reacted similarly with a rating of 1, there was a range of ratings in other lines.

More plants showed low rust ratings in Group A2 than in A1. Hence the factor(s) involved in the slight restriction of pustule size may be associated with slower and more limited development of rust in the field.

Blanca del Pais and Puerto Rican which showed identical ITs to Bonita in the greenhouse, also reacted identically to Bonita in the field.

The locus involved in seedling rust reaction in Bonita segregated independently from that conditioning growth habit.

Some F3 plants with a bush habit, small white seeds, a rust rating of 1 but lacking *Ur-L* were selected as slow-rusting germplasm for bean breeding purposes.

5.3.1.2.6. Aurora

The ITs of parents, F1 and F2 plants when tested with race h on two separate occasions are presented in Table 38. The distinct differences in the ITs in the

Table 38. Infection types of Sanilac, Aurora and their F1 and F2 progeny when tested with race h on separate occasions

Parents		F1	F2
Sanilac	Aurora		
(i) 3+4	;N	;N	;N, ;1NR, 2, 3+
(ii) 3+3+4	;N		;, 12-, 2, 2/3+, 3+, 3+4

first test indicated the possibility of two genes, one with IT ;N or ;1NR and another with IT 2. However, classification into two discrete classes was not possible in the second test and therefore 28 seedlings with ITs 2- and 2 were progeny-tested. The pooled results for the F2 and F3 progeny tests were 360 seedlings with IT ;N, ;1NR and ;, five with IT 2 or 2+2 and 109 with IT 3+ or 3+4. The IT of F1 plants indicated a dominance of resistance.

The ratio of 365 resistant plants (ITs ;N, ;1NR, 2 and 2+2) : 109 susceptible seedlings (ITs 3+ and 3+4), showed satisfactory agreement with a 3:1 ratio (χ^2 = 1.01, P>0.3), and very poor agreement with a 15:1 ratio (χ^2 = 173.29, P<0.001).

The ITs of seedlings in F3 lines were ;N, 12-NR, 2, 2+3-, 3+ and 3+4. One line, 185.11, showed only ITs 2, 2+3- and 3+4 and nine lines produced IT 2+3 as well as ITs ;N and 12-NR. The isolation of a line in which ITs ;N and 12-NR were absent provided further support for a two-gene hypothesis. In this line, 10 plants showed IT 2, three IT 2+3- and six IT 3+4, conforming to the expected 3:1 ratio (13:6, χ^2 = 0.44, P>0.6).

Seventy-six lines were inoculated with race h. A further 41 families, plus 29 of those previously tested were inoculated with race efgj. There was complete agreement in reactions of the 29 lines tested with both races, so presumably the same gene was involved in each instance. Hence results of both races were pooled.

The distribution of disease reaction classes among F3 lines is presented in Table 39. Again there was good agreement with a 1:2:1 and poor agreement with a 12:3:1 ratio. The total frequencies of 920 resistant : 273 susceptible plants in the 59 segregating lines in which IT ;N was observed conformed with expectation for segregation at a single locus ($\chi^2_{3:1}$= 2.85, P>0.05), but not for segregation at two independently inherited loci ($\chi^2_{15:1}$= 562.83, P<0.001).

Sixteen plants of family 185.11 were progeny tested. The distribution of 5 homozygous resistant (IT 22+ to 2+2) 8 segregating (IT 2+2, 2+3-, 3, 3+) and 3 homozygous susceptible (IT 3+) conformed with single-gene segregation

Table 39. Distribution of disease reaction classes in F3 lines of Sanilac/Aurora when tested with races h and efgj

Family group	Race	Reaction classes					$\chi^2_{1:2:1}$	$\chi^2_{12:3:1}$
		Hom. res.	Segregating			Hom. sus.		
		;N	;N, 12-NR, 3+, 3+4	;N, 12-NR, 2, 2+3-, 3+, 3+4	2, 2+3- 3+, 3+4	3+, 3+4		
1	h	13	14	0	0	16		
	h, efgj	5	9	3	1	5		
		18	23	3	1	21	2.45	79.99**
2	h, efgj	4	5	0	0	1		
	efgj	10	22	6	0	13		
		14	27	6	0	14	0.41	43.24**
							2.86	123.23
Grand totals		32	50	9	1	35	0.53	114.45**
Heterogeneity (2 d.f.)							2.33	8.78**

($\chi^2_{1:2:1}$ = 0.5, P>0.3) and was identical with each of the four races used. In three tests the pooled frequencies of resistant and susceptible seedlings for the eight segregating lines conformed with those expected (Table 40), but with race h there was a deficiency of susceptible plants. This was attributed to chance. In these progeny tests, the ITs of resistant plants (22+ and 2+3-) were clearly different from those of IT ; and ;N shown by Aurora.

Since the ITs were sufficiently distinct, two genes are proposed, one designated *Ur-M* with IT ;N or ; and another *Ur-N*, governing IT 2, 2+2 or 2+3-. Seedlings with *Ur-M* have an identical IT to those with *Ur-I* isolated from NEP 2. While the IT attributed to lines with *Ur-N* is similar to that of B1627 Gallaroy

Table 40. Frequencies of resistant and susceptible seedlings in segregating F4 lines derived from 185.11 (Sanilac/Aurora) when tested with four races of the bean rust fungus

Race	No. of families	Reaction Resistant:Susceptible	χ^2 3:1
b	8	119:24	5.15*
gb	4	61:19	0.07
cfh	4	47:12	0.68
adehi	3	41:14	0.01
			5.91
		268:69	3.68
Heterogeneity (3 d.f.)			2.23

Genotype I with *Ur-A*, *Ur-N* is effective against race adehi as well as other Australian races and therefore may be distinguished on the basis of reaction with the a group of races.

The distribution of the F3 lines and the pooled frequencies of resistant and susceptible seedlings in segregating lines agreed closely with a one-gene and poorly with a two-gene hypotheses. Hence linkage is indicated. With the cultures available only limited classification could be carried out. The following F2 classifications were obtained by testing F2 plants and F3 lines.

	F2	F3	Totals
Ur-M —— —— ——	360	90	450
ur-M ur-M Ur-N ur-N	5	1	6
ur-M ur-M ur-N ur-N	109	35	144
	474	126	600

The recombination value was estimated using equation 10, Table 6 of Allard (1956) *viz.*

$$456 \frac{(-2r)}{(4-r^2)} + 144 \left(\frac{2}{r}\right) = 0$$

$r = 0.98$

Coupling linkage r' was estimated by subtraction from unity *i.e.* r' = 1 - 0.98 = 0.02

$$s = \sqrt{\frac{1}{I}} = \sqrt{\frac{1}{600} \times 1.316} = 3.6$$

i.e. r' = 2 $\pm$ 4%

Line 185.11.7 was chosen as single gene stock for *Ur-N* and was accessioned as B2056. No attempt was made to obtain a stock with the gene conditioning IT ;N as the close linkage and absence of a culture virulent on *Ur-N* and avirulent on *Ur-M* made this impractical. However, by using both Aurora and B2056 as testers, the former acts as a tester for *Ur-M* since any cultures virulent on plants with this gene would be expected to produce IT 2+2 or 2+3- rather than IT ; or ;N on Aurora.

The field and greenhouse reactions were in complete agreement. Thus both genes are effective in the field and in the Sanilac background.

The relationship between the loci conditioning rust reaction and habit was examined. Because of the close linkage, the results for *Ur-M* and *Ur-N* were pooled. These loci were inherited independently of habit.

5.3.1.2.7. Sister lines of Actopan/Sanilac Selection 37

Analysis of Actopan/Sanilac Selection 37 in Section 5.3.1.2.1. (p. 60) indicated two genes, *Ur-E* and *Ur-F*, at independently inherited loci. While Selection 37 showed promise in early trials, two sister lines, Actolac (Selection 39) and Actosan (Selection 51) subsequently proved agronomically superior and were selected by the Queensland Department of Primary Industries for release.

In order to apply the results of Actopan/Sanilac Selection 37, F2 populations of crosses of Sanilac with Actolac and Actosan were tested in the same experiment.

In each cultivar seed from a Single Typical Plant was used to derive plants used in crossing.

Actolac

While three ITs were observed on resistant F2 plants in tests with race h, IT ;1, 2- and 22-, only IT ;1 was observed in tests with egh.

The ratios of resistant and susceptible seedlings (Table 41) suggest that two genes are present. Since *Ur-F* was detected in Actopan/Sanilac Selection 37 and the IT is consistent with this gene, there is little doubt that the gene identified by egh is *Ur-F*. The IT of the other gene was identical with *Ur-E*.

Table 41. Frequencies of resistant and susceptible seedlings in F2 populations of Sanilac/Actolac when tested with races h and egh

Race	Reaction		χ^2 3:1	χ^2 15:1
	Resistant	Susceptible		
h	75	5	15.0**	0
egh	42	9	1.47	12.81**

Actosan

The ITs of resistant F2 plants in tests with race h were ;1, typical of *Ur-E* and 2-, consistent with *Ur-F*. The ITs in tests with race egh were ;1, typical of *Ur-E* and IT 2-NR identical to single gene line B2052, carrying *Ur-J*, inoculated at the same time. Plants with IT 2-NR also showed occasional IT 3+ pustules which were increased and typed as efgj.

The ITs and ratios of resistant and susceptible seedlings (Table 42) suggest that three genes are present. One is probably identical with *Ur-E*, effective against all Australian races, another may be identical to *Ur-F* and a third may be either *Ur-J* or the factor associated with resistance in differential f (Veracruz 1A6).

Table 42. Frequencies of resistant and susceptible seedlings in F2 populations of Sanilac/Actosan when tested with races h, egh and efgj

Race	Reaction		χ^2 3:1	χ^2 15:1	χ^2 63:1
	Resistant	Susceptible			
h	69	5		0.03	12.23**
egh	38	8	1.43	9.57**	
efgj	32	14	0.72		

While there is evidence for three genes, the ratio with race h showed much closer agreement with segregation for two genes than for three. Similarly, ITs and reaction patterns indicate that two genes were detected in the test with race egh, yet the ratio agreed more closely with segregation of one gene than of two. Thus it is likely that linkage between unspecified genes may account for the discrepancy.

5.3.1.2.8. Discussion and summary of genetic analyses

The genotypes of the ten accessions analysed are presented in Table 43. The typical ITs, sources of the genes and races virulent for these genes are given in Table 44.

Six of the genes, namely *Ur-A, Ur-B, Ur-C, Ur-D, Ur-F* and *Ur-J*, may be distinguished on the basis of reaction patterns with Australian races. Another, *Ur-Red*, may be differentiated, but with less certainty. The remaining eight genes are effective against all Australian races.

Data from the five accessions carrying two or more genes in combination give a limited indication of location and number of loci involved, as genes occurring in combination cannot be allelic. Linkage was established in two instances. Four (possibly five) genes were detected in NEP 2.

Table 43. Genotypes of the accessions analysed in crosses with Sanilac

Accession	Gene(s) *Ur-*
Gallaroy Genotype I	*A*
Gallaroy Genotype II	*A* & *B* (r = 25±9%)
Brown Beauty	*C*
Redlands Greenleaf B	*C* *D* & *Red*
Actopan/Sanilac Selection 37	*E* *F*
PR 5	*G*
Cornell 49-242	*H*
NEP 2	*F* *I* *J* & *K*
Bonita	*L*
Aurora	*M* & *N* (r = 2±4%)

With the exception of *Ur-L*, all genes governing low IT in the greenhouse with all Australian races conferred protection in the field. Genes *Ur-1*, *Ur-2* and *Ur-D* were apparently effective until races virulent for them appeared and *Ur-F* and *Ur-Ver* were effective in field trials not reported here (Ballantyne unpublished data).

All genes were either dominant or incompletely dominant.

Table 44. Genes for rust resistance: their range of ITs, sources, reference stocks and races virulent on seedlings carrying them.

Gene *Ur-*	Range of ITs	Race group(s) with virulence	Source(s)	Reference accession or single gene stock
A	2+2 to 2+3-	a	Gallaroy Genotype I	B1627 Gallaroy Genotype I
			Gallaroy Genotype II	
B	; to ;1	(i) & i	Gallaroy Genotype II	B2090
C	22-NR22- to 22+NR22+	h	Brown Beauty	B1561 Brown Beauty
			Redlands Greenleaf B	
D	;	(d) & d	Redlands Greenleaf B	B2113 with *Ur-D Ur-Red*
Red	22+3- to 2+3-33+	d & i	Redlands Greenleaf B	
E	;N, ; and ;1	none	Actopan/Sanilac Selection 37	B2055
F	; to ;1	e	Actopan/Sanilac Selection 37	B2054
			NEP 2	B2051
G	;, occasionally 2-22+ 2NR	none	PR 5	B1667 PR 5
H	;1	none	Cornell 49-242	B1672 Cornell 49-242
I	; to ;N	none	NEP 2	B2091
J	;1 to 2 or 12-NR to 2-22+3-3NR	j	NEP 2	B2053
K	2-/2PA or -WA to 2/2PA or WA	none	NEP 2	B2052
L	2/2-WA to 2+/2WA	none	Bonita	B2091
M	; to ;N	none	Aurora	B1568 Aurora with *Ur-M Ur-N*
N	2+2 to 2+3-	none	Aurora	B2056

5.3.2. INTERCROSSES

Tests were made on 13 intercrosses between resistant stocks to determine allelic and linkage relationships of the genes identified in the crosses with Sanilac. Some intercrosses involved the original accessions, some of which carried more than one gene; others involved the derived single gene stocks. Priority was given firstly to genes effective against all Australian races, and secondly to those showing similarities in IT (Table 45). For example, members

Table 45. Rust resistance genes classified according to similarities in infection type and origin

Typical IT	Gene *Ur-*	Origin
2+2	*A*	Gallaroy Genotypes I and II
	N	Aurora
2+/2WA	*L*	Bonita
2/2-WA	*K*	NEP 2
22+3-3	*Red*	Redlands Greenleaf B
22-NR22-	*C*	Redlands Greenleaf B & Brown Beauty
; or ;1	*B*	Gallaroy Genotype II
	D	Redlands Greenleaf B
	E	Actopan/Sanilac Sel. 37 & NEP 2
	F	Actopan/Sanilac Sel. 37 & NEP 2
	G	PR 5
	H	Cornell 49-242
	J	NEP 2
;N	*I*	NEP 2
	M	Aurora

of the gene pairs *Ur-A* & *Ur-N*, *Ur-K* & *Ur-L* and *Ur-I* & *Ur-M* were indistinguishable from each other on the basis of IT, as well as other criteria. Hence, testing of intercrosses was the first step in determining if such genes were at different loci. Additionally, certain crosses were made to investigate linkage between some of the four genes isolated from NEP 2.

5.3.2.1. Gallaroy Genotype II//Actopan/Sanilac Selection 37

The genes isolated earlier were:-

	Gene	IT	Race groups with virulence	Relationship
Gallaroy Genotype II	*Ur-A* *Ur-B*	2+2 or 2+3- ; or ;1	a (i) & i	*Ur-A* and *Ur-B* linked, r = 25±9%
Actopan/Sanilac Selection 37	*Ur-E* *Ur-F*	; or ;1 ; or ;1	none e	*Ur-E* and *Ur-F* independent

By choosing appropriate test races, linkage involving *Ur-E* and *Ur-A* or *Ur-B* was investigated. F3 lines were screened for this purpose.

Race adehi, virulent on seedlings possessing *Ur-A*, *Ur-B* and *Ur-F* was used to classify for *Ur-E*. The distribution of 34 *Ur-E Ur-E* : 56 *Ur-E ur-E* : 31 *ur-E ur-E* ($\chi^2_{1:2:1}$ = 0.81, P>0.5) and the pooled frequencies of 792 *Ur-E* —— : 282 *ur-E ur-E* individuals in the 56 heterozygous lines ($\chi^2_{3:1}$ = 0.91, P>0.3) conformed with expectation for single-gene segregation.

Since a race virulent on seedlings with *Ur-A* and avirulent on those with *Ur-B* was not available, race egh was used to test further samples of F3 lines. In this situation *Ur-A* and *Ur-B* were distinguished on the basis of IT. When 16 - 20 seedlings of each line were tested, all produced ITs ranging from ;N to 12- and characteristic of both *Ur-B* and *Ur-E*. This suggested that *Ur-B* and *Ur-E* were closely linked or allelic.

Residue seed available in 50 of the 56 lines classified as *Ur-E ur-E* was used for further testing with race egh. On average 130 seedlings (range 111 - 147) were tested in each line. Twenty-nine of the 6,532 total gave ITs 2-22+2- or 2+2, and hence could not be classified as either *Ur-B* or *Ur-E*. Twenty-six

of the 29 seedlings transplanted produced seed. When 16 - 20 F4 seedlings from each of the 26 lines were inoculated with race egh, all gave IT ; or ;1, whereas plants of Gallaroy Genotype I, with *Ur-A*, inoculated at the same time gave IT 2+2. It was concluded that the 29 plants did not result from recombination of *Ur-B* and *Ur-E*. Hence *Ur-B* and *Ur-E* may be allelic.

If *Ur-B* and *Ur-E* are not allelic and assuming that the genetic classifications of all F3 lines were correct, then one estimate of the maximum map distance separating these genes at P = 0.05 is given by the expression

$$(1 - \frac{r^2}{4})^n = 0.05.$$

$$(1 - \frac{r^2}{4})^{6532} = 0.05$$

$$r = 0.043.$$

In this situation, the F2 plants classified as *Ur-B ur-B Ur-E ur-E* on the basis of F3 testing would be genetically similar to F1 plants of genotype *Ur-B ur-B Ur-E ur-E*.

Thus an estimate of the maximum of upper recombination value is 4 *per cent*.

5.3.2.2. Aurora/NEP 2

The genes isolated in previous studies were:-

	Gene	IT	Race groups with virulence	Relationship
Aurora	*Ur-M* *Ur-N*	;N 2+2 or 2+3	None None	*Ur-M* and *Ur-N* are linked, r = 2±4%.
NEP 2	*Ur-F* *Ur-I* *Ur-J* *Ur-K*	; or ;1 ;N ; or ;1 2-/2-WA	e None j None	Linkage suspected but not established

Genes *Ur-I* and *Ur-M* have similar ITs and are epistatic to genes with higher ITs, *viz*. *Ur-N* in Aurora and *Ur-K* in NEP 2. Hence by using race efgj, virulent for *Ur-F* and *Ur-J*, a test of allelism of *Ur-I* and *Ur-M* is possible.

F2 populations derived from six F1 plants were tested. All 135 seedlings showed IT ;N. The absence of recombinants susceptible to race efgj or showing IT

2-/2-WA typical of *Ur-K* or IT 2+2, typical of *Ur-M*, indicated that *Ur-I* and *Ur-M* are either allelic or linked in repulsion. If the genes are not allelic, then the maximum distance at P = 0.05 separating these two genes is given by the expression:-

$$(1 - \frac{r^2}{4})^n = 0.05,$$

$$(1.0 - \frac{0.30^2}{4})^{135} = 0.05$$

$$r = 0.30 \text{ or } 30\%$$

Since there are no overseas data suggesting that Aurora and NEP 2 react differentially, *Ur-I* and *Ur-M* may be identical.

There was no doubt that the progenies tested were from hybrids since the F2 seeds showed slight darkening, typical of the NEP 2 male parent.

5.3.2.3. B2055/Aurora

The genes isolated from Aurora and present in B2055 were:-

	Gene	IT	Race groups with virulence	Relationship
B2055	*Ur-E*	;1	None	
Aurora	*Ur-M* *Ur-N*	;1 2+2 or 2+3-	None	*Ur-M* and *Ur-N* are linked, r = 2±4%

To test for allelism between *Ur-E*, isolated from Actopan/Sanilac Selection 37 and *Ur-M*, F2 populations derived from two F1 plants were tested with race h. One-hundred-and-ninety-five seedlings showed either IT ;N, ; or ;1, whereas two showed IT 2+3-, typical of *Ur-N*. The two seedlings showing IT 2+3- were not progeny-tested because the difference between the low and intermediate ITs was considered obvious both in the F2 population and on the reference stocks, *viz.* B2055, Aurora and B2056, the single gene line for *Ur-N*.

One hypothesis is that *Ur-E* and *Ur-M* are not allelic. Considering only *Ur-E* and *Ur-M*, the seedlings showing IT 2+3- were of genotype *ur-E ur-E ur-M ur-M*.

The ratio of 195 *Ur-E* —— *Ur-M* —— : 2 *ur-E ur-E ur-M ur-M* gives poor agreement with a 15:1 ratio (χ^2 = 9.2, P<0.01) and indicates repulsion linkage of *Ur-E* and *Ur-M*. Using Allard's formula 7, Table 6, the recombination value was calculated as 21±9 *per cent*.

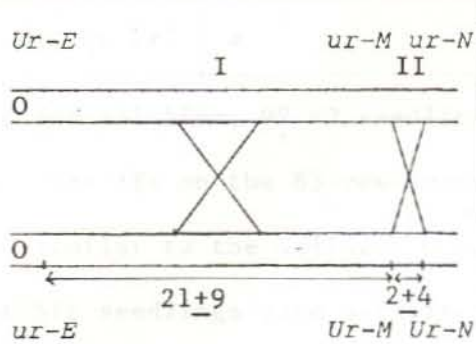
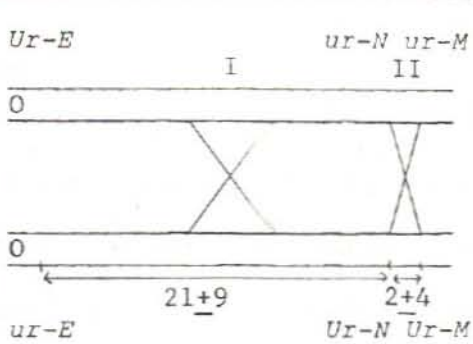
The basis of proposing two distinct genes, in Aurora was an obvious difference in IT (Section 5.3.1.2.6 p. 85). However, if *Ur-M* and *Ur-N* are distinct genes and are very closely linked (p. 89), it is difficult to explain the occurrence of two recombinants with IT 2+3- and the absence of recombinants with IT 3+4.

Two models involving different sequences of genes, in single- and double-crossing-over situations are given in Table 46.

Recombinants with IT 2+3- may occur if (i) gene order is *Ur-E Ur-M Ur-N* and a double-crossover occurs or (ii) if gene order is *Ur-E Ur-N Ur-M* and a single-crossover occurs between *Ur-M* and *Ur-N*. However, earlier results (Section 5.3.1.2.6. p. 85) and those obtained in this Section indicated that the genetic length of region I is possibly ten times greater than that of region II. Thus the the occurrence of two recombinants with phenotype *ur-E ur-M ur-N* was expected to be rare in the small population tested. According to the models in Table 46, cross-overs in regions I (sequence *Ur-E Ur-M Ur-N*) and II (sequence *Ur-E Ur-N Ur-M*) should occur more frequently and may give rise to susceptible plants in each instance.

An alternative hypothesis is that a single gene (*Ur-M*) in Aurora confers IT ;N in some genetic backgrounds and IT 2+2 or 2+3- in others. According to this hypothesis, *Ur-N* does not exist and seedlings showing IT 2+3- also carry *Ur-M*. On this basis, *Ur-E* and *Ur-M* may be allelic and the maximum recombination value at P = 0.05 is then 25 *per cent*.

Table 46. Predicted consequences of crossing over in B2055/Aurora assuming alternative gene sequences

Sequence	*Ur-* *E M N*	*Ur-* *E N M*
Region		
Parental phenotypes	Predicted ITs	Predicted ITs
	Ur-E ur-M ur-N ;1 *ur-E Ur-M Ur-N* ;N	*Ur-E ur-N ur-M* ;1 *ur-E Ur-N Ur-M* ;N
SCO† region I		
	Ur-E Ur-M Ur-N ;N *ur-E ur-M ur-N* 3+	*Ur-E Ur-N Ur-M* ;N *ur-E ur-N ur-M* 3+
region II		
	Ur-E ur-M Ur-N ;1 *ur-E Ur-M ur-N* ;N	*Ur-E ur-N Ur-M* ;1 *ur-E Ur-N ur-M* 2+3-
DCO‡ regions I & II		
	Ur-E Ur-M ur-N ;N *ur-E ur-M Ur-N* 2+3-	*Ur-E Ur-N ur-M* ;1 *ur-E ur-N Ur-M* ;N

† Single-crossover

‡ Double-crossover

5.3.2.4. B2056/Gallaroy Genotype I

The genes identified in these accessions were:-

	Gene	IT	Race groups with virulence	Remarks
B2056	*Ur-N*	22+ or 2+3-	None	Derived from Aurora
Gallaroy Genotype I	*Ur-A*	2+2 or 2+3-	a	

In order to test for allelism, 92 F2 seedlings derived from two F1 plants were screened with race h. The ITs on the 83 resistant plants were either 22+, similar to the B2056 or 2+3-, similar to the Gallaroy Genotype I parent. The ratio of 83 resistant : 9 susceptible seedlings gave satisfactory agreement with a 15:1 ratio (χ^2 = 1.95, P>0.10). Thus *Ur-A* and *Ur-N* are located at different loci and may be genetically independent.

This intercross failed to give any seedlings with an IT lower than 22+. Thus the data support the contention that *Ur-N* is distinct from *Ur-M* (Section 5.3.1.2.6 p.85 and Section 5.3.2.3. p. 97).

5.3.2.5. B2053/PR 5

Genes identified in these stocks were:-

	Gene	IT	Race groups with virulence	Remarks
B2053	*Ur-J*	;N, ;1 or 2	j	Derived from NEP 2
PR 5	*Ur-G*	;	none	

One-hundred-and-fifty F2 seedlings derived from two F1 plants were tested with race h in two separate experiments. The ITs in the first test were ;N similar to ;N seen on B2053. In the second test ITs ;1, similar to B2053 and ;, identical with that shown by PR 5, were observed. No segregates producing ITs distinctly different from those of the parents were seen, hence *Ur-G* and *Ur-J* are either allelic or linked in repulsion with an upper recombination limit of 0.28 at P = 0.05. However, these genes are not identical since they may be distinguished using a race with virulence on differential j carrying *Ur-J*.

The presence of seedlings with purple hypocotyls resembling those of the PR 5 male parent indicated that the populations tested were hybrids.

5.3.2.6. PR 5/Cornell 49-242

Genes identified in these stocks were:-

	Gene	IT	Race groups with virulence
PR 5	*Ur-G*	;, occasionally 2-22+ & 2+NR	none
Cornell 49-242	*Ur-H*	;1	none

F2 plants derived from one F1 plant were tested with race h in two separate experiments. In one test, the ITs were ;, typical of PR 5, ;1 typical of Cornell 49-242 and 2-22+3-3, somewhat similar to the IT seen occasionally on PR 5. In the second test, plants with ;, ;1, 2-, 2-22+2- and 2+3- were seen. However, no susceptible recombinants were recovered among the 61 seedlings tested. Since the earlier genetic analyses indicated that only *Ur-G* and *Ur-H*, respectively, were present in these stocks and that seedlings with *Ur-G* sometimes produced ITs as high as 2+3-, all individuals were considered to be parental types. The small population barely exceeded the minimum size of 46, which, at P = 0.05, is needed to distinguish segregation at two independently inherited loci from allelism, and is smaller than the 71 needed to perform the same distinction at P = 0.01. Consequently, the upper recombination value of 43 *per cent* (P = 0.05) is high.

While there were no obvious markers to indicate that the seedlings were derived from hybrids rather than selfed plants, the variation in ITs suggested hybrid origin. Furthermore, this variation suggested that even if the genes do not recombine, they may be different.

5.3.2.7. Cornell 49-242/Aurora

The genes identified in these accessions were:-

	Gene	IT	Race groups with virulence	Relationship
Cornell 49-242	*Ur-H*	;1	none	
Aurora	*Ur-M*	;N	none	Either linked,
	Ur-N	2+2 or 2+3-	none	r = 2+4, or identical

The F2 seedlings derived from two F1 plants showed four ITs, ;N which is typical of *Ur-M* in Aurora, ; and ;1 typical of *Ur-H* in Cornell 49-242 and 3+4. The ratio of 45 resistant : 2 susceptible seedlings indicated variation at two independently inherited loci ($\chi^2_{15:1}$= 0.4, P>0.5). There was no indication that presence or absence of hypocotyl colour was associated with either gene for rust resistance.

Although Aurora was derived from Cornell 49-242 (Anon. 1973a), these results indicate that the particular accessions used for this study do not share any genes for rust resistance.

No recombinants with IT 2+3- were observed as in Section 5.3.2.3 (p. 97), but with close coupling linkage with *Ur-M*, such individuals would be expected to be rare.

5.3.2.8. NEP 2/Cornell 49-242

Genes identified in these accessions were:-

	Gene	IT	Race groups with virulence
NEP 2	*Ur-F*	; to 12-	e
	Ur-I	;N	none
	Ur-J	;1 to 2	j
	Ur-K	2-/2-WA	none
Cornell 49-242	*Ur-H*	;1	none

F2 populations were derived from six F1 plants. In tests with race efgj, the three ITs observed on resistant plants were ;N, typical of *Ur-I*, ;1, typical of *Ur-H* and 2-/2-WA typical of *Ur-K*. The ratio of 220 resistant : 6 susceptible seedlings showed satisfactory agreement with an hypothesis of three independently segregating loci ($\chi^2_{63:1}$= 1.76, P>0.4).

Progeny-tests on five of the six plants susceptible to efgj, with races h, egh and efgj indicated all were genotype *Ur-J Ur-J*.

In the analysis of NEP 2, *Ur-I* and *Ur-K* segregated independently. In the intercrosses B2053 (*Ur-J*)/PR 5 and PR 5/Cornell 49-242, no susceptible recombinants

were recovered. This suggested that *Ur-J* from NEP 2 and *Ur-H* from Cornell 49-242 may be allelic or closely linked in repulsion. The results of progeny-tests on plants susceptible to race efgj in the present cross supported this hypothesis.

5.3.2.9. Bonita/B2052

The genes identified in these accessions were:-

	Gene	IT	Race groups with virulence
Bonita	*Ur-L*	2/2WA	none
B2052	*Ur-K*	2-/2-WA	none

F2 populations derived from two F1 plants were infected with race h in two separate experiments, (i) and (ii). The IT classes observed in the two tests and the frequencies of seedlings in them are presented in Table 47. The ITs seen in the two tests were slightly different, with a greater proportion of plants being classified in the lower IT classes in test (ii) than in (i). Susceptible recombinants were recovered and the pooled frequencies of 243 resistant : 21 susceptible seedlings agreed satisfactorily with expectation for two independently segregating loci ($\chi^2_{15:1} = 1.31$, $P>0.2$). The lower ITs of some F2 plants relative to the parents indicate interaction between *Ur-K* and *Ur-L*, or between either gene and the genetic background.

In order to test for interaction between *Ur-K* and *Ur-L*, the following models were considered:-

(a) If double homozygotes were necessary to produce the lower ITs, a ratio of 1:14:1 would be predicted. The frequencies of 10:25:4 in test (i) and 44:164:17 in test (ii) gave poor agreements with this ratio ((i) $\chi^2 = 26.9$, $P<0.001$; (ii) $\chi^2 = 119.62$, $P<0.001$).

(b) If the homozygote of one gene interacted with a heterozygote of the other, the predicted ratio was 5:10:1. The frequency of 10:25:4 in (i) showed satisfactory agreement with this ratio ($\chi^2 = 1.26$, $P>0.5$), but that of 44:164:17 (ii) showed poor agreement ($\chi^2 = 14.48$, $P<0.001$).

Table 47. Frequencies of seedlings in IT groups in F2 populations of Bonita/B2052

Test	Parents			F1	F2								
	Bonita		B2052										
	2/2-WA	22+2WA	2-/2-WA	;	;	;/;WA	12-/12-WA	2-/2-WA	2/2WA	22+2+2WA	2+3-3	3-	3+ or 3+4
(i)	4		4	2	8	2		11	11	3			4
(ii)		4	5		44		60		87		5	12	17

(c) If the heterozygote of *Ur-K* interacted with that of *Ur-L* to give the lower IT, the expected ratio is 9:6:1. The frequencies of 10:25:4 in (i) and 44:164:17 in (ii) gave poor agreements with this ratio ((i) χ^2 = 172.77, P<0.001; (ii) χ^2 = 129.62, P<0.001). Pooling of the two lower classes in (ii) also gave poor agreement (χ^2 = 9.18, P<0.01). It is possible that environmental conditions in test (i) permitted accurate classification of seedlings homozygous at one locus and heterozygous at the other, but those in test (ii) did not.

A further hypothesis is that there was interaction between *Ur-K* and the factor(s) responsible for slight restriction of pustule size as observed in Section 5.3.1.2.5. However, this aspect was not investigated.

Interactions giving lower ITs may be of potential value in plant breeding as they may provide means of selection for gene combinations. However, the combination of *Ur-K* and *Ur-L* need not be of value. Among the progeny of Sanilac/Bonita some plants with *Ur-L* were as susceptible as Sanilac whereas others lacking this gene were as resistant as Bonita (Section 5.3.1.2.5. p. 81).

5.3.2.10. Redlands Greenleaf B//Actopan/Sanilac Selection 37

Genes identified earlier in these accessions were:-

	Gene	IT	Race groups with virulence	Remarks
Redlands Greenleaf B	*Ur-C* *Ur-D* *Ur-Red*	22-NR22- ; 22+3-3	h (d) and d d	*Ur-C* and *Ur-D* independent. *Ur-D* and *Ur-Red* appear to be closely linked.
Actopan/ Sanilac Selection 37	*Ur-E* *Ur-F*	;1 ;1	none e	*Ur-E* and *Ur-F* independent

For this cross, F3 lines were produced and three races, h, egh and efgj were chosen to test the relationships of four loci, *viz.* those involving *Ur-C*, *Ur-D*, *Ur-E* and *Ur-F*. The ITs of the parents and their component genes when tested with the three races are given in Table 48.

Table 48. Infection types of Redlands Greenleaf B//Actopan/Sanilac Selection 37 and their component genes when tested with three races

Accession	Component genes *Ur-*	Race		
		h	egh	efgj
Redlands Greenleaf B	*C D Red*	;	;	;
Brown Beauty	*C*	3+4	3+4	2-NR to 2-2
B2113	*D Red*	;	;	;
Actopan/Sanilac Selection 37	*E F*	; to ;1	; to ;1	; to ;1
B2055	*E*	; to 12-	; to 12-	; to 12-
B2054	*F*	; to 12-	3+4	3+4

Race h. This race was used to test the genetic relationship of *Ur-D* with *Ur-E* and *Ur-F*. Lines in which few or no susceptible seedlings were detected were retested to give a minimum population of 35 per line. This number enabled lines segregating at one locus to be distinguished at P = 0.05 from those which were segregating at two or three loci, or were homozygous resistant. The distribution of F3 lines, given in Table 49 was consistent with expectation for three independently inherited genes.

Race egh. This race, virulent on seedlings with *Ur-C* and *Ur-F* enabled a test of linkage of *Ur-D* and *Ur-E*. Seventy-four lines were either homozygous resistant or segregating and two were homozygous susceptible, a distribution which conformed with expectation for segregation of two independently inherited loci ($\chi^2_{15:1} = 2.12$, $P>0.10$).

Race efgj. This race is avirulent for *Ur-C*, *Ur-D* and *Ur-E* but is virulent for *Ur-F*. Seventy-five lines were either homozygous resistant or segregating, whereas one line was homozygous susceptible, a ratio consistent with expectation for segregation at three independently inherited loci ($\chi^2_{63:1} = 0.03$, $P>0.5$).

Thus the loci involving *Ur-C*, *Ur-D*, *Ur-E* and *Ur-F* appear to be independently inherited.

Table 49. Distribution of reaction classes of F3 lines of Redlands Greenleaf B // Actopan/Sanilac 37 when tested with race h

Reaction classes	Ratios	
	Expected	Observed
Homozygous resistant	37	
Segregating at 3 loci	8 } 57 = 67.69	69
Segregating at 2 loci	12	
Segregating at 1 locus	6 6 = 7.12	7
Homozygous susceptible	1 1 = 1.19	0
Total	64 64 = 76	76
$\chi^2_{57:6:1}$ = 1.22		

5.3.2.11. B2055/PR 5

Genes identified in these stocks were:-

	Gene	IT	Race group with virulence	Source
B2055	*Ur-E*	;1	none	Actopan/Sanilac Selection 37
PR 5	*Ur-G*	;	none	

F2 populations derived from two F2 plants were inoculated with race h. The ITs seen on resistant F2 plants were ;1, 12-, 2-2 and 2+3-, whereas PR 5 exhibited IT ; and B2055 showed IT ;1. The ratio of 53 resistant : 7 susceptible seedlings suggested segregation at two independently inherited loci ($\chi^2_{15:1}$= 2.98, P>0.05).

There were no indications of linkage involving *Ur-E* or *Ur-G* with stem colour.

5.3.2.12. B2055/Cornell 49-242

Genes identified in these stocks were:-

	Gene	IT	Race groups with virulence	Source
B2055	*Ur-E*	;1	none	Actopan/Sanilac Selection 37
Cornell 49-242	*Ur-H*	;1	none	

F2 populations derived from two F1 plants were tested with race h. The only IT produced on resistant F2 plants was ;1 typical of both genes. The observed frequency of 56 resistant : 5 susceptible seedlings suggested variation at two independently segregating loci ($\chi^2_{15:1}$= 0.39, P>0.5). Both genes were inherited independently of stem colour.

5.3.2.13. B2051/B2052

Genes identified in these stocks were:-

	Gene	IT	Race groups with virulence	Source
B2051	*Ur-E*	;1 to 2-	none	NEP 2
B2052	*Ur-K*	2/2WA	none	NEP 2

Although the results of inheritance studies involving NEP 2 suggested linkage, there were no indications as to the genes involved. F2 populations derived from two F1 plants in cross B2051/B2052 were infected with race h to test for linkage between *Ur-F* and *Ur-K*.

The ITs observed on resistant F2 plants were ;1 and 2-, typical of *Ur-F* and 2/2WA and 22+/2WA resembling the *Ur-K* parent. The ratio of 77 seedlings with ITs ;1 and 2- : 23 with 2/2WA and 22+/2WA : 10 susceptible showed close agreement with the ratio 12:3:1 (χ^2 = 0.64, P>0.7) indicative of independent segregation of two genes conferring distinguishable ITs.

5.3.2.14. Allocation of gene symbols

Fourteen genes were tentatively designated *Ur-A* to *Ur-M*. The existence of two other genes, *Ur-N* and *Ur-Red*, was not established beyond doubt.

Because the studies were designed to gain a broad perspective of variation in the host and pathogen in a restricted time, the data available on the identities and relative locations of the genes are insufficient for permanent naming of all genes.

Each of the genes *Ur-A*, *Ur-B*, *Ur-C*, *Ur-D*, *Ur-F* and *Ur-J* were identified by unique reaction patterns with Australian races. Some indication of gene location was provided by certain combinations of genes in differentials and other accessions, since genes occurring in combination must be at different loci. For example, Gallaroy Genotype II has *Ur-A* and *Ur-B* which were linked, r = 25±9 *per cent.*, and Redlands Greenleaf B has *Ur-C* and *Ur-D* which were independent.

In addition, reaction patterns and ITs with a range of races, as well as parentages suggest that 643 carries *Ur-A*, *Ur-B* and *Ur-D* and that Redlands Greenleaf C carries *Ur-B* and *Ur-C*.

The linkage relationships determined in the crosses with Sanilac and in intercrosses are summarized in Table 50. No susceptible recombinants were recovered in the cross involving *Ur-B* and *Ur-E*, despite the testing of more than 6000 plants in F3 lines known to be heterozygous for *Ur-E*, as well as smaller numbers of other F3 lines classified as homozygous for this gene, or its recessive allele. This suggests either allelism or close linkage. If *Ur-B* and *Ur-E* are not at the same locus, an estimate of the maximum recombination value separating them at P = 0.05 is 4 *per cent.* For purposes of nomenclature, non-recombining genes were considered to be allelic if their maximum recombination values at P = 0.05 were less than five *per cent.* On the other hand, where the values equalled or exceeded five *per cent*, as with *Ur-I* and *Ur-M* and with *Ur-G*, *Ur-H* and *Ur-J*, the genes retain their temporary symbols.

However, even though *Ur-G*, *Ur-H* and *Ur-J* may be allelic, they are not likely to be identical. *Ur-J* is clearly different from *Ur-G* and *Ur-H* on the basis of reaction patterns with Australian races. At CIAT, in Colombia, PR 5 remained resistant in the field during 1974, whereas Cornell 49-242 was susceptible (Ballantyne unpublished data). If the same gene is conferring resistance in both

Table 50. Summary of relationships between genes for seedling resistance as determined in (i) crosses with Sanilac and (ii) intercrosses

	Genes *Ur-*	Relationship
(i)	*A B*	Linked r = 25 ± 9%
	M N	Linked r = 2 ± 4% or identical
	C D	Independent
	E F	Independent
	F I J K	Linkage between certain genes suspected
(ii)	*E M*	Linked r = 21 ± 9%
	B E	Allelic mrv† = 4%
	I M	Allelic mrv = 30%
	G J	Allelic mrv = 28%
	G H	Allelic mrv = 43%
	A N	Independent
	E H	Independent
	E G	Independent
	F K	Independent
	H M N	Independent
	K L	Independent
	C D E F	Independent

† mrv = maximum recombination value at P = 0.05

countries and if the genotypes of Cornell 49-242 and PR 5 in this collection are the same as those at CIAT in respect of rust reaction, then the genes they carry, *Ur-H* and *Ur-G*, respectively, are not identical.

In the linkage group presented in Figure 3, Part I, *Ur-A* was taken as a reference point. Possible locations of *Ur-B*, *Ur-E* and *Ur-M* are given in I (i), (ii) and (iii). In part II of this Figure, *Ur-M* is taken as the reference point and possible locations of *Ur-I* and *Ur-N* are presented in II (i) and (ii). Genes *Ur-A* and *Ur-N* appeared to segregate independently.

Because these data clearly indicate that *Ur-A* and *Ur-B* are at separate loci, they can be given permanent symbols, *Ur-1* and *Ur-2*, respectively. *Ur-E* takes the form $Ur\text{-}2^2$ since it was allelic with *Ur-2*.

Because there is doubt that *Ur-M* and *Ur-N* are distinct genes, *Ur-M* is named *Ur-3* and *Ur-N* retains its temporary designation. In Section 5.3.1.2.6 (p. 85) *Ur-M* and *Ur-N* were proposed as distinct genes on the basis of IT. The failure to recover recombinants with an IT lower than 2+3- in the intercross B2056(*Ur-N*)/ B1627(*Ur-A*) supported the hypothesis that *Ur-M* and *Ur-N* are distinct genes. However, data from the intercross B2055(*Ur-E*/Aurora) tended to contradict this conclusion. The considerable discrepancy between observed and predicted numbers in IT classes in this cross suggested that B2056 possesses *Ur-M* and that the genetic background modified the expression of *Ur-M* to an intermediate level. In addition, the genetic ratios in Sanilac/Aurora gave satisfactory agreement with a single-gene hypothesis (Section 5.3.1.2.6., p.85).

The location of a culture virulent for *Ur-M* but avirulent for *Ur-N* and its use in tests with Aurora, B2056 and Sanilac would enable a critical assessment of the results.

There are insufficient data for location and permanent naming of the other eight genes. However, it would appear that there are more than the three loci named above. NEP 2 was found to carry four genes, only one of which, *Ur-I*, was involved in linkage (Figure 3). Linkage of *Ur-I* with another of the remaining

Figure 3. Diagram of linkage group giving possible locations of six genes *Ur-*

Ur-A = *Ur-1*
Ur-B = *Ur-2*
Ur-E = *Ur-2*²
Ur-M = *Ur-3*

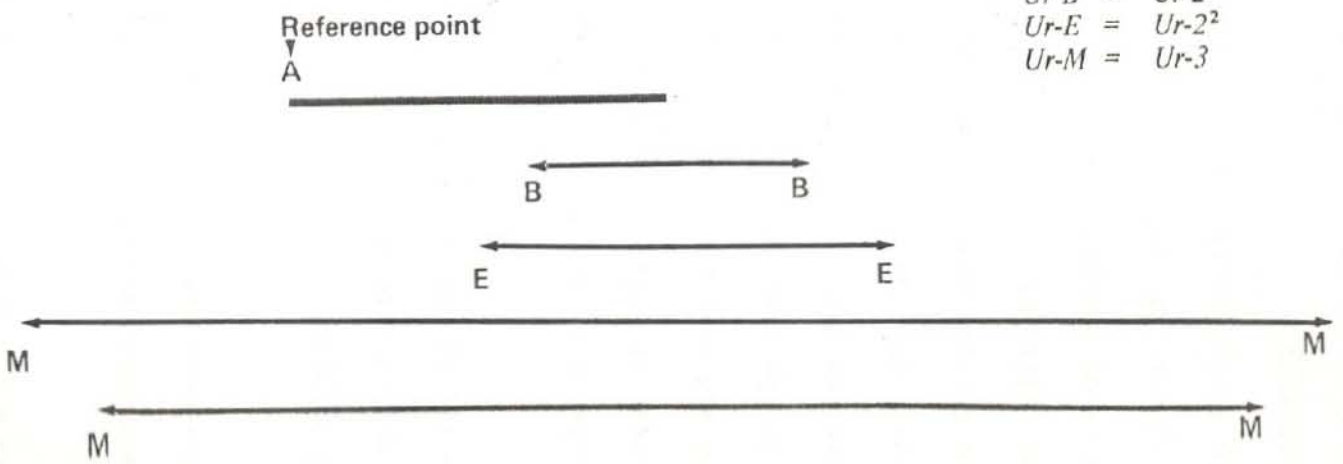
I

(i) A & B r = 25 ± 9

(ii) B & E mrv = 4

(iii) E & M r = 21 ± 9

OR mrv = 25



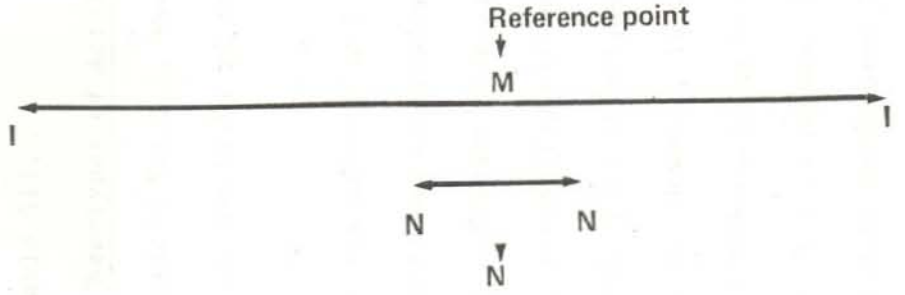
II

(i) I & M mrv = 30

(ii) M & N r = 2 ± 4

OR are identical

(iii) A & N segregate independently



Scale

1 unit = 2 mm

recombination value ⟷ confidence limits or standard error distances

three NEP 2 genes is possible, hence at least two other loci may be postulated. In addition, the results of the intercross Redlands Greenleaf B//Actopan/Sanilac Selection 37 indicate that the four genes present (*Ur-2*^{2}, *Ur-C*, *Ur-D*, *Ur-F*) were likely to be independently inherited. Only one of these, *Ur-2*^{2}, was involved in linkage.

The ITs of each member of the gene pairs which yielded no susceptible recombinants were generally similar (Table 45). However, two gene pairs with similar ITs segregated independently, *viz*. *Ur-I* and *Ur-N*, with ITs 2+2 or 2+3- and *Ur-K* and *Ur-L*, with ITs 2-/2WA or 2/2+WA.

More extensive testing of certain intercrosses which yielded no susceptible recombinants and of additional intercrosses are necessary to locate all the genes isolated (Table 51).

5.3.2.15. Phenotypes of Actolac and Actosan

In Section 5.3.1.2.1. the genotype of Actopan/Sanilac Selection 37 was established on the basis of tests on F1, F2, F3 and F4 generations as *Ur-E Ur-E Ur-F Ur-F*.

The results of testing F2 populations of Sanilac/Actolac and Sanilac/Actosan in 5.3.1.2.7. were not conclusive. However the ITs, reaction patterns and ratios suggest both cultivars possessed *Ur-E* and *Ur-F* and that Actolac carried an additional gene, resembling either *Ur-J* or the resistance factor(s) in differential f.

The single gene effective against the three races used to test these crosses resembles *Ur-E*. However, in the absence of a culture virulent for *Ur-E*, intercrossing of B2055 carrying *Ur-E*, with Actolac and Actosan would be necessary to confirm that *Ur-E* is present in these cultivars.

The standard tests used in crosses with Sanilac and other resistant accessions in Section 5.2.10.1. (p. 37) would indicate if *Ur-F* is present in these cultivars, and if *Ur-J* or the gene(s) present in differential f is also present in Actosan.

5.3.2.16. Phenotypes of the races isolated

A genetic study of resistance in the host also provides data on the phenotype

Table 51. Summary of relationships of seedling resistance genes to each other

	Ur-A	*Ur-B*	*Ur-C*	*Ur-D*	*Ur-E*	*Ur-F*	*Ur-G*	*Ur-H*	*Ur-I*	*Ur-J*	*Ur-K*	*Ur-L*	*Ur-M*	*Ur-N*
Ur-A Ur-1		Linked r = 25 ± 9		P † 643	PGGII IC B/E ‡		PGGII IC B/E	PGGII IC B/E	Fig. 3				Fig. 3	Indep.
Ur-B = Ur-2			P RGC	P 643	Allelic	Indep *								
Ur-C				Indep	Indep	Indep								
Ur-D					Indep	Indep								
Ur-E = Ur-2						Indep		Indep					Linked r = 21 ± 9	
Ur-F							Indep			P NEP 2	P NEP 2 Indep			
Ur-G										Allelic? mrv = 28				
Ur-H										Allelic? mrv = 43			Indep	Indep
Ur-I										P NEP 2	P NEP 2		Allelic? mrv = 30	
Ur-J											P NEP 2			
Ur-K												Indep		
Ur-L														
Ur-M = Ur-3														Linked r = 2 ± 4 or identical
Ur-N														

† Parentage GGII = Gallaroy Genotype II; RGB = Redlands Greenleaf B; RGC = Redlands Greenleaf C.

‡ IC - Intercross.

* Indep - independently inherited.

of the pathogen. A listing of the phenotypes of the 22 races isolated in Section 3 is presented in Table 52. The symbols *Ur-Gol*, *Ur-Epi* and *Ur-Ver* are used to refer to the hypothesized genes present in differentials c (Golden Gate Wax), g (Epicure) and f (Veracruz 1A6). There is no evidence to suggest that each carries more than a single gene for resistance. However, it should be emphasized that each must carry at least one gene which is both different in each tester and different from those isolated in the genetic studies.

5.3.2.17. Relationship of genes for rust resistance to genetic markers

Since there was variation in growth habit and presence or absence of seed coat colour in certain accessions used in crosses, breeding behaviour of certain progenies were examined in order to test for linkage with certain genes for rust resistance. In certain crosses the presence or absence of purple pigment in the stem, a character generally associated with presence or absence of seed coat colour, was classified as an additional test. There was no evidence for linkage of genes for rust resistance with these markers.

Table 52. Phenotypes of 22 races of *U. appendiculatus* isolated in Section 2, expressed as avirulence/virulence formulae

Race	Formula†
h	*P-1,2,D,F,J,Gol,Epi,Ver/p-C*‡
eh	*P-1,2,D,J,Gol,Epi,Ver/p-C,F*‡
dh	*P-1,2,F,J,Gol,Epi,Ver/p-C,D,Red*
dfh	*P-1,2,F,J,Gol,Epi/p-C,D,Red,Ver*
(a)(d)h(i)	*P-1,F.J.,Gol,Epi,Red,Ver/p-2,C,D*
(a)c(d)h(i)	*P-1,F,J,Epi,Ver/p-2,C,D,Gol,Red*
(a)c(d)eh(i)	*P-1,J,Epi,Ver/p-2,C,D,F,Gol,Red*
(a)dhi	*P-1,F,J,Gol,Epi,Ver/p-2,C,D,Red*
(a)cdhi	*P-1,F,J,Epi,Ver/p-2,C,D,Gol,Red*
(a)dfhi	*P-1,F,J,Epi,Gol/p-2,C,D,Red,Ver*
a(d)h(i)	*P-F,H,Epi,Gol,Red,Ver/p-1,2,C,D*
adhi	*P-F,J,Epi,Gol,Ver/p-1,2,C,D,Red*
adehi	*P-J,Epi,Gol,Ver/p-1,2,C,D,F,Red*
a	*P-C,F,J,Epi,Gol,Ver/p-1,2,D*‡
af	*P-C,F,J,Epi,Gol/p-1,2,D,Ver*‡
gh	*P-1,2,D,F,J,Gol,Ver/p-C,Epi*‡
eg	*P-1,2,C,D,J,Gol,Ver/p-F,Epi*‡
egh	*P-1,2,D,J,Gol,Ver/p-C,F,Epi*‡
fg	*P-1,2,C,D,F,J,Gol/p-Epi,Ver*‡
efgj	*P-1,2,C,D,J,Gol/p-F,J,Epi,Ver*‡
ch	*P-1,2,D,F,J,Epi,Ver/p-C,Gol*‡
cfh	*P-1,2,D,F,J,Epi/p-C,Gol,Ver*‡

† As *Ur-3*, *Ur-E*, *Ur-G*, *Ur-H*, *Ur-I*, *Ur-K*, *Ur-L* and *Ur-N* were effective against all races and were common to all formulae, they were omitted.

‡ Pathogenicity of these races on *Ur-Red* is unknown, except that culture 74.27, race h is *p-Red*, on the basis of reaction patterns and ITs when this was tested on segregating lines of Sanilac/Redlands Greenleaf B.

6. SURVEY OF ACCESSIONS OF *Phaseolus vulgaris*

6.1 Materials and Methods

A germplasm collection comprising 534 entries was tested with a range of cultures. The collection included commercial cultivars, advanced breeding lines, rust differentials used in earlier Australian and overseas surveys, and accessions with reported resistance to rust and other diseases. One-hundred-and-nineteen of the accessions were included in the International Bean Rust Nurseries (IBRN) for 1975 and 1976. Details of rust reactions and other features of certain accessions are listed in Rey and Lozano (1961), Hudson *et al.* (1973), Westphal (1974), Pompeu (1975), Zaumeyer and Meiners (1975), Anon. (1976b), Groth and Shrum (1977), Meiners and Rogers (1977) and Schwartz and Galvez (1977). While an abbreviated notation, *e.g.* PR 5, adopted by bean rust workers was used for the 22 lines distributed by Dr. N. Vakili, U.S.D.A., Puerto Rico, the full designations are included in Appendix 1.

Generally, seedlings from seven seeds sown in a single pot were inoculated with each culture, but fewer seedlings were tested when seed supplies were limited. Inoculation was carried out at the rate of approximately 8 - 10 ml of spore suspension to 50 pots situated in an area of 0.5 m^2.

Seedling tests on the collection were carried out as two experiments. The first group of 340 accessions was tested with all eleven races available in 1974, *viz.* h, ch, dh, eg, fg, gh, cfh, egh, (a)dhi, a(d)h(i) and (a)dfhi. Accessions resistant to all races were subsequently screened with efgj, adehi and (a)c(d)h(i). The second group of 194 accessions was screened with eight races, *viz.* h, dh, fg, gh, cfh, efgj, (a)dfhi and adehi. Those resistant to these races were tested with (a)c(d)h(i). This more limited testing was possible because of the more complex races available, and experience with the first group. Other races were used for discriminatory tests where appropriate.

The accessions were grouped according to reaction patterns and ITs, with ITs ;, 1 and 2 being regarded as low and 2+2 and 2+3- as intermediate. These ITs were

regarded as indicating host resistance, whereas ITs 3+ and 3+4 were regarded as high and indicating host susceptibility. Hypothetical phenotypes were allocated to all accessions reacting differentially or showing distinctive ITs such as that of Bonita with *Ur-L* . Only genes conferring resistance are listed.

6.2. Results

6.2.1. CLASSIFICATION INTO HOST PHENOTYPE GROUPS

Twenty-nine groups were differentiated on the basis of reaction patterns and ITs. The type accessions and hypothetical phenotypes of the 24 groups where these could be assigned, together with the numbers of accessions in each group, are given in Table 53. Details of ITs of these groups with 12 races are presented in Table 54. The accessions within host groups showed similar or identical ITs, but three groups, *viz.* 8, 13 and 23 were subdivided because even though the reaction patterns were similar, the ITs were different.

Group 1 was susceptible to all Australian races, whereas Groups 2 to 15 were susceptible to one or more races. Other groups, except 28 and 29, showed seedling resistance to all Australian races. Hypothetical phenotypes were assigned to Groups 16 and 18 on the basis of ITs with the 12 races, and to Group 19 on the basis of both parentage (Appendix 2) and the ITs which suggest phenotype *Ur-1 Ur-Gol*. Groups 25 and 26 were resistant to all races, but ITs of Group 25 were consistently higher, *e.g.* IT 2+2 or 2+3-, than those of Group 26, *e.g.* ITs ;, ;1, ;N or 2-. Certain accessions, *e.g.* Westralia, showed no signs of infection and were rated IT 0 in some tests. However, in other tests chlorotic flecks were seen. Group 27 comprised accessions resistant to all races but showing different ITs, *e.g.* ;, 2+2 , in tests with a single race. Accessions consisting of mixtures of resistant and susceptible plants were classified with the particular phenotypic groups of the resistant plants where this was clearly indicated, but were otherwise placed in Group 28. Group 29 included accessions whose reactions varied from intermediate to high in experiments with different races, but in no discernible pattern. However, when these were inoculated separately with a range of races, and all plants kept in one greenhouse under similar conditions, they reacted similarly with all

Table 53. Twenty-four of twenty-nine reaction groups classified according to ITs and reactions with a range of cultures, including type accessions and hypothetical phenotypes, where applicable

Group	Type accession		Hypothetical phenotype	No. of accessions in group
	Number	Name	*Ur-*	
1	B1555	Sanilac	-	165
2	B1627	Gallaroy Genotype I	*1*	6
3	B1264	Small White 1C-91	*2*	5
4	B1561	Brown Beauty	*C*	96
5	B1203	Small White UI 40	*D*	2
6	B1558	CCGB 44	*F*	15
7	B2053	Single gene line	*J*	8
8a†	B1556	Golden Gate Wax	*Gol*	7
b	B1797	Bayo Camana	*Gol*ǂ	2
9	B1560	Epicure	*Epi*	10
10	B1559	Veracruz 1A6	*Ver*	9
11	B1629	Gallaroy Genotype II	*1,2*	33
12	B1553	Luna	*2,Gol*	3
13a†	B1431	PR 22	*Epi,Ver*	4
13b	B1597	El Salvador 184	*Epi,Ver*	1
14	B1557	Redlands Greenleaf B	*C,D,Red*	8
15	B1562	Redlands Greenleaf C	*2,C,Red*	3
16	B1232	Bonita	*L*	4
17	B1765	PI 163 372	*K,Gol*	2
18	B1767	PI 207 262	*K,Ver*	2
19	B1341	Colorado 453	*1,Gol*	2
20	B1527	Cundinimarca 116	*F*ǂ	1
21	B1528	Diacol Nutibara	*J*ǂ	1
22	B1787	ICA Pijao	*Ver*ǂ	3
23a†	B1349	PI 203 958	*Epi*ǂ	3
b	B1660	Rufus	*Epi*ǂ	1
24	B1417	PR 8	*C,Epi*	1

† Reaction patterns similar, but ITs different.

ǂ Intermediate IT produced by certain races.

Table 54. Hypothetical phenotypes and typical seedling ITs produced by 26 of 29 groups of accessions in greenhouse tests when inoculated with eight races

Group	Hypothetical phenotype *Ur-*	h	dh	(a)dfhi	adehi	(a)c(d)h(i)	a(d)h(i)	ch	gh	egh	eg	fg	efgj
1	-	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+	3+
2	*1*	2+2	2+2	2+2	3+	2+2	3+	2+2	2+2	2+2	2+2	2+2	2+2
3	*2*	;	;	3+	3+	3+	3+	;	;	;	;	;	;
4	*C*	3+	3+	3+	3+	3+	3+	3+	3+	3+	22-NR	22-NR	22-NR
5	*D*	;	3+	3+	3+	3+	3+	;	;	;	;	;	;
6	*F*	;1	;1	;1	3+	;1	;1	;1	;	3+	3+	;	3+
7	*J*	;1	;1	;1	;1	;1	;1	;1	;1	;1	;1	;1	3+
8a	*Gol*	;1	;1	;1	;1	3+	;1	3+	;1	;1	;1	;1	;1
b	*Gol*	2+2	2+2	2+2	2+2	3+	2+2	3+	2+2	2+2	2+2	2+2	2+2
9	*Epi*	;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	3+	3+	3+	3+	3+
10	*Ver*	;1	;1	3+	;1	;1	;1	;1	;1	;1	;1	3+	3+
11	*1,2*	;1	;1	2+2	3+	2+2	3+	;1	;1	;1	;1	;1	;1
12	*2,Gol*	;1	;1	;1	;1	3+	;1	;1	;1	;1	;1	;1	;1
13a	*Epi,Ver*	;N	;N	;N	;N	;N	;N	;N	2-NR	2-NR	2-NR	3+	3+
13b	*Epi,Ver*	;	;	;	;	;	;	;	;	;	;	3+	3+
14	*C,D,Red*	;	3+	3+	3+	2+3-3	2+3-3	;	;	;	;	;	;

Table 54. Hypothetical phenotypes and typical seedling ITs produced by 26 of 29 groups of accessions in greenhouse tests when inoculated with eight races (cont.)

Group	Hypothetical phenotype *Ur-*	h	dh	(a)dfhi	adehi	(a)c(d)h(i)	a(d)h(i)	ch	gh	egh	eg	fg	efgj
15		;	;	3+	3+	2+3-3	2+3-3	;	;	;	;	;	;
16		22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA	22+/ 2-WA
17		;1	;1	;1	;1	;1	;1	2/2-PA	;1	;1	;1	;1	;1
18		;1	;1	2/2-PA	;1	;1	;1	;1	;1	;1	;1	2/2-PA	2/2-PA
19		;1	;1	;1	;1	2+2	;1	2+2	;1	;1	;1	;1	;1
20		;1	;1	;1	2+2	;1	;1	;1	;1	2+2	2+2	;1	2+2
21		;1	;1	;1	;1	;1	;1	;1	;1	;1	;1	;1	2+2
22		;1	;1	2+2	;1	;1	;1	;1	;1	;1	;1	2+2	2+2
23a		;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	2+2	2+2	2+2	2+2	2+2
b		;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	;N+++	2+3-NR/ 2NR or 3+	2+3-NR/ 2NR or 3+	2+3-NR/ 2NR or 3+	2+3-NR/ 2NR or 3+	2+3-NR/ 2NR or 3+
24		;N	;N	;N	;N	;N	;N	;N	;N	2+	;N	;N	;N
25		2+2	2+2	2+2	2+2	2+2	2+2	2+2	2+2	2+2	2+2	2+2	2+2
26		; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2	; to 2

races, indicating slight resistance influenced by environmental conditions. The accessions in each group are listed in Appendix 3.

6.2.2. LIMITATIONS OF THE HYPOTHETICAL PHENOTYPES

In instances where previous work established a single gene, the reactions with a range of races enabled the phenotype to be given with some confidence. For example, accessions susceptible to races eg, egh, efgj and adehi were resistant to other races avirulent on differential e, and thus were classified with some certainty as Group 6, carrying *Ur-F*. However, in other instances several alternative phenotypes were possible:-

(i) Where phenotypes were assigned solely on the basis of susceptibility to only one or two races, there may be either a previously unnamed gene or a combination of genes. For example, in Group 13 (Table 54), where two subgroups were distinguished on the basis of ITs, two hypotheses may be considered. The simplest is that both subgroups carry *Ur-Epi Ur-Ver*, and this is supported by susceptibility to races fg and efgj and resistance to all others. However, IT data do not support this because the IT ;N with races avirulent on g and IT 2-NR with races eg, gh and egh differs from IT ;N+++ typical of seedlings possessing *Ur-Epi* and IT ;1 or 12- expected on seedlings with *Ur-Ver*. The IT ; observed on El Salvador 184 with all cultures, is even more dissimilar and hence the likelihood of its phenotype being *Ur-Epi Ur-Ver* is much less. The second hypothesis that another gene, *Ur-PR 22*, is present may account for the differences in IT. If so, El Salvador 184 may have only *Ur-PR 22*, conditioning IT ;N and PR 22 may have *Ur-PR 22* plus a second or third gene. Should the presence of *Ur-PR 22* be confirmed by genetic analysis and a stock with *Ur-PR 22* be nominated as differential k, the designation of races fg and efgj may be more precisely given as fgk and efgjk, respectively.

Another example occurs with Group 7 where the simplest phenotype to account for the observed results is *Ur-J*, but other possible phenotypes include combinations of *Ur-F*, *Ur-J*, *Ur-Ver* or *Ur-PR 22*.

(ii) where the particular combination of virulence and avirulence factors present in local races do not permit detection of all genes present. For example, while the phenotypes of Groups 2, 3 and 11 are presented only as *Ur-1* or *Ur-2* or both, *Ur-D* may also be present in the accessions not subjected to genetic analysis. However, as all available races with virulence for *Ur-1* and *Ur-2* also have virulence for *Ur-D*, detection of *Ur-D* in such accessions is not possible without genetic analysis. Similarly, *Ur-D* may also be present in accessions in Group 16 typified by Redlands Greenleaf C, which is closely related to Redlands Greenleaf B (Section 5.3.1.1.3., p. 50). The gene tentatively named *Ur-Red* was not separated from *Ur-D* in this analysis, so the presence of *Ur-Red* in the related Strain C indicates that *Ur-D* is likely to be present also.

A further example concerns the Black Turtle Soup Selection made by Coyne for resistance to Sclerotinia rot†. There is little doubt that one component of this has *Ur-Ver*, as susceptible plants occurred among resistant ones in tests with all the f group of races except efgj which was virulent on all seedlings; all plants gave resistant reactions with other races. The simplest phenotype of the second component is *Ur-J*. However, as this accession was derived originally from a single plant, and allocation of the *Ur-J* phenotype is based on reaction with race efgj, it is more likely that all plants have *Ur-Ver* and that some also possess either *Ur-F* or *Ur-J*, or both.

6.2.3. GENOTYPE OF REDLANDS GREENLEAF B

To assist in resolving the genotype of Redlands Greenleaf B, analysed in Section 5.3.1.1.3. (p. 50), a limited range of accessions was inoculated separately with races h, (a)(d)h(i), (a)dhi, a(d)h(i), and adhi, and maintained in one greenhouse to ensure comparable environmental conditions. The contrasting ITs

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† caused by *Sclerotinia sclerotiorum* (Lib.) De Bary

for Redlands Greenleaf B and Small White UI 40 in tests with the (d) group of races, and the same ITs with other races (Table 55), support the hypothesis that Redlands Greenleaf B carried *Ur-Red* in addition to *Ur-D*, whereas Small White UI 40 carried only *Ur-D*.

These tests did not show *Ur-Red* in the samples of the reported parents of Redlands Greenleaf B, *viz.* 643 and Brown Beauty. One explanation is that the seed stock of one of the original parents, most likely 643, comprised both *Ur-D Ur-Red* and *Ur-D ur-Red* plants; the former being used as a parent and the latter maintained by selfing. It is less likely that Brown Beauty, or Brown Beauty 17A, was the source as there is little doubt that *Ur-D* originated from 643 and there is evidence that *Ur-D* and *Ur-Red* are linked in coupling in Redlands Greenleaf B.

6.2.4. REACTIONS OF INDIVIDUAL ACCESSIONS

Of the 534 accessions screened, 483 were homogeneous for rust reaction and 51 were heterogeneous.

6.4.4.1. Variation of reaction within Australian cultivars

Three Australian cultivars bred for resistance during the past two decades were heterogeneous in reaction to prevalent races. These were Gallaroy (*Ur-1*, *Ur-1 Ur-2*), released in 1966, Actolac (*Ur-F*, *Ur-2*^{2} *Ur-F*) released in 1978 and Ormiston (*Ur-C*, *Ur-C Ur-D Ur-Red*) which was named but not released. Another Queensland cultivar, Redlands Autumncrop (-, *Ur-C*) was heterogeneous in reaction to uncommon races.

6.2.4.2. Variation in reaction of accessions received under the same name but from different collections

There were 24 instances where accessions received under the same name but from different sources reacted similarly. These included two accessions of Bonita and all accessions of the differentials 3, 643, 765, 780, Canario 101 and Golden Gate Wax.

However, in 13 instances, cultivars and PI entries with the same name but

Table 55. Infection types of a range of genotypes derived from Small White types, when tested with races h, dh, (a)(d)h(i), (a)dhi, a(d)h(i) & adhi and hypothetical genotypes of the host accessions

Accession	Race						Hypothetical phenotype *Ur-*
	h	dh	(a)(d)h(i)	(a)dhi	a(d)h(i)	adhi	
Sanilac	3+	3+	3+	3+	3+	3+	-
643	;	;	2+2	2+2	3+	3+	*1 2 D*
Brown Beauty	3+	3+	3+	3+	3+	3+	*C*
Redlands Greenleaf B†	;	3+	22+3-3	3+	22+3-3	3+	*C D Red*
Small White UI 40ǂ	;	3+	3+	3+	3+	3+	*D*
Redlands Greenleaf C§	;	;	22+3-3	3+	22+3-3	3+	*2 C Red*
Small White 1C-91¶	;	;	3+	3+	3+	3+	*2*

† Redlands Greenleaf A, Redlands Pioneer, Ormiston Genotype II } react identically to each other.

ǂ Small White UI 74 reacts identically.

§ Redlands 120-2B reacts identically.

¶ Small White 1C-93 reacts identically.

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from different sources did not behave similarly. For example, three accessions were received as CCGB 44 or its synonym, Costa Rica 2. Accession B1526 from CIAT gave ITs and reaction patterns similar to those of differential e, carrying *Ur-F*, represented by B969 (bulk original) and B1558 (single plant stock) from Johnson, but B1494 (PI 207 198) from United States Department of Agriculture Germplasm Resources produced ITs and reaction patterns similar to differential j, carrying *Ur-J*.

Some accessions of a cultivar or a PI entry were uniformly resistant but a second contained an occasional susceptible plant. These included the differential 814, Great Northern US 1140, PI 207 262 and Rico 23.

6.2.5. DISTRIBUTION OF GENES

The distribution of the 534 accessions among 24 of the 29 groups is summarized in Table 53. The largest groups were those susceptible to all races (Group 1 with 165 accessions), resistant to all races (Groups 25, 26 and 27 with 15, 118 and 12 accessions respectively) and carrying *Ur-C* (Group 4 with 96 accessions). Groups 28 and 29 included 14 and four accessions respectively.

6.2.5.1. Influence of agronomic† or horticultural‡ type

Some genes occurred frequently in certain agronomic or horticultural types of beans. For example, *Ur-C* was present in 79 of the 119 fleshy-podded beans surveyed, but in only one commercial dry bean, Charlottetown. Genes *Ur-1* and *Ur-2* were mostly confined to Small White beans and Great Northern cultivars US 1140, Jules and Tara.

6.2.5.2. Influence of geographic area of origin

Most of the cultivars and breeding lines selected in areas where rust is not troublesome were susceptible to at least some Australian races. Exceptions were Aurora, Bush Romano 14, some accessions of Black Turtle Soup, and three breeding lines from Dr. M.H. Dickson, NYAES, Geneva, New York, U.S.A. However, many of the beans selected in rust-liable areas showed seedling resistance to all races. These comprised 125 of the 232 Central American and South American types, five of the 26 Ethiopian cultivars and three of eight other African introductions.

6.2.6. COMPARISONS WITH REPORTED REACTIONS IN OTHER GEOGRAPHIC AREAS

Although no details of IBRN results are available, brief summeries have been published (Meiners 1977, Schwarz and Galvez 1977). However, caution must be

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† agronomic type refers to dry beans.

‡ horticultural type refers to fleshy-podded beans.

observed in comparing such results, mostly of field reactions, with those determined here on the basis of greenhouse seedling tests.

The beans resistant to all Australian races in these greenhouse tests included 69 of the 119 IBRN entries tested. In the 1975 and 1976 nurseries at 11 and 13 locations respectively, in Australia and the Americas, no entry was immune or resistant at all locations. Of the ten lines which showed at least some resistance at many locations in both years, eight were resistant to all Australian races. These were Compuesto Chimaltenango 3, Cornell 49-242, Ecuador 299, ICA Pijao (*Ur-Ver* +), Mexico 309, San Pedro Pinula, SB-30-iI-PM-PI and Turrialba 1. The other two were PI 226 895 which is susceptible to all Australian races and Redlands Pioneer (*Ur-C Ur-D Ur-Red*) (Schwarz and Galvez 1977).

All other lines which showed field resistance in three North American locations (Meiners 1977) were resistant in these greenhouse tests. These were Actolac, Actopan/Sanilac Selection 37, Actosan, Aurora, Compuesto Chimaltenango 2, Costa Rica 1031, Cuilapa 72, Cuva 168-N, ICA Tui, La Vega, Mexico 235, Miss Kelly, Mulatinho, Negro Jalpatagua, NEP 2, Panamito Corriente, Pinto Serrano, Preto 897, PI 165 426 (black-seeded and white-seeded), PI 163 372 (*Ur-K Ur-Gol*), PI 310 878, PI 313 524, PR numbers 1,2,3,4,6,9 and 19, Venezuela 54, Villa Guerrero, and Vi 1013 (Meiners 1977).

Of the 11 beans with reported horizontal resistance in Central America and South America, seven showed seedling resistance to all Australian races. These were Costa Rica 1031, Preto 897, PR 3, PR 9, Ricobaio 1013, Ricopardo 896 and Vi 1013, most of which are listed above.

Introductions susceptible to at least some Australian races included four cultivars with reported horizontal resistance to rust in Central America and South America. These were Manteigao Preto 20, susceptible to all races, PR 12 (*Ur-C*), PR 15 (*Ur-C*), PR 16 (*Ur-Epi Ur-Ver*) and Rico 23 (B1403 *Ur-F*; B1793 -, *Ur-F*). Cacahuete 72, used as a source of rust resistance in the CIAT programme, carried *Ur-C*. The gene *Ur-Epi* was present singly in PI 150 414 and Wis HBR 72,

and with another gene(s) conditioning an intermediate IT in R-275 and PI 203 958. In some tests with a race virulent on *Ur-Epi*, the Red Mexican cultivar Rufus reacted similarly to PI 203 958 from which it was derived, but in another experiment, Rufus showed a high rather than an intermediate IT (Group 23b, p. 121).

It is difficult to interpret the results of greenhouse testing of 44 cultivars with six North American races carried out by Groth and Shrum (1977). However, if cultivars reacting similarly to the different races are grouped as in Table 56, 41 of the cultivars may be placed in four general categories. The others (Charity, Kentucky Wonder and Mecosta), gave mixed reactions with particular collections. Thus the maximum of 34 "virulence differences" reported for these isolates would appear to be an overestimate when such similarities are considered. Collection W73 is obviously different from the other five, which may not be significantly different from each other. The simplest interpretation of these results is that:-

- Many of the dry beans, plus three fleshy-podded types (Group i) carry one gene for resistance, and this is effective only against W73, and
- Seven fleshy-podded beans (Group ii) possess another gene effective against all collections except W73.
- Additionally, it is possible that the four dry and three fleshy-podded beans in subgroup a of Group iii carry a gene(s) for intermediate IT which is ineffective against W73, but effective against other cultures. Subgroup b of Group iii generally produced higher ITs than subgroup a, and Topcrop showed more promise of suitability as a Universal Suscept than others in Group iii. The hypothetical phenotypes assigned on the basis of Australian tests were also included in Table 56 for the 19 cultivars tested in both studies. The patterns indicate that the phenotypes of the North American pathogen populations differ considerably from those in Australia.

In a study of F2 populations with Brasilian race B11, Great Northern US 1140 was reported to carry a single gene R_{B11} (Augustin *et al.* 1972). With Australian

Table 56. Reanalysis of Table 2 of Groth & Shrum 1977 according to similarity of reactions with different collections, plus hypothetical phenotypes of certain cultivars as determined with Australian isolates

Group	Cultivar	Collection (North America)						Hypothetical phenotype (Australia)
		P1	P3	B5	KW7	W73	K9	
(i)a	EMERSON†	8ǂ	8-9	7-8	7-8	1	7	
	GLORIA	8	9	7-8	7-8	0-1	8	-
	GRATIOT	7	7	6-7	7	0	7	
	PINTO UI 111	8	8	8	8	0-1	8	-
	PINTO UI 114	7-8	7	8	8	0-1	8	-
	ROZA	8	7	7-8	7	0-1	7	-
	SANILAC	6-7	6-7	7	7	0-1	7	-
	SEAFARER	6-7	7	5-6	6	0-1	7	-
	SNOW BUNTING	6	6	6	6-7	0	7-8	
	SNOWFLAKE	6-7	7-8	6-7	7	0-1	7-8	
	SUTTER	7-8	8	8	7-8	0-1	7	
	VALLEY	7-8	8	7	7-8	1	8	
	VIVA	8	7	7	8	0-1	7	-
	US No 3§	7-8	8	7-8	7	1	7-8	-
	814§	6-7	6-7	7	6-7	0	6-7	¶
b	Seminole§	5	5	6	6	0-1	5	
c	Golden Gate Wax	7	7	7	6	3	7	*Ur-Gol*

† Upper case indicates dry beans.

ǂ 0 = no visible reaction, 1 = hypersensitive flecks, 2 = necrotic flecks mixed with small necrotic pustules, 3-9 = non-necrotic pustules of increasing size.

§ Lower case or this symbol indicates fleshy-podded beans. ¶ Resistant to all Australian cultures.

Table 56 .Reanalysis of Table 2 of Groth & Shrum 1977 according to similarity of reactions with different collections, plus hypothetical phenotypes of certain cultivars as determined with Australian isolates (cont.)

Group	Cultivar	Collection (North America)						Hypothetical phenotype (Australia)
		P1	P3	B5	KW7	W73	K9	
(ii)	Bush Blue Lake	1	1	1	1	8	1	
	Cascade	0-1	0-1	0-1	1	8	2	*Ur-C*
	Early Gallatin	1	1	1	1	7-8	1	
	Improved Tendergreen	1	1	1	2	8	1	
	Kinghorn Wax	1	1	1	1	8	1	
	Pencil Pod Wax	2	2	1	2	8	1	
	Tenderette	2	2	1	1	8	1	
(iii)a	CHARLEVOIX	4-5	4-5	4-6	5-9	7-8	4-5	
	MICHICRAN	5	4-5	3-6	6-9	8-9	4	
	MONTCALM	5	5	6	6	8	5	
	ROYAL RED	6-7	6-7	7	5-9	7-8	5-7	-
	Slimgreen	5	5	5	5-8	8	5-8	
	Topnotch	6	7	5-6	8	7	5	
	Truegreen	5	4-5	4-5	7	8	4-5	
b	Topcrop	7	8	7	7	9	8	
	181[§]	6-7	6-7	8	7	8-9	8	*Ur-C*

Table 56. Reanalysis of Table 2 of Groth & Shrum 1977 according to similarity of reactions with different collections, plus hypothetical phenotypes of certain cultivars as determined with Australian isolates (cont.)

Group	Cultivars	Collection (North America)						Hypothetical phenotype (Australia)
		P1	P3	B5	KW7	W73	K9	
(iv)a	AURORA	1	1	1	1	1	1	*Ur-3 Ur-4*
	BLACK TURTLE SOUP	1	1	1	0	0	0	¶
	CHIEF	1	1	2-3	2	0	2-3	*Ur-1 Ur-2*
	643	1	1	1	1	0	1	*Ur-1 Ur-2 Ur-D*
	765	2	2	2	2	1	2	*Ur-1 Ur-2*
	780	2	2	1	1	2	2	–
b	Roma	1	1	2	2	4	2	
Other	CHARITY	7	8	6-7	7	X#	7-8	
	Kentucky Wonder	8	8	7-8	X	0-1	7	
	MECOSTA	5	4-5	4-5	6-7	X	5	

Mesothetic

races this cultivar reacted identically to Gallaroy Genotype II, shown by genetic analysis to possess *Ur-1* and *Ur-2*.

6.2.7. GENES PRESENT IN DIFFERENTIALS

Hypothetical phenotypes of differentials and other testers used in race surveys in other geographic areas are presented in Table 57. The IT of Dade varied between intermediate and high in different environments, so unless its reaction can be stabilized in controlled environmental conditions, it will be unsuitable for inclusion in an Australian set of testers.

Hypothetical phenotypes of differentials and other testers used for race determination in this work, given in Table 58, indicate that some testers have more than one gene and some have genes in common.

6.2.8. GENES PRESENT IN AUSTRALIAN CULTIVARS

Hypothetical phenotypes of cultivars, presented in Table 59, show that *Ur-C* occurs in all green bean cultivars grown commercially in Eastern Australia, whereas *Ur-1*, *Ur-2*, *Ur-D* and *Ur-Red* occur less frequently.

6.2.9. FREQUENCIES OF RACES VIRULENT FOR PARTICULAR HOST GENES

A knowledge of the genes present in the differentials and commercially grown cultivars permits detailed analysis of the race survey data presented in Section 4.3.8. (p. 30).

The minimum frequencies of races virulent for the ten host genes identified by local races and the minimum frequencies of these genes in the beans sampled in the race survey are presented in Figure 4. The parentages of many of the breeding lines of unknown phenotype suggests that they either carry *Ur-C* or are susceptible to all races. These results support the trend evident in Section 4.3.8. (p. 30) that the frequencies of races virulent for genes exposed in broad-acre plantings were higher than of races lacking virulence, except for *Ur-Epi*, which is present in Epicure grown in home gardens.

Table 57. Hypothetical phenotypes of differentials and other testers used in race surveys in other geographic areas

Hypothetical phenotype *Ur-*	Accession number and name
-	B1892 Aguascalientes 13†, B1392 B1734 Canario 101, B1894 Guanajuato 10-A-5, B1486 Idaho Pinto, B1622 Panamito Sanilac, B960 B1485 B1563 Pinto UI 111, B1199 Pinto US 14‡, B963 B1667 B2089 3, B967 B2080 780.
C	B2076 181.
1	B1656 Panamito mejorado‡.
1 2	B1833 Mexico 6, B1656 Panamito mejorado‡, B1679 Pinto US 5, B1199 Pinto US 14‡, B965 B1554 B1595 B1665 643.
Gol	B1797 Bayo Camana§, B1015 B1556 Golden Gate Wax.
2 C Red	B751 B1562 Redlands Greenleaf C.
J+	B1528 Diacol Nutibara.
H	B1672 Cornell 49-242.
unknown - homogeneous	B1844 Aguascalientes 13†, B1654 B1804 Canario LM, B1623 Caraotas, B1655 B1777 Cocasho, B1400 B1786 Cuva 168-N, B1658 Ecuador 66, B1822 Guerrero 6, B1399 Mulatinho, B1484 Turrialba 4, B1725 B2081‡ 814.
unknown - heterogeneous	B1639 B1768 Canario Divex 8120.
unknown - variable	B1729 Dade.

† another accession reacted differently.

‡ some plants only; heterogeneous accession.

§ may possess another gene conferring IT 2+2 or 2+3-.

Table 58. Genes present in differentials and other testers used for race determination in this study

Tester	Genotype *Ur-*	Hypothetical phenotype *Ur-*	
		Combination of genes detected in genetic analysis	Others
Differentials			
a	*1 2 D*		
c			*Gol*
d	*C D Red*		
e	*F*		
f			*Ver*
g			*Epi*
h	*C*		
i		*2 C Red*	
j	*J*		
Other testers			
Actopan/Sanilac			
Selection 37	2^2 *F*		
Aurora	*3 N*		
Bonita	*L*		
Cornell 49-242	*H*		
NEP 2	*F I J K*		
PR 5	*G*		

6.2.10. USE OF RESISTANT ACCESSIONS

6.2.10.1. In current bean production

Seedling resistance effective against all races was detected only in a few cultivars or breeding lines close to requirements for an Australian commercial type. Those which are currently being grown or may enter commercial production are:-

(i) Actolac and Actosan, Navy beans which were bred in Queensland. Some Actolac plants carry only *Ur-F*, and F2 tests in Section 5.2.1.2.7. (p. 90) indicated that

Table 59. Hypothetical phenotypes of cultivars grown commercially in Eastern Australia during race survey period

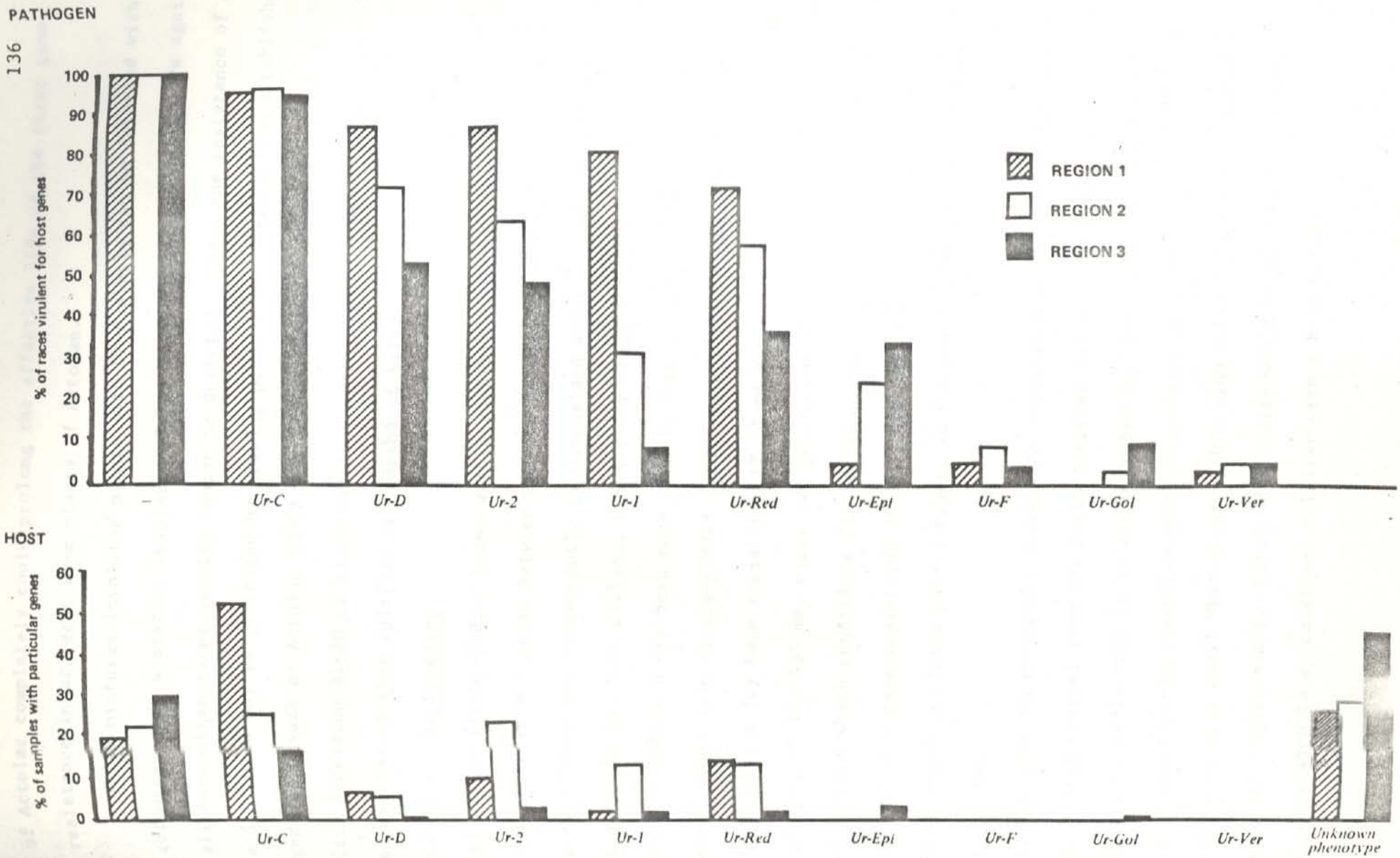
Hypothetical phenotype *Ur-*	Cultivars	
	Dry	Fleshy podded
-	Borlotto, Cannellino, Great Northern UI 31, Limelight, Pinto UI 111, Red Mexican UI 36, Seafarer	
C		Redlands Autumncrop Apollo, Canyon, Bush Blue Lake 274, Gallatin 50, Providor
1, 1 2	Gallaroy	
1 2	Kerman	
2 C Red		Redlands Greenleaf C†
C D Red		Redlands Pioneer

† this replaced Redlands Greenleaf B (*Ur-C Ur-D Ur-Red*) at the beginning of the survey period.

others carry *Ur-F* plus another gene, possibly *Ur-2²*. In addition, F2 studies indicated that at least the plant sampled for crossing in Actosan carried *Ur-F* plus two other genes, one resembling *Ur-2²* and another, possibly *Ur-J* or *Ur-Ver*. Gene *Ur-2²* is effective against all Australian races, while *Ur-F* and *Ur-Ver* are each effective against about 95 *per cent*. of isolates. The frequency of isolates virulent on *Ur-J* is unknown as this gene was not represented in the set of testers on a routine basis. However, only one race, efgj is known to be virulent on seedlings with this gene.

These results and those of the race survey suggest that elimination from Actolac of plants which carry only *Ur-F* could be expected to increase the durability of the combined resistance of other plants in Actolac. At present, races virulent on *Ur-F* have no selective advantage, but if Actolac with some plants carrying *Ur-F* singly was released, these races could be expected to become prevalent, leaving only a single gene to give rust protection in other plants. Withdrawal

Figure 4. Minimum frequencies of races virulent for nine host genes in Regions 1, 2 and 3 and minimum frequencies of host genes in the bean population sampled



of Actolac completely could prolong the effective life of the three gene resistance in at least some plants of Actosan.

While mixtures involving plants with *Ur-F* singly can be detected with the cultures used, a mixture involving plants with single genes effective against all Australian races usually cannot be detected. Hence the resistance of Actolac and Actosan may be prolonged by growing populations derived from established genotypes such as Actolac B2092 and Actosan B2093 (Section 5.2.1.2.7. p. 90).

(ii) Accession B1680 of Black Turtle Soup is used on a small scale, but other accessions of this cultivar are worthy of trial.

6.2.10.2. In breeding

(i) Three fleshy-podded beans with bush habit were B2047, selected for rust resistance from a cross between unnamed accessions of *P.vulgaris* and *P.coccineus* by Waterhouse and subsequently by Queensland workers, and G71.1856 and G71.1318A selected by Dr. M.H. Dickson, U.S.A., for other features. Breeding line GL1 which resembles a dry bean more closely than a fleshy-podded type and G71.1856 were derived from fleshy-podded beans susceptible to common Australian races and PI 165 435, a dry bean resistant (IT ;) to all races. If parentages are as reported, PI 165 435 may carry two genes, one which confers IT ; in G71.1856 and another which conditions 2+2 in GL1. However, this would not explain the presence of a gene conferring IT 2+2, with all races, in G71.1318A derived from two susceptible fleshy-podded types, so the possibility of outcrossing must be considered.

(ii) Two pole fleshy-podded beans with resistance to all Australian races were 814, a differential from the North American set and Westralia, shown to have a single gene resistance in relation to New Zealand races (Yen and Brien 1960). These races closely resembled Australian races at that time.

(iii) Aurora, a Small White bean with a vine habit is close to Australian commercial requirements. Little difficulty would be anticipated in transferring its resistance to a commercial cultivar with a bush habit.

(iv) Accession B1553 Luna, a Pinto bean with hypothetical phenotype *Ur-2 Ur-Gol* is susceptible only to races (a)c(d)h(i), (a)c(d)eh(i) and (a)cdhi, each isolated only once. Thus it has one gene *(Ur-2)*, effective against all races except the (i) and i groups and *Ur-Gol*, effective against all races except the c group. In trials to date, this accession has been of satisfactory agronomic type. One strategy involving a minimum of time and resources would be the transfer of a gene effective against all Australian races, *e.g. Ur-G* in PR 5 or *Ur-H* in Cornell 49-242, to this Luna selection in a backcross programme. This may permit the production of a Pinto bean with two genes effective against all except three rare Australian races, and one gene effective against these. It is desirable that testing with the uncommon race be carried out in isolation and where chances of overwintering are minimal.

(v) Jalisco 33, similar to commercial Pinto types appeared to be a mixture of some plants with *Ur-F* and others resistant to all Australian races. If testing with a range of races confirms this, genetic analyses of selections with resistance to all races would be useful. Should a single gene be detected in any selection, this may be used in a breeding programme, possibly utilizing other Pinto types such as B1553 Luna (*Ur-2 Ur-Gol*), Ouray (*Ur-Gol*), Colorado 453 (*Ur-1 Ur-Gol*) or certain other plants of Jalisco (*Ur-F*). If more than one gene is detected, the selection may be assessed for commercial production.

6.2.11. FIELD EFFECTIVENESS OF GENES FOR SEEDLING RESISTANCE

Data from field reactions of the parental controls of the accessions resistant to all Australian races and of the single gene lines derived from those with combinations of genes have shown that a number of the genes confer a high degree of field protection to a range of cultures (Section 5.3.2.1.8., p. 91). Data from field trials in subsequent sections will indicate the effectiveness of others. Field testing of untrialed lines is necessary before their use is recommended as some genes, for example *Ur-L*, which confer a low IT in greenhouse seedling tests confer little or no protection in the field.

6.2.12. PREDICTION OF RELATIONSHIPS OF CERTAIN GENES

Examination of certain combinations of genes in the hypothetical phenotypes listed in Table 53 enables predictions to be made regarding the relationships of genes. For example, *Ur-1* and *Ur-Gol* which have similar ITs are unlikely to be allelic because there is evidence that they are both present in single plant selections of B1553 Luna, and this is homogeneous for rust reaction.

6.2.13. OTHER OBSERVATIONS

There were no obvious differences between susceptible accessions in times from inoculation to opening of pustules or of attaining maximum size. Thus as far as seedling reactions were concerned, there was no evidence for differences in latent period which could contribute to slow rust development in the field.

7. FIELD TRIALS

The aims of the field assessments were firstly to determine the reactions of a wide range of cultivars and advanced breeding lines, and secondly to study the mode of inheritance of the resistance expressed as slow rusting using inbred populations derived from selected crosses.

7.1. Materials and methods

7.1.1. DERIVATION OF INBRED POPULATIONS FOR INHERITANCE STUDIES

The single seed descent method was used to derive inbred populations from three two-way crosses between Sanilac and two less severely affected cultivars, Apollo and California White Kidney. The latter were chosen because they represent different horticultural types with no known common parentage, thus increasing the likelihood that the rust resistance of each is controlled by different genes. One-hundred-and-twenty F2 plants were grown for each cross, one plant per 10 cm pot. All plants were staked and pots were placed as closely together as possible in the greenhouse. One seed from each plant was used to produce the subsequent generation and this was repeated for three generations. Three to five F5 seeds from each F2-derived line were sown in the field for seed increase. In 15 to 24 months this procedure enabled random populations of near-homozygous lines to be established in sufficient seed quantities for replicated field trials.

7.1.2. EXPERIMENTAL DESIGN

Accessions grouped according to agronomic or horticultural type were sown in two replicates in the summer-1974 trials, but randomized block designs with four replications or plots were used in subsequent experiments. Sowings made in October are described as spring plantings and those made in January as summer plantings.

7.1.2.1. Summer-1974

The aim was to obtain a broad perspective of field reactions of a wide range of host cultivars and advanced breeding lines when exposed to eight different races of the pathogen. One-hundred-and-fifty-nine accessions or entries were

planted at two sites approximately 10 kilometres apart, *viz.* Plant Breeding Institute, Castle Hill and Department of Agriculture, Rydalmere. An additional cultivar, Manteigao Preto 20, was sown only at Castle Hill because of limited seed supplies. Cultivars known to become severely rusted were interspersed through the plantings.

All races then available were released, *viz.* ch, dh, eg, fg, gh, egh, cfh and a(d)h(i).

7.1.2.2. Spring-1974

A smaller trial of 72 entries was sown at Castle Hill to determine reactions to one race, a(d)h(i). This comprised:-

(i) some of the entries which rusted slowly and were slightly affected in the previous trial. However, these may have been susceptible only to one or few of the components of the mixed inoculum.

(ii) green bean cultivars bred or selected in Eastern Australia and not included in the earlier trial.

(iii) dry beans susceptible only to the a(d)h(i) group of races and not previously evaluated for rust reaction under field conditions.

(iv) single plant progenies of Gallaroy which was of mixed genotype for rust reaction in greenhouse tests (Section 4.3.1. p. 22).

7.1.2.3. Summer-1976

Three-hundred-and-fourteen F6 inbred lines were assessed in three separate experiments at Castle Hill. To facilitate sowing, recording and comparisons of reactions these were divided into three experiments, A, B and C, which were planted adjacent to each other on successive days. Each experiment included approximately one-third of the lines from each cross, the parents, and seven additional reference accessions whose field and greenhouse reactions to a range of races has been determined previously. One of these, Bonita, showed seedling resistance in greenhouse tests and developed few pustules in field trials. The other six were susceptible as seedlings to the race released. Five of these,

viz. NB3-S3, Redlands Autumncrop, Redlands Greenleaf Strains B & C and Small White 1C-93 were slightly affected in earlier trials whereas Great Northern US 1140 was severely or very severely rusted.

Race adhi was released onto spreader rows.

7.1.2.4. Summer-1977

Three-hundred-and-thirty-one F6 inbred lines were sown in three experiments as outlined above. An additional planting of 11 lines was also made to determine if the differences between these lines and the parents were significant. These lines were chosen on the basis of reactions in 1976 and represented a range of resistant and susceptible material.

Race adhi was inoculated onto spreader rows on three occasions.

7.1.3. DISEASE ASSESSMENTS

In replicated trials with cultivars and homozygous genotypes the first records were of numbers of pustules per plot in the ranges:-

0 - 4	50 - 100
5 - 9	100 - 299
10 - 24	300 - 499
25 - 49?	500+

These estimates were of pustules which were obvious without turning over the plant. Plants on which no pustules were apparent were examined closely towards the end of the trial.

The approximate date of pod-fill was recorded. After pod-fill or when more than 500 pustules were observed, each plot was classified according to the 0 - 10 rust rating scale outlined in Section 3.2.2.2. (p. 12).

In the first trial with cultivars and advanced breeding lines, entries were ranked according to the mean of

(i) final rust rating

(ii) time to first appearance of rust after inoculum release

(iii) time to develop 500 pustules on each plot after first appearance on the plot (where applicable)

(iv) time to develop 500 pustules relative to pod-fill (where applicable).

The susceptible cultivars developed this level of disease before pod-fill and some of the resistant ones showed 500 pustules after pod-fill. Other resistant cultivars developed fewer than 500 pustules per plot.

Entries which remained free of rust in all replicates were classified as Group A. Entries which were uniformly rusted within each plot were classified as Group B while those showing variation in field reaction within at least one plot were designated Group C. Groups B and C entries were further classified according to an estimate of the numbers of pustules per plot. Those which failed to develop more than 500 pustules on both replicates were classified B(o) and C(o) respectively, whereas those developing this level on one, two, three and four plots were placed in Groups B(i), (ii), (iii), (iv) and C(i), (ii) (iii) and (iv) respectively.

In subsequent trials the results were presented only as mean final rust ratings.

7.1.4. RACE DETERMINATIONS

During the experiments rust-affected leaves were collected for race determinations. In the spring-1974 planting in which a(d)h(i) was released early in the season, another possibly more sensitive method was used. Eight pots, each containing three or four plants of the differentials a, c, e, f, & g were placed in the field area during the evening and moved next morning to a misting chamber for 22 hours.

7.2. Results

7.2.1. FIELD TRIALS WITH CULTIVARS AND ADVANCED BREEDING LINES

7.2.1.1. Summer-1974, Castle Hill and Rydalmere

7.2.1.1.1. Classification of accessions according to field reaction

Twenty-one accessions (Group A) either remained free of rust or developed necrotic flecks or small pustules late in the season at both sites. Of the other lines, 138 at Rydalmere and 139 at Castle Hill where Manteigao Preto 20 was

included, eight (Group C) were mixed within at least one plot and the remainder (Group B) were uniform within each plot.

Entries were ranked in decreasing order of resistance determined by mean results at each site. The four rankings were divided into classes 1 - 5, with approximately the same numbers of entries in each class at each site and for each type of assessment or parameter, as indicated in Table 60. These represented natural divisions as much as possible. For the two parameters where the development of more than 500 pustules was critical for classification, the lower class (1) or classes (1 & 2) were not used. In order to make numbers in each ranking class comparable with those for other parameters, the symbol I was used for B(o) entries. Similarly, where pods were not produced, no measure of rust development in relation to pod fill could be given and the symbol V was used.

A summary of rankings of each entry in decreasing order of resistance is presented in Table 61, together with hypothetical phenotype and maturity. Different type faces have been used in Table 61 to distinguish dry from fleshy-podded beans and those selected in rust-liable areas from others.

7.2.1.1.2. Races present

At Castle Hill, race gh was present in four samples, and in a fifth sample, races dh and egh were recovered. At Rydalmere, races dh, gh and egh were found in the trial and races h, dh, cfh, egh and a(d)h(i) were identified in other collections from an adjacent planting.

7.2.1.1.3. Influence of seedling resistance on field reaction

Entries with seedling resistance to all races were free of rust with the exception of four which showed slight infection. These were PI 203 958 (*Ur-Epi* plus an unknown gene(s)) and Redlands Greenleaf Strains B (*Ur-C Ur-D Ur-Red*) and C (*Ur-2 Ur-C Ur-Red*).

The rust ratings of accessions susceptible to at least one race and classified according to hypothetical phenotype for seedling reaction are in Appendix 4.

Table 60. Criteria for ranking class of four parameters of rust development and number of accessions in each class in summer - 1974 trials (at Rydalmere and Castle Hill)

Parameter	Ranking order class	Rydalmere		Castle Hill	
		Range	No. in class	Range	No. in class
Final rust rating (0-10)	1	1	8	1	9
	2	1.5 to 3.5	25	1.5 to 2.5	23
	3	4 to 7.5	42	3 to 5.5	45
	4	8 to 9.5	44	6 to 8.5	43
	5	10	19	10	19
	Total		138		139
Days to first appearance	1	16-25	7	27 to 32.5, B(0)	11
	2	12-14	22	18 to 23	24
	3	11	50	11.5 to 17.5	45
	4	9-10.5	38	7.5 to 11.0	37
	5	7-8	21	3 to 6	22
	Total		138		139
Days to develop 500 pustules	I†	B(0)	13	B(0)	27
	2	14 to 24 B(i) & (ii)	18	21.5 to 32 B(i) & (ii)	11
	3	10 to 13.5 B(i) & (ii)	46	16.5 to 21 B(i) & (ii)	42
	4	7.5 to 9	30	14 to 16 B(i) & (ii)	42
	5	3.5 to 7	31	7 to 13.5 B(i) & (ii)	17
	Total		138		139
Days after (+) or before (-) pod fill when 500 pustules were recorded.	I†	B(0)	13	B(0)	28
	2	-4 to +7	14	Not used	0
	3	+10 to -5	52	-0.5 to 13.5	45
	4	-20.5 to -12	38	-14 to 1.5	45
	5	-30 to -21	16	-30 to -14	16
	V		5		5
	Total		138		139

† Rating class 1 was not applicable in these situations. However, to maintain consistency of numbers in the classes, I was substituted for B(0) accessions.

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin

Group	Accession		Ranking orders								Maturity[§]	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R[†]	C[ǂ]	R	C	R	C	R	C		
A	B982	*ACTOLAC*[¶#]	0	0	0	0	0	0	0	0	L	*F, 2² F*
	B990	*ACTOSAN*	0	0	0	0	0	0	0	0	L	*2² F + J* or *Ver*
	B980	*ACTOPAN/SANILAC SEL. 37*	0	0	0	0	0	0	0	0	M	*2² F*
	B1366	AURORA[ǂǂ]	0	0	0	0	0	0	0	0	L	*3 4*
	B1680	BLACK TURTLE SOUP	0	0	0	0	0	0	0	0	L	unknown
	B418	*CORNELL 49-242*	0	0	0	0	0	0	0	0	L	*H*
	B1049	*COSTA RICA 1031*	0	0	0	0	0	0	0	0	L	unknown
	B1400	*CUVA 168-N*	0	0	0	0	0	0	0	0	L	unknown
	B1399	*MULATINHO*	0	0	0	0	0	0	0	0	L	unknown
	B1284	*NEP 2*	0	0	0	0	0	0	0	0	VL	*F J I K*
	B2083	*PORILLO NO. 1*	0	0	0	0	0	0	0	0	L	unknown
	B1405	*PRETO 897*	0	0	0	0	0	0	0	0	L	unknown
	B1403	*RICO 23*	0	0	0	0	0	0	0	0	L	*F*
	B1408	*RICOBAIO 1014*	0	0	0	0	0	0	0	0	L	unknown
	B1406	*RICOPARDO 896*	0	0	0	0	0	0	0	0	L	unknown

Key on p. 156.

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking orders								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to podfill			
			R	C	R	C	R	C	R	C		
A	B1796	*VI 1013*	0	0	0	0	0	0	0	0	L	unknown
	B1412	*PR 3*	0	0	0	0	0	0	0	0	L	unknown
	B1418	*PR 9*	0	0	0	0	0	0	0	0	L	unknown
	B1281	*PI 165 426 (BLACK)*	0	0	0	0	0	0	0	0	L	unknown
	B1365	*PI 313 524*	0	0	0	0	0	0	0	0	L	unknown
	B759	*Westralia*‡‡	0	0	0	0	0	0	0	0	VL	unknown
B	B1349	*PI 203 958*	1	0	1	0	I	0	I	I	L	*Epi* + §§
	B1425	*PR 16*	1	1,0	3	0	I	I,0¶¶	I	I	E	*Epi Ver*
	B752	*Redlands Autumncrop*	1	1	1	1	I	I	I	I	E	-, *C*
	B1266	SMALL WHITE 1C-93	1	1	1	1	I	I	I	I	L	*2*
	B1232	*BONITA*	1	1	4	2	I	I	I	I	L	*L*
	B1557	*Redlands Greenleaf B*	1	1	4	2	I	I	I	I	E	*C D Red*
	B1352	PI 226 895‡‡	1	2	2	1	I,5	I	I	2	E	-
	B1441	*Ormiston*	1	2	3	2	I	I	I	I	E	*C, C D Red*

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking orders								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to podfill			
			R	C	R	C	R	C	R	C		
B	B755	*Redlands Pioneer*	2	1	2	1	I	I	I	I	E	*C D Red*
	B751	*Redlands Greenleaf C*	2	1	2	1	I,2	I	2	I	E	*2 C Red*
	B1207	SMALL WHITE 59	2	2	1	1	I	I	I	I	L	*1 2*
	B1234	*BORINQUENA*	2	2	2	2	2	I	2	I	M	*C*
	B1235	CHARLOTTETOWN	2	2	2	2	3	I	3	I	M	*C*
	B767	Valgold	2	2	2	3	I,3	I	3	I	M	*C*
	B977	Wis HBR 72	2	2	2	4	I	I	I	I	M	*Epi*
	B1732	Sunbeam	2	2	3	2	I	I	I	I	M	*C*
	B1730	Goldcrop	2	2	3	2	I,2	I	2	I	M	*C*
	B958	Resistant Kinghorn Wax	2	2	3	2	3	I	3	I	M	*C*
	B1424	*PR 15*	2	2	3	3	I,3	I	3	I	M	*C*
	B1211	WHITE KIDNEY	2	2	3	3	2	I	3	I	M	-
	B801	Canyon	2	2	3	3	3	I	3	I	M	*C*
	B1016	Royal Red	2	2	3	3	3	I	2	I	M	-
	B294	BORLOTTO	2	2	3	3	3	3	4	3	L	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking orders								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R	C	R	C	R	C	R	C		
B	B1210	CALIFORNIA WHITE KIDNEY	2	2	3	4	2	I,2	2	3	M	-
	B1226	SALUGGIA	2	2	4	3	3	3	3	3	M	-
	B1147	SALUGGIA	2	2	4	3	3	4	3	3	M	-
	B1559	643 (SMALL WHITE)	2	3	1	1	I	I	I	I	VL	*1 2 D*
	B1231	SMALL WHITE 38	2	3	1	1	2	4	2	3	L	*1 2 D*
	B1204	SMALL WHITE FM51	2	3	2	2	2	3	2	4	L	*1 2*
	B1697	*B 3088* ** ##	2	3	3	3	2	I,3	2	2	E	*C*
	B1528	*Brown Beauty*	2	3	3	3	3	2	3	3	M	-, *C*
	B941	Tendercrop	2	3	3	3	4	3	3	3	M	*C*
	B1355	Bush Blue Lake 274	3	2	3	2	3	I,4	3	3	M	*C*
	B940	Gallatin 50	3	2	3	3	3	I	3	I	M	*C*
	B617	Apollo	3	2	4	2	3	I,3	3	3	M	*C*
	B1273	SMALL WHITE 1C-101	3	2	4	5	5	2	4	3	L	-
	B1404	*MANTEIGAO PRETO 20*	-	3	-	1	-	5	-	4	L	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking orders								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R	C	R	C	R	C	R	C		
B	B1205	SMALL WHITE FM52	3	3	1	1	3	3	3	3	L	*1 2*
	B1213	BERNA	3	3	2	2	3	3	3	3	M	-
	B1131	ROMAN CRANBERRY	3	3	2	2	3	4	3	3	L	*Gol*
	B909	Coram	3	3	2	3	3	3	2	3	M	*C*
	B1707	Oregon 3606	3	3	3	2	2	3	2	3	M	*C*
	B1731	*Goldcoast*	3	3	3	2	3	I	3	I	M	*C*
	B1743	Nr 66	3	3	3	2	3	I	3	I	M	-
	B1340	OURAY	3	3	3	2	5	4	4	4	L	*Gol*
	B1698	*B 3787* ++ ##	3	3	3	3	2	I,3	3	3	M	*C*
	B976	Wis HBR 40	3	3	3	3	2	5	2	4	M	*C*
	B1693	G4	3	3	3	3	3	3	3	3	M	*C*
	B1212	BROWN SWEDISH	3	3	3	3	3	3	3	3	M	-
	B1745	Nori B30	3	3	3	3	3	3	3	4	M	-
	B1744	Nori A25	3	3	3	3	3	4	3	3	M	-
	B1704	Oregon 3044	3	3	3	4	2	3	3	3	M	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rusting		Period to infection		Rate of increase		Rate of increase relative to podfill			
			R	C	R	C	R	C	R	C		
B	B901	GOUDKORRELL	3	3	3	4	2	3	3	3	M	-
	B1740	CANNELLINO BIANCO	3	3	3	4	2	3	3	3	M	-
	B1709	Oregon 3664	3	3	3	4	3	3	3	3	M	*C*
	B1191	BORLOTTO LINGUA DI FUOCO NANO	3	3	4	3	2	3	4	3	M	-
	B1228	BROWN DUTCH	3	3	4	3	3	3	3	3	M	-
	B1197	TAISHO KINTOKI	3	3	4	3	3	3	3	4	M	-
	B1716	Oregon 3887	3	3	4	4	3	3	3	3	M	*C*
	B1289	SMALL WHITE 1C-111	3	3	4	4	5	3	5	4	L	-
	B1274	SMALL WHITE 1C-102	3	3	4	5	5	I,2	4	3	L	-
	B1672	GCW199##	3	4	3	3	3	4	3	3	M	*C*
	B1705	Oregon 3596	3	4	3	3	5	3	3	3	M	-
	B1209	CANNELLINO	3	4	3	4	5	3	4	4	L	-
	B1713	Oregon 3948	3	4	3	5	4	4	3	4	M	*C*
	B1285	SMALL WHITE 1C-106	3	4	3	5	5	4	4	4	M	-
	B1673	BEKA	3	4	4	3	2	3	4	3	M	-
	B1293	SMALL WHITE 1C-114	3	4	4	5	4	4	4	5	L	-
	B995	Bush Blue Lake 290	3	5	3	2	3	5	4	4	L	*C*

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R	C	R	C	R	C	R	C		
B	B1715	Oregon 4022	4	3	3	3	4	3,I	3	3	M	*C*
	B1703	Oregon 2832-3	4	3	3	4	4	3	3	3	M	*C*
	B1180	GREAT NORTHERN US 1140	4	3	4	2	3	3	2	3	E	*1 2*
	B1708	Oregon 3634	4	3	4	3	3	3	3	3	M	*C*
	B1374	FLAGEOLET ROI DES VERTS	4	3	4	3	5	4	4	4	N	-
	B1267	SMALL WHITE 1C-94	4	3	4	4	4	2	5	4	L	-
	B900	Oregon 58.2	4	3	4	4	4	3	3	3	M	*C*
	B1269	SMALL WHITE 1C-97	4	3	4	4	5	3	5	4	L	-
	B1272	SMALL WHITE 1C-100	4	3	5	4	4	4	4	4	M	-
	B1270	SMALL WHITE 1C-98	4	3	5	5	4	2	4	4	L	-
	B1286	SMALL WHITE 1C-107	4	4	2	4	5	5	4	5	L	-
	B1331	CORVETTE	4	4	2	4	4	4	4	4	L	-
	B1271	SMALL WHITE 1C-99	4	4	2	5	5	4	4	5	L	-
	B1773	Sungold	4	4	3	2	3	3	3	3	M	*C*
	B1682	Lake Shasta	4	4	3	3	3	4	3	4	M	*C*

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydaimere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R	C	R	C	R	C	R	C		
B	B1265	SMALL WHITE 1C-92	4	4	3	3	3	4	4	4	L	-
	B1702	Oregon 2665	4	4	3	3	4	2	3	3	M	*C*
	B1710	Oregon 3785	4	4	3	3	4	4	3	3	M	*C*
	B1701	Oregon 2217-23	4	4	3	3	5	3	3	3	M	-
	B1712	Oregon 3800	4	4	3	3	5	4	3	4	M	*C*
	B1683	Oregon 1604-4	4	4	3	4	3	3	3	3	M	*C*
	B1711	Oregon 3792	4	4	3	4	3	3	3	3	M	*C*
	B1676	SMALL WHITE UI 74	4	4	4	3	3	3	4	4	L	*D*
	B1203	SMALL WHITE UI 40	4	4	4	3	3	5	4	4	L	*D*
	B1348	FLAGEOLET CHEVRIER	4	4	4	3	5	5	4	4	L	-
	B1714	Oregon 3953	4	4	4	4	4	4	3	4	M	*C*
	B1268	SMALL WHITE 1C-95	4	4	4	4	4	4	5	4	L	-
	B1706	Oregon 3597	4	4	4	4	4	5	3	4	M	-
	B1295	SMALL WHITE 1C-116	4	4	4	4	5	4	5	5	L	-
	B1288	SMALL WHITE 1C-109	4	4	4	4	5	5	4	4	L	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to pod-fill			
			R	C	R	C	R	C	R	C		
	B1290	SMALL WHITE 1C-112	4	4	4	4	5	5	5	5	L	-
	B1700	Oregon complex 1-1	4	4	4	5	3	2	3	4	M	C
	B1397	CLIPPER	4	4	4	5	5	3	4	4	L	-
	B722	SEAFARER	4	4	5	4	4	4	4	4	E	-
	B1275	SMALL WHITE 1C-103	4	4	5	4	4	4	5	4	L	-
	B1283	SMALL WHITE 1C-110	4	4	5	4	4	4	5	5	L	-
	B1287	SMALL WHITE 1C-108	4	4	5	4	5	4	5	4	L	-
	B1415	Purple King	4	5	3	4	5	5	5	5	VL	-
	B1294	SMALL WHITE 1C-115	4	5	4	4	5	4	4	5	L	-
	B574	SEAWAY	4	5	5	4	4	4	4	4	M	-
	B1291	SMALL WHITE 1C-113	4	5	5	4	5	5	5	5	L	-
	B574	SANILAC	4	5	5	5	4	3	4	4	M	-
	B1345	LITTLE NAVY	4	5	5	5	5	4	4	5	L	-
	B1396	HARKELL	5	4	2	5	4	3	4	4	L	-
	B1748	FVE	5	4	3	3	4	3	4	4	L	-
	B1747	FVX	5	4	3	3	4	5	4	5	L	-
	B1208	OTENASHI	5	4	4	3	4	4	4	5	L	-
	B1296	SMALL WHITE 1C-117	5	4	4	4	3	4	5	4	L	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to podfill			
			R	C	R	C	R	C	R	C		
B	B1435	CAPITAL	5	4	4	5	5	5	4	5	L	-
	B1276	SMALL WHITE 1C-104	5	4	5	3	5	5	4	4	L	-
	B1277	SMALL WHITE 1C-105	5	4	5	5	5	4	5	4	L	-
	B502	GREAT NORTHERN UI 59	5	4	5	5	5	4	V	5	L	-
	B716	Blue Lake	5	5	2	3	4	4	5	4	L	-
	B501	GREAT NORTHERN UI 31	5	5	4	5	5	3	V	5	L	-
	B1677	CHILEAN WHITE PEA	5	5	5	4	3	4	4	4	L	-
	B1393	KENTWOOD	5	5	5	4	4	3	4	4	L	-
	B1337	BANAT	5	5	5	4	4	5	5	5	VL	-
	B505	PINTO UI 114	5	5	5	5	4	4	5	5	L	-
	B500	RED MEXICAN UI 36	5	5	5	5	4	4	5	5	L	-
	B1338	SITAN BELI	5	5	5	5	4	4	V	5	VL	-
	B1003	CANNELLINO	5	5	5	5	5	V	V	5	VL	-
	B865	LIMELIGHT	5	5	5	5	5	5	V	4	E	-

Table 61. Summary of ranking orders of four parameters of rust development of 160 entries in summer-1974 trials at Castle Hill & Rydalmere together with maturity, hypothetical phenotype, type and origin (cont.)

Group	Accession		Ranking order								Maturity	Hypothetical phenotype *Ur-*
	Number	Name	Final rust rating		Period to infection		Rate of increase		Rate of increase relative to podfill			
			R	C	R	C	R	C	R	C		
C	B1264	SMALL WHITE 1C-91	2	3	2	2	2	3	2	2	L	*2*
	B750	*CALLAROY*	3	1	2	1	3	I	4	I	L	*1, 1 2*
	B1681	*Providor*	3	2	2	3	3	I	2	I	E	*C*
	B1436	CHIEF	3	3	2	1	3	4	4	3	L	*1 2*
	B1444	G71.1318A‡‡	3	3	2	3	2	I,2	3	2	M	*C, unknown*
	B1206	SMALL WHITE FM53	3	3	4	2	2	I,3	3	I 3	L	*1, 1 2*
	B1439	ARCHER	3	4	2	3	2	2	3	3	L	*1 2*
	B1675	BONUS	4	4	4	5	5	4	4	5	M	-

†R indicates Rydalmere.

†C indicates Castle Hill.

§E indicates early maturity; podfill 5.3 at Castle Hill.

M indicates mid season; podfill 12.3 & 15.3 at Castle Hill.

L indicates late maturity; podfill 19.3 & 23.3 at Castle Hill.

VL indicates very late maturity; podfill 26.3, 29.3 & 3.4 at Castle Hill.

Sown 15.1

¶ upper case indicates dry beans.

Italic type indicates selected in rust-liable area.

†† Standard type indicates selected in non rust-liable area

†† Lower case or this symbol, indicates fleshy podded beans

§§ See Table 53,23a.

¶¶ Replicates differed in reaction.

Numbers prefixed by B, in italics and bracketed, *e.g.* (*B 3088*) refer to germplasm releases from the U.S.D.A., Charleston, South Carolina, U.S.A.

The rust ratings varied between 1 - 10 for the 74 accessions susceptible to all races, for the 40 with *Ur-C* and the eight with *Ur-1 Ur-2*. Both cultivars carrying *Ur-D* were severely rusted.

Thus there were marked contrasts in field reaction which could not be explained by seedling reactions to prevalent races. In several instances cultivars susceptible in all greenhouse seedling tests showed slow and limited disease development while others were rapidly and severely affected. This resistance was effective at high inoculum levels since other cultivars and spreaders by comparison were very heavily rusted.

The relationship of seedling resistance to field reaction will be discussed in Section 7.2.1.1.9 (p. 160).

7.2.1.1.4. Resistance in agronomic and horticultural groups

Resistance expressed as slow rusting was common in fleshy-podded beans (presented in lower case type in Table 61), although it did not occur in most of the Oregon, and other bush Blue Lake types. The fleshy-podded beans with the greatest resistance included the Queensland cultivars Redlands Autumncrop and Ormiston, selected for slow rusting, as well as Redlands Greenleaf Strains B and C, and Redlands Pioneer, selected for seedling resistance. Slow rusting was present in the Borlotti, Red Kidney and White Kidney and in certain Small White and Cannellini dry beans (dry beans are listed in upper case type in Table 61). The finding for the first time of a moderate level of resistance in occasional Oregon bush Blue Lake beans, *viz.* Oregon 3603 and the possible identification of slow rusting Small White beans should be of value to breeders.

7.2.1.1.5. Influence of area of origin

As indicated in Table 61, most of the 34 beans selected in rust-liable areas (italics) and approximately 26 from other areas (standard type) showed little or no infection. In contrast, most of the moderately, severely or very severely rusted beans were selected in non rust-liable areas.

7.2.1.1.6. Variation

The variation between final rust ratings at each site and between sites is given in Table 62. There was more variation at Rydalmere than at Castle Hill and there was more variation between than within sites.

Table 62. Distribution of Group B entries according to differences in final rust rating between replicates in summer-1974 trials at Castle Hill & Rydalmere

Site	Number of accessions which differed by rust rating units of									
	0	1	2	3	4	5	6	7	8	Total
Castle Hill	71	45	15	5	1	0	0	1	0	139
Rydalmere	60	43	27	3	3	0	2	0	0	138
Both sites	13	36	24	13	5	2	3	1	1	138

The variation between the other disease parameters summarized in Table 63 indicates that there was closer agreement between replicates at Rydalmere than at Castle Hill and again that there was more variation between than within sites.

Uneven distribution of the races was undoubtedly responsible for the variation between replicates and sites for entries susceptible only to certain races, for example, Ouray and 643. However, some of the error may be attributed to differences in microclimate.

7.2.1.1.7. Production of telia

Telia formed in Small White Nos 38, 59, FM 51 and FM 52 late in the season, but were not seen on plots of Epicure and Golden Gate Wax adjacent to the trials. The Small White cultivars are susceptible only to the (a)dhi and adhi groups of races. These field observations are in agreement with greenhouse findings (Section 4.3.11., p. 33) that the (d)h and dh groups of races formed telia readily, but the g and ch groups virulent on Epicure and Golden Gate Wax respectively, did not.

Table 63. Distribution of accessions according to differences in three parameters of rust development in summer-1974 at Rydalmere and Castle Hill

Site	No. of entries in B(0), (i), (ii), C(0), (i) & (ii) groups which differed by the following numbers of days for rust to appear after inoculum was available								
	0	1-3	4-7	8-10	11-14	15-21	22-29		Totals
Rydalmere	77	17	43	1	0	0	0		138
Castle Hill	27	27	60	10	12	1	1		139
Both sites	6	10	67	20	19	104	2		138
	No. of B(ii) & C(ii) entries which differed in rate of increase from time of first appearance on each plot by the following number of days for plots of the same entry to develop 500 pustules								
	0	1-3	4-7	8-10	11-14	15-21	22-29	No of entries B(0) C(0) B(i) C(i)	Totals
Rydalmere	35	23	44	12	4	1		13 6	138
Castle Hill	15	21	46	7	9	2		27 12	139
Both sites	0	2	16	20	42	16	3	(27) (12)	138
	No. of B(ii) & C(ii) entries which differed in time to develop 500 pustules relative to pod fill by the following numbers of days								
	0	1-3	4-7	8-10	11-14	15-21	22-29	No of entries B(0) C(0) B(i) C(i)	Totals
Rydalmere	41	22	48	7		1		13 6	138
Castle Hill	21	30	32	4	11	0		27 12	139
Both sites	0	1	19	24	39	14	2	(27) (12)	138

7.2.1.1.8. Comparisons with overseas results

Seven of 11 beans reported to have "horizontal" resistance in Central America and South America had seedling resistance effective against all Australian races (Section 6.2.6., p. 126). These were Costa Rica 1031, Preto 897, Ricobaio 1014, Ricopardo 896, Vi 1013, PR 3 and PR 9. The other four each showing limited development of rust were Manteigao Preto 20, susceptible to all races in the seedling stage, PR 15 (*Ur-C*), PR 16 (*Ur-Epi Ur-Ver*) and Rico 23 (*Ur-F*). The remaining ten introductions from Central America and South America were either free of rust or showed slight infection.

Redlands Pioneer and PI 226 895 were among 10 of the most resistant beans in both years and at most locations at which the IBRN was assessed (Schwarz and Galvez 1977). These entries were both susceptible in greenhouse tests to races prevalent in this trial and developed little rust in the Castle Hill and Rydalmere trials. In the absence of more detailed data on the overseas trials, little comment can be made on the possibility of these lines possessing non-specific resistance. While Redlands Greenleaf B and Redlands Pioneer reacted identically in field and greenhouse tests with Australian cultures, Redlands Pioneer apparently showed superior resistance to Redlands Greenleaf B in some other areas. Thus it would appear to have an additional factor(s) not recognized by Australian races.

7.2.1.1.9. Discussion of relationship of seedling resistance to field reaction

In these trials the presence of certain seedling resistance genes appeared to delay the onset of the epidemic. For example, Ouray and Roman Cranberry, which both carry *Ur-Gol*, effective against all except the c group of races, did not show rust until late, but were soon moderately or severely affected. Similarly, some of the accessions with *Ur-1* and *Ur-2*, effective against all except the a(i) and ai groups of races, were infected late. Rust development was rapid on Great Northern US 1140 and slower on others such as 1C-93 (bred by Ferry Morse Seed Company) and 643. Great Northern US 1140 was one of the earliest-maturing beans, and rust increase may have been halted by senescence. However, the Small White

types 1C-93 and 643 were later-maturing, so there was considerable time for further infections to develop. Thus it is likely that 1C-93 and 643 have resistance.

There have been indications that seedling resistance genes whose effects have been overcome by a change to virulence in the pathogen may still operate to restrict disease development in epidemics with races possessing corresponding genes for virulence. For example, Slesinski and Ellingboe (1971) reported that the level of uptake from the host of radioactive sulphur for the host resistant : pathogen virulent combination was intermediate to that of the resistant : avirulent and either the susceptible : virulent or susceptible : avirulent interactions. Riley (1973) gave further examples. However, in this trial, two lines of evidence enabled rejection of the hypothesis that the genes *Ur-1*, *Ur-2*, *Ur-C*, *Ur-D* and *Ur-Red* restricted disease development when races with the corresponding virulence factors were present. These were:-

- The wide range of field reactions shown by entries carrying these genes (Appendix 4) and
- The homogeneous field reactions shown by certain cultivars which gave heterogeneous seedling reactions. These were Brown Beauty (-, *Ur-C*), Ormiston (*Ur-C*, *Ur-C Ur-D Ur-Red*) and Redlands Autumncrop (-, *Ur-C*). Thus these cultivars, plus Redlands Greenleaf Strains B and C and Redlands Pioneer have both forms of resistance and these appear to act independently.

7.2.1.1.10. Discussion of possible mechanisms of resistance expressed as slow disease development.

Accessions on which rust appeared early were generally rapidly and severely affected, whereas those on which the disease appeared late in the trial usually showed slow and limited rust development. Pustules on older leaves of beans which were severely affected were generally larger than on comparable leaves of slightly rusted beans. However, pustules were smaller on all cultivars where pustule number was so high that development was restricted.

The mechanisms suggested for resistance expressed as slow disease development

are differences in receptivities of host tissues (Groth and Urs 1977), incubation periods, rates and periods of spore production (Van der Zaag 1959, Zadoks 1972, Parlevliet and Van Ommeren 1975).

The large differences in times to first appearance of rust on the different cultivars and the smaller numbers of pustules on those infected last, support the contention of Groth and Urs (1977) that differences in receptivities occur. No experimental details are available at present.

In the greenhouse seedling inoculation tests, there were no differences between host cultivars in the time between inoculation and eruption of pustules. This suggested that there were no differences in incubation periods. However, Parlevliet (1975a) advocated a cautious approach to seedling data as incubation periods were much shorter in seedlings than in adult plants.

The presence of smaller pustules on older leaves of slow rusting entries than on comparable leaves of rapid rusting lines suggested that a type of mature leaf resistance may be operating.

7.2.1.2. Spring-1974

Seventy entries were rusted uniformly within each replicate (Group B), whereas two were mixed (Group C). The criteria for ranking of mean final rust ratings are presented in Table 64. The ranking of accessions is given in Table 65, together with data on hypothetical phenotype, agronomic or horticultural type and if selected in a rust-liable area. The analysis of variance of rust ratings outlined in Table 66 indicated significant differences between at least some lines. The formulae for the analyses of variance are given in Appendix 5.

Since a(d)h(i) was the only race detected, there was no possibility that low ratings resulted from low levels of inoculum of less frequent races or that interactions between races occurred.

Classification of entries according to hypothetical phenotypes and rust ratings is given in Appendix 6. Cultivars with seedling resistance to race a(d)h(i) and low ratings were Bonita *(Ur-L)*, Redlands Greenleaf Strains

Table 64. Criteria of ranking of final rust ratings and numbers of accessions in each class in spring-1974 trial at Castle Hill

Parameter	Ranking	Range	No. in each class
Final rust rating (0 - 10)	1	1	12
	2	1.25 - 2.75	15
	3	3 - 5	19
	4	5.5 - 8	18
	5	8.25 - 10	8
			Total 72

Table 65. Summary of ranking of final rust rating for 72 accessions in spring - 1974 together with hypothetical phenotype, type and origin

Group	Ranking of final rust rating	Accession		Hypothetical phenotype *Ur-*
		Number	Name	
B	1	B1232	*BONITA*[†‡]	*L*
		B753	*College Pride*[§]	*C*
		B713	*NB2-S2*[†]	*1 2*
		B1441	*Ormiston*	*C, C D Red*
		B752	*Redlands Autumncrop*	*-, C*
		B754	*Redlands Belle*	*C*
		B1557	*Redlands Greenleaf B*	*C D Red*
		B751	*Redlands Greenleaf C*	*2 C Red*
		B755	*Redlands Pioneer*	*C D Red*
		B545	Spartan Arrow[#]	*C*
		B767	Valgold	*C*
	2	B1210	CALIFORNIA WHITE KIDNEY	-
		B1391	*College Early*	*C*
		B1263	*Hawkesbury Wonder*	*C*
		B712	*NB1 - S1*[†]	*1 2*
		B714	*NB3 - S3*[†]	*1 2*
		B1424	*PR-15*[†]	*C*
		B1207	SMALL WHITE 59	*1 2*
		B1266	SMALL WHITE 1C-93	*2*
		B1272	SMALL WHITE 1C-100	-
		B1273	SMALL WHITE 1C-101	-
		B1289	SMALL WHITE 1C-111	-
		B1295	SMALL WHITE 1C-116	-
		B1254	*Tweed Wonder*	-
		B1257	*Windsor Longpod*	*C*
		B965	643 (SMALL WHITE)	*1 2 D*
	3	B617	Apollo	*C*
		B1528	*Brown Beauty*	*-, C*
		B940	Gallatin 50	*C*
		B1352	PI 226 895	-
		B1231	SMALL WHITE 38	*1 2 D*
		B1265	SMALL WHITE 1C-92	-

Key on p.166.

Table 65. Summary of ranking of final rust rating for 72 accessions in spring - 1974 together with hypothetical phenotype, type and origin (cont.)

Group	Ranking of final rust rating	Accession		Hypothetical phenotype *Ur-*
		Number	Name	
B	3	B1267	SMALL WHITE 1C-94	-
		B1271	SMALL WHITE 1C-99	-
		B1274	SMALL WHITE 1C-102	-
		B1276	SMALL WHITE 1C-104	-
		B1277	SMALL WHITE 1C-105	-
		B1285	SMALL WHITE 1C-106	-
		B1286	SMALL WHITE 1C-107	-
		B1287	SMALL WHITE 1C-108	-
		B1288	SMALL WHITE 1C-109	-
		B1283	SMALL WHITE 1C-110	-
		B1290	SMALL WHITE 1C-112	-
		B1293	SMALL WHITE 1C-114	-
	4	B1439	ARCHER	*1 2*
		B1435	CAPITAL	-
		B1436	CHIEF	*1 2*
		B1627	*GALLAROY*	*1*
		B1628	*GALLAROY*	*1 2*
		B1629	*GALLAROY*	*1 2*
		B1630	*GALLAROY*	*1*
		B1145	*KERMAN*	*1 2*
		B575	SANILAC	-
		B1204	SMALL WHITE FM 51	*1 2*
		B1205	SMALL WHITE FM 52	*1 2*
		B1206	SMALL WHITE FM 53	-
		B1676	SMALL WHITE UI 74	*D*
		B1268	SMALL WHITE 1C-95	-
		B1270	SMALL WHITE 1C-98	-
		B1275	SMALL WHITE 1C-103	-
		B1291	SMALL WHITE 1C-113	-
		B1294	SMALL WHITE 1C-115	-

Table 65. Summary of ranking of final rust rating for 72 accessions in spring - 1974 together with hypothetical phenotype, type and origin (cont.)

Group	Ranking of final rust rating	Accession		Hypothetical phenotype *Ur-*
		Number	Name	
B	5	B1675	BONUS	-
		B1188	*GREAT NORTHERN US 1140*	*1 2*
		B1679	*PINTO US 5*	*1 2*
		B1199	*PINTO US 14*	*-, 1 2*
		B1339	*SCOUT*	*1 2*
		B1203	SMALL WHITE UI 40	*D*
		B1264	SMALL WHITE 1C-91	*2*
		B1296	SMALL WHITE 1C-117	-
C		B1445	GL1§	*-, unknown*
		B1269	SMALL WHITE 1C-97	-

† Upper case or this symbol indicates dry beans.

ǂ Italics indicate selected in rust-liable area.

§ Lower case or this symbol indicates fleshy-podded beans.

Standard type indicates selected in non-rust-liable area.

Table 66. Analysis of variance of final rust ratings of 72 accessions in spring-1974 trial at Castle Hill.

Source of variation	d.f.	SS.	MS	F - Value
Block	3	14.9005	4.9668	10.38
Treatment	71	2113.5868	29.7688	62.26**
Error	213	101.8495	0.4782	
Totals	287	2230.3358		

B *(Ur-C Ur-D Ur-Red)* and C *(Ur-2 Ur-C Ur-Red)*, Ormiston (*Ur-C, Ur-C Ur-D Ur-Red*), and GL1 (a mixture of plants resistant to all races and others susceptible to all).

The presence of *Ur-1* and *Ur-2* did not restrict epidemic progress of race a(d)h(i) which is virulent for these genes. Evidence for this came from

- The wide range of field reactions of cultivars with these genes (Appendix 6).
- The homogeneous field reaction of Pinto US 14 which was heterogeneous for greenhouse seedling reaction, having one component with no seedling resistance and a second with *Ur-1* and *Ur-2*.
- The similar behaviour of Gallaroy Genotype I, B1627 and B1630, with *Ur-1* and Gallaroy Genotype II, B1628 and B1629, carrying *Ur-1* and *Ur-2*.

The low ratings in this trial of all cultivars with *Ur-C* in contrast to the wide range of ratings in the summer trials (Section 7.2.1.1.3. p.157) was due to the biased sample. The fleshy-podded cultivars tested were selected over a period of years for suitability in rust-liable areas of Eastern Australia. Certain cultivars, *e.g.* the College and Redlands series were selected in Australian breeding programmes whereas others such as Spartan Arrow were chosen in trials of introduced cultivars. Again, two cultivars, Brown Beauty and Redlands Autumn crop, with heterogeneous greenhouse reactions were homogeneous for field reaction. The presence of *Ur-C* in some plants among others susceptible to all races in these cultivars indicated that *Ur-C* did not restrict epidemic development in the presence of a race with the corresponding gene for virulence.

All the bush fleshy-podded beans grown commercially during the past two decades in coastal areas of Eastern Australia showed resistance. Certain Small White dry beans which showed resistance in the summer trials, *viz.* 643, 1C-93, plus the NB lines trialed for the first time also showed resistance. The three Pinto beans lacked resistance. The parentages, hypothetical phenotypes, field reactions and periods grown, for all cultivars used commercially in Eastern Australia are summarised in Appendix 7.

As in earlier trials, accessions showing early infection were often the most severely and the most rapidly rusted. Similarly, the entries with the longest delay in rust appearance were the least severely affected and were those on which rust development was slowest. However, this trend was less marked than in the summer trials.

There was much less variation between replicates in this spring trial than in the previous summer plantings in which a mixture of eight races was released (Tables 62 and 67). The accessions showing the largest total variation in all three trials were Great Northern US 1140 and many Small White beans. The variation in Great Northern US 1140, Small White numbers 38, FM 51, FM 52 and 1C-91 in the early trials may be attributable to the uneven distribution of race a(d)h(i), the only race virulent on them. However, this does not explain the wide variation between replicates of others, such as Small White 1C-101, with ratings of 2 in summer at Castle Hill, of 4 and 10 at Rydalmere and 1 and 3 in spring (Table 68). The two most obvious differences between the trials were the environment and the prevalence of the different races. Thus the adult plant resistance of the Small White lines susceptible to all races as seedlings may be either influenced by the environment or by the genotypes of the prevalent races. In contrast, the resistance of the fleshy-podded and certain dry beans such as PR 15 appeared to be more stable.

Table 67. Distribution of Group B entries according to differences in final rust rating between replicates in spring-1974 trial at Castle Hill

No. of entries which differed by rust rating units of					
0	1	2	3	4	Total
24	24	14	6	1	70

Table 68. Variation in final rust ratings of 72 accessions within and between trials during summer-1974 and spring-1974 trials

Relative behaviour	Entry	Range of final rust rating			
		all trials	Summer		Spring
			Castle Hill	Rydalmere	Castle Hill
Same in all trials	Bonita	1	1	1	1
	Redlands Autumncrop	1	1	1	1
	Redlands Greenleaf B	1	1	1	1
	Small White 59	1-2	1-2	1-2	1-2
Lower in summer than in spring	Small White 1C-93	1-3(2)	1	1	2-3
	Apollo	2-4(2)†	2	4	3-4
	Gallatin 50	2-4(2)	2	4	3-4
	Brown Beauty	2-4(2)	3-4	2-4	4
	PI 226 895	1-4(3)	1	2	2-4
	Small White UI 40	7-10(3)	7-8	8-10	8-9
	Small White 38	2-6(4)	3-6	2-3	4-6
	Chief	4-8(4)	4	4-5[10]	7-8
	Archer	5-9(4)	5-6(8)	5-7	7-9
	Small White 1C-98	6-10(4)	6	6-10	6-7
	Small White FM 52	3-8(5)	3-5	4-6	7-8
	Small White FM 53	3-8(5)	3-6	4-6	8
	Great Northern US 1140	5-10(5)	5	9-10	10
	Small White 1C-91	3-9(6)	3-6	3[8]†-4	8-9
	Small White FM 51	1-8(7)	4	1-5	4-8
Higher in summer than in spring	Ormiston	1-2(1)	1-2	1	1
	Redlands Greenleaf C	1-2(1)	1	2	1
	Redlands Pioneer	1-2(1)	1	2	1
	Valgold	1-2(1)	1-2	2	1
	Bonus	9-10(1)	7-10	9-10[1]	9[1]-9[6]
	Small White 1C-117	8-10(2)	8-9	9-10	10
	PR 15	1-3(2)	2-3	1-2	1-2
	Small White 643	1-3(2)	3	1-2	1-2
	California White Kidney	1-3(2)	2-3	2-3	1-2

Key on p. 171.

Table 68. Variation in final rust ratings of 72 accessions within and between trials during summer-1974 and spring-1974 trials

Relative behaviour	Entry	Range of final rust rating			
		all trials	Summer		Spring
			Castle Hill	Rydalmere	Castle Hill
Higher in summer than in spring	Small White 1C-115	7-9(2)	7-8	9	9
	Small White 1C-103	7-10(3)	8-9	8-10	7
	Small White UI-74	7-10(3)	7-10	9-10	7-9
	Small White 1C-113	7-10(3)	8-10	8-10	7
	Sanilac	7-10(3)	7	8-10	8-10
	Small White 1C-112	4-8(4)	4-6	7-8	7-9
	Capital	6-10(4)	7	10	6-7
	Small White 1C-95	6-10(4)	6-7	6-7	8-10
	Small White 1C-111	2-7(5)	4	6-7	2
	Small White 1C-106	3-8(5)	6-8	6-7	3-4
	Small White 1C-114	3-8(5)	3-4	7	7-8
	Small White 1C-110	4-9(5)	3-5	6-7	8-10
	Small White 1C-109	4-9(5)	4-5	7	8-10
	Small White 1C-116	2-8(6)	2-4	7-8	7-9
	Small White 1C-105	4-10(6)	4-5	7-8	10
	Small White 1C-104	4-10(6)	4-6	8	10
	Small White 1C-102	1-8(7)	2-4	7-8	1-4
	Small White 1C-94	2-9(7)	4	8-10	2-4
	Small White 1C-97	3-10(7)	5-6	6-10	3-6[7]
	Small White 1C-108	3-10(7)	3	6-8	8-10
	Small White 1C-92	2-10(8)	5-6	7-10	2-3
	Small White 1C-99	2-10(8)	2-5	6-8	8-10
	Small White 1C-100	2-10(8)	2-4	7	9-10
	Small White 1C-107	2-10(8)	2-5	7-9	8-10
	Small White 1C-101	1-10(9)	2	4-10	1-3
Only in spring trial	College Pride				1
	NB2 - S2				1
	Redlands Belle				1
	Spartan Arrow				1
	Tweed Wonder				1

Table 68. Variation in final rust ratings of 72 accessions within and between trials during summer-1974 and spring-1974 trials

Relative behaviour	Entry	Range of final rust rating			
		all trials	Summer		Spring
			Castle Hill	Rydalmere	Castle Hill
Only in spring trial	Windsor Longpod				2
	Kerman				7
	Pinto US 14				10
	GL1				1[0]
	NB3 - S3				1-2(1)
	Gallaroy B1628				5-6(1)
	Gallaroy B1630				5-6(1)
	Scout				8-9(1)
	Pinto US 5				9-10(1)
	NB1 - S1				1-3(2)
	Gallaroy B1627				5-7(2)
	Gallaroy B1629				5-7(2)
	College Early				1-4(3)
	Hawkesbury Wonder				1-4(3)

† round brackets ( ) indicate the maximum range

† square brackets [ ] indicate variation in disease rating within a plot.

7.2.2. FIELD TRIALS WITH INBRED POPULATIONS

7.2.2.1. General observations

In 1976 rust was well distributed through the trials three weeks after sowing. Frequent light rain-showers early in the trial favoured disease development, but prolonged heavy rain at later stages did not. On some lines severe infection prevented pod formation.

In 1977 rust showed slow initial development, because of prolonged periods of hot and wet weather. The disease was widely distributed through the trials two months after sowing, when most plants were in full pod.

Some plants in low areas died during wet weather, particularly in 1977. Where only one replicate of a line died, a missing plot value was calculated according to the method given by Steel and Torrie (1960). Where more than one replicate was missing, results of that line were omitted from analysis.

The lines showing the greatest degrees of resistance did so even when growing next to severely rusted beans (Plates 3 and 4). In these trials early infection was not a good indicator of severe final rust rating.

7.2.2.2. Races present

Race adhi, the one released, was predominant in 1976, but low levels of other races were found, *viz.* (a)dhi in all samples, egh in one sample and both a(d)h(i) and efgj in another. Only race adhi was detected in 1977. Thus all lines except the reference cultivar, Bonita, were susceptible as seedlings to the prevalent race.

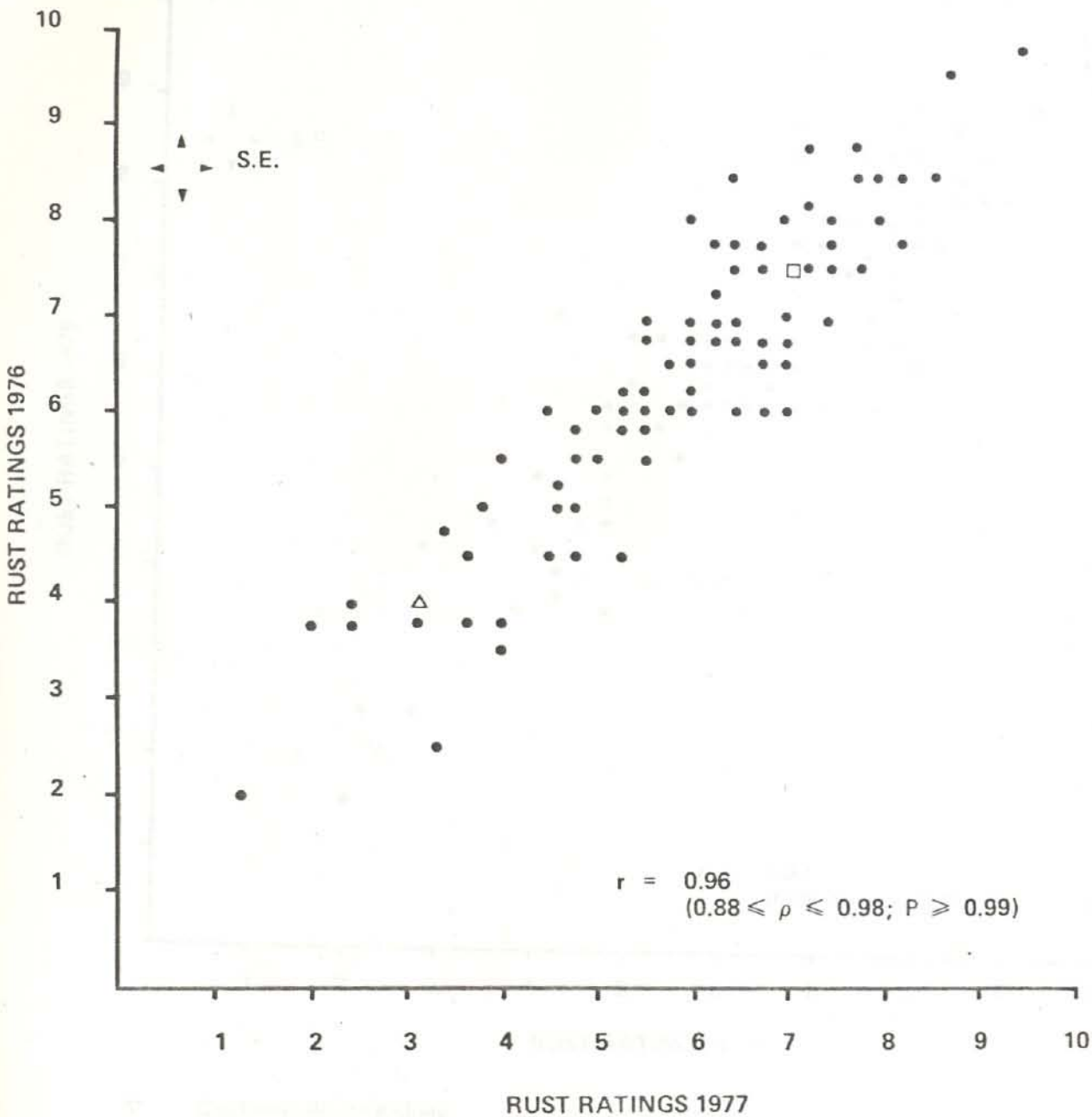
7.2.2.3. Rust ratings

The mean ratings of the parents and some of the reference cultivars differed from one experiment to another (Table 69), but were slightly higher for most lines in 1976 than in 1977 (overall mean 1976 = 5.25, 1977 = 4.82). This difference occurred despite the much earlier development of rust in 1976, suggesting that the resistance is, to a considerable extent, independent of growth stage. The unadjusted mean ratings for the two seasons are compared in scatter diagrams presented in Figures 5, 6 and 7. The high correlations between results in the two seasons suggest that the resistance is relatively stable during late summer

Table 69. Rust rating means and ranges of 10 reference genotypes in a series of trials between 1974 and 1977 at Rydalmere and Castle Hill

	Summer-1974				Spring-1974		Summer-1976						Summer-1977					
	Rydalmere		Castle Hill		Castle Hill		A		B		C		A		B		C	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Apollo	4		4		3.25	3-4	4.5	4-5	3	2-4	4.5	4-5	3.75	3-4	2.75	2-4	3	
California White Kidney	2.5	2-3	2.5	2-3	1.5	1-2	3	2-4	1.5	1-2	2		2.5	2-3	2.5	2-3	2	
Sanilac	9	8-10	7		7	8-10	8		6.75	6-7	7.75	7-8	7.75	7-8	7.7	7-8	6	
Redlands Autumncrop	1		1		1		1.75	1-2	1.75	1-2	3	2-4	1.25	1-2		1	1	
Redlands Greenleaf B	1		1		1		1	1	1.25	1-2	1		1			1	1	
Redlands Greenleaf C	2		1		1		1.25	1-2	1		1		1			1	1	
NB3-S3	-		-		1.5	1-2	3.5	3-4	3.75	3-4	4.25	4-5	2		2.25	2-3	2.75	2-4
Small White 1C-93	1		1		2.75	2-3	4	3-5	4	3-5	4	4	2.5	2-3	3	2-4	2.75	2-3
Bonita	1		1		1		1		1		1	1	1		1		1	
Great Northern US1140	9.5	9-10	5		10		10		10		10		10		10		10	

Figure 5. Scatter diagram of mean final rust ratings in 1976 and 1977 of 106 inbred lines derived from Sanilac/Apollo



△ Apollo

□ Sanilac

S.E. Standard error (weighted means of trials A, B & C)

In the cross Apollo/California White Kidney, most lines showed less disease and a few showed more rust than either parent. Ten of these lines, chosen on the basis of reactions in 1976 and representing a range of resistant and susceptible material, were significantly different from the parents in a small experiment conducted in 1977. As indicated in Table 72, seven lines showed less rust, three more disease than either parent and one reacted similarly to California White Kidney. Thus transgressive segregation produced some lines with better resistance and others with poorer resistance than the parents. These results also indicate that Apollo and California White Kidney do not share all genes for slow rusting, although they may share a factor(s) which slightly retards the epidemic as the highest rating was similar to that of Sanilac.

7.2.2.4. Relationship between rust rating and presence of *Ur-C*

As indicated in Table 73, in Sanilac/Apollo, the rust rating was independent of *Ur-C*, carried by Apollo. However, in Apollo/California White Kidney in 1977 there were fewer lines than expected with *Ur-C* among those with the best resistance. There was no similar deficiency in 1976 when fewer lines showed such a high level of resistance. The deficiency in 1977 may be attributed to a relatively loose linkage in repulsion of *Ur-C* and one of the genes involved in slow rusting in California White Kidney.

Table 72. Mean rust ratings of five replicates of plots of parents and 11 single seed descent lines of Apollo/California White Kidney, and analysis of variance

Entry	Mean rust rating
Parents	
California White Kidney	2.2
Apollo	3.4
Sanilac	7.2
Progeny of Apollo/California White Kidney	
162	1
173	1
217	1
225	1
240	1
242	1
250	1
239	2.2
166	5
197	5
160	5.6

Analysis of variance

Source of variation	d.f.	SS	MS	F-value
Block	4	1.3751	0.3438	2.0
Treatment	13	301.0482	23.1576	133.4**
Error	52	9.0249	0.1736	
Totals	69	312.8182	23.6750	

L.S.D. 5% = 0.53
1% = 0.70

Table 73. Relationship between mean final rust ratings during 1976 and 1977 and genes *Ur-C* and *ur-C* in single seed descent populations

Cross	Year	Mean final rust rating class	Genotype		Interaction χ^2
			Ur-C —	ur-C ur-C	
Sanilac/Apollo	1976	1 - 4.75	4	10	
			39	52	
					0.67
	1977	1 - 4.75	9	15	
		5 - 9.75	36	47	
					0.13
Apollo/California White Kidney	1976	1 - 1.75	7	30	
		2 - 6.75	30	26	
					3.57
	1977	1 - 1.75	7	35	
		2 - 5.75	36	21	
					7.3**

8. DISCUSSION

8.1. Survey of populations of *U.appendiculatus*

Race surveys on a well-chosen set of host testers are a prerequisite to successful breeding programmes aimed at resistance to bean rust. When continued over long periods, race surveys provide data on the extent of variation and the evolution of new races, together with indications of the value of individual seedling resistance genes in breeding. This information assists in determining breeding strategies. Moreover, surveys provide a collection of cultures which permit identification of seedling resistance genes in the host and may be used in breeding.

(i) Extent of variation and evolution of new races

As only 299 samples were examined in this four-year survey and about one-third of these were from commercial crops, only limited conclusions may be reached about the means by which new races arise. As each race generally differed from at least one other in pathogenicity on single differentials, the accumulation of virulence genes by the pathogen appeared to occur mainly by single-step changes. The sequence of development of the races of the bean rust fungus in Eastern Australia indicates that single-gene resistances have been short-lived. However, two findings suggest that combinations of certain genes for seedling resistance would be a suitable breeding strategy in at least some groups of beans. These are the gradual single-step accumulation of virulences which usually occurred and the detection of host genes which are effective against all Australian races as well as occurring at different loci.

The cultivars grown have a considerable influence on the detection, survival and hence the frequency of new rust races (Luig and Watson 1970). Extensive experience with the cereal rusts has shown that as more and more genes conferring seedling resistance are transferred to cultivars, the fungi show a remarkable ability to increase the number of genes for virulence in a stepwise manner. A similar accumulation of virulence genes has occurred in *U. appendiculatus* in

Eastern Australia. The resistance (possibly due to *Ur-C*, see Sections 4.3.6. p. 29 and 5.3.1.1.2. p. 50) of the "Wonder" cultivars which were grown in the early 1940's was overcome by a new race in 1948. Similarly, race dh, virulent on Redlands Greenleaf A (*Ur-C Ur-D Ur-Red*), released in 1960, was recorded in 1964. Race adhi was found in 1970, even before the release of Redlands Greenleaf C (*Ur-2 Ur-C Ur-Red*), selected for resistance to race dh. Furthermore, this race was virulent on Gallaroy (*Ur-1*, *Ur-1 Ur-2*) and Kerman (*Ur-1 Ur-2*). Another race, (a)dhi, virulent on Redlands Greenleaf C but not on Gallaroy and Kerman, was recognized a short time later when this study began. However, this may have been present, undetected, with race adhi which was found at the end of Johnson's survey. It would appear that these cultivars exerted selection pressure and favoured races which had accumulated genes for virulence. The separation of the three genes in the original source of resistance, *viz. Ur-1*, *Ur-2* and *Ur-D*, during breeding undoubtedly facilitated the accumulation of virulences. The sequence of development of the dh group of races is also not clear as the (d)h group avirulent for *Ur-Red* present in Redlands Greenleaf Strains A, B and C as well as Redlands Pioneer was reported in this survey but not in Johnson's.

The frequencies of isolates virulent for genes present in commercial cultivars, *viz.* 95 per cent for *Ur-C*, 66 per cent for *Ur-D*, 61 per cent for *Ur-2*, 54 per cent for *Ur-Red* and 30 per cent for *Ur-1*, were generally higher than those for genes not present in such cultivars. A finding of particular note was the frequency of five per cent of races avirulent for *Ur-C*, even though this gene is present in all fleshy-podded beans grown commercially. Furthermore, as indicated in Section 4.3.8. (p. 30), this may be an underestimate as simple races may not be readily detected when present at a low level in collections with more than one race. Similarly, the five to six per cent frequency of races virulent for *Ur-F*, *Ur-Gol* and *Ur-Ver* was noteworthy as these genes have not been exposed in commercial plantings.

While most of the races within groups differed form one another in pathogenicity

on single differentials, a number of races showed greater differences. An additional differential, B2053 (*Ur-J*), coded j for naming of races, was detected during the investigation. Thus there are greater differences between the prevalent and some of the rare races than indicated in Section 4.3.5. (p. 27). Thus race efgj (rare) differs from race gh (prevalent) in reaction on four differentials, from egh (intermediate) on three and from both fg (intermediate) and eg (rare) on two differentials. If analysis of El Salvador 184 or PR 22 confirms the presence of *Ur-PR 22*, proposed hypothetically in Section 6.2.2. (p.122), then races fg and efgj differ from other races to an even greater extent than their present designation indicates.

Mutation, considered to be a common cause of variation in other rust fungi (Watson 1970a, Green 1975), may account for differences in pathogenicity on single differentials. For example, variants virulent on differential e (*Ur-F*) were detected in greenhouse tests from apparently pure cultures of similar races avirulent on e. According to this hypothesis, there is a relatively high rate of mutation to virulence on e. A similar situation may exist with differentials c (*Ur-Col*) and f (*Ur-Ver*), although variants virulent on these were not detected in greenhouse tests with pure cultures.

Similarly, a high mutation rate from virulence to avirulence on h (*Ur-C*) may be proposed. However, three of the five races avirulent on this differential differ from all others or from the more prevalent races in reaction on two or more differentials, indicating an additional cause(s) of variation.

Four facts suggest that sexual recombination has not contributed to the variation:-

- Telia were found only for races in the (d)h and dh groups, yet the maximum variation was recorded in the g group.
- Aecia of the bean rust fungus have not been recorded in Eastern Australia.
- The uncommon races usually occurred in the same samples as more prevalent races with similar virulence formulae.

- The virulence formulae of most races in each group were similar.

Thus some asexual process may be involved. Hyphal anastomosis followed by gene or nuclear rearrangement may be responsible for changes in pathogenicity on differentials e, f, h and j. Experiments with mixtures of races may provide insight into such a possibility. The location and use of variants with paler urediniospores than the wild type would permit precise experiments to be carried out.

The rare races and some of those of intermediate abundance mostly had one virulence factor more than their more prevalent counterparts, but five had one virulence factor less than the abundant races. In all the latter instances, the races were avirulent on differential h (*Ur-C*). Thus it would appear that one of the mechanisms of variation commonly gives rise to races avirulent for *Ur-C*. This mechanism may operate more frequently than these results suggest, since the widespread presence of *Ur-C* in most plantings sampled may not favour survival of such avirulent races.

In other host : pathogen systems, virulence factors have usually proved to be recessive. If this applies to virulence for *Ur-C*, the derivation of avirulent from virulent races by a process other than mutation cannot be explained on a simple model.

An inhibitor system as outlined in the flax rust interaction by Lawrence *et al.* (1976) may account for such an observation. In this system a dominant inhibitor gene I interacts with the dominant gene normally conditioning avirulence in the host, *e.g. P-Ur-C* to give a virulent pathogen phenotype. According to this model, races of genotype *ii P-Ur-C* ——— are avirulent for *Ur-C*, whereas rust races of genotype *I-* ——————— and *ii p-Ur-C p-Ur-C* are virulent.

This hypothesis may be tested by selfing and crossing a range of cultures. No difficulty has been reported in germinating teliospores, although a dormancy period was found to be necessary (Andrus 1931, Harter *et al.* 1935). The failure to find telia of some races such as the ch and g groups may be a problem.

(ii) The value of certain genes in breeding

Genes of greatest value to breeders are those effective against most or all known races. Even among the 299 samples tested in this survey, there were differences in the frequencies of isolates virulent on single genes not exposed in commercial cultivars. While five to six per cent of races isolated were virulent on each of differentials c (*Ur-Gol*), e (*Ur-F*) and f (*Ur-Ver*), no race was virulent on Bonita (*Ur-L*), Cornell 49-242 (*Ur-H*) or PR 5 (*Ur-G*). Genes such as *Ur-F* in differential e may still be of value in breeding programmes, if used in combination with other genes for which there are low frequencies of virulence.

New set of testers

The frequencies of races virulent for genes occurring only in combination could not be accurately determined on the set of testers used in this survey, as six of the stocks carried more than one gene and five genes were each present in more than one tester. A new set of testers comprising single gene stocks derived from accessions with genes in combination as well as accessions shown to have single genes is proposed. This will give better resolution of races as well as more precise data on frequencies of races virulent for particular genes. Three groups of accessions are necessary:-

Firstly, an Australian Universal Suscept, *viz.* Sanilac as used in this study.

Secondly, ten differentials, mostly single gene stocks derived in this study (Table 44):-

- B1627 (*Ur-1*), B2090 (*Ur-2*), B2113 (*Ur-D Ur-Red*); these would replace the present a, d and i differentials.
- Differentials c (*Ur-Gol*), e (*Ur-F*), f (*Ur-Ver*), g (*Ur-Epi*) and h (*Ur-C*) as currently used.
- Two accessions not routinely used, B2053 (*Ur-J*) and Small White UI 40 (hypothetical phenotype *Ur-D*) which would supplement B2113 (*Ur-D Ur-Red*).
- El Salvador 184 or PR 22 which may carry *Ur-PR 22*; not currently used.

Thirdly, twelve stocks resistant to all Australian races. Seven of these known to carry single genes are Bonita (*Ur-L*), Cornell 49-242 (*Ur-H*), PR 5 (*Ur-G*), B2055 (*Ur-2*^{2}), B2091 (*Ur-I*), B2052 (*Ur-K*) and B2056 (*Ur-N*). Aurora (*Ur-3 Ur-N*) should be included to supplement B2056 (*Ur-N*) as *Ur-3* could not be separated from *Ur-N* with the cultures available. The other four stocks have not been subjected to genetic analysis but are resistant to rust in many locations, *viz*. Compuesto Chimaltenango numbers 2 and 3, Ecuador 299, Mexico 309 and others listed in Section 6.2.6. (p. 126).

Other accessions may be added if they are nominated as differentials of international status. A set of candidate differentials has been distributed to bean rust workers throughout the world from the stocks used in this study (Ballantyne, unpublished report 1976). The results of greenhouse testing of these accessions with a range of races from other geographic areas should permit selection of a set of suitable testers.

With the introduction of a new set of testers, it is desirable that the present system of race nomenclature be replaced by a system acceptable to other bean rust workers as discussed in Ballantyne (1974a and 1976).

The races identified on the set of differentials used in this study permit the identification of ten genes for rust resistance in the host. Five of these (*Ur-1*, *Ur-2*, *Ur-C*, *Ur-F* and *Ur-J*), were isolated in genetic analyses. Two others (*Ur-D* and *Ur-Red*), were not separated in the analysis of Redlands Greenleaf B, presumably because of close linkage. Three others (*Ur-Epi*, *Ur-Gol* and *Ur-Ver*) were proposed hypothetically in differentials g, e and f, respectively. Analysis of El Salvador 184 or PR 22 would indicate if another proposed gene, *Ur-PR 22*, can be detected by Australian rust populations. This analysis may also indicate the genetic basis of resistance in differentials f or g or both, as these accessions were susceptible only to the fg group of races.

8.2. Seedling resistance genes

Data on both the identities and locations of the genes for seedling resistance

are needed in breeding programmes using such resistance. Because of the limited time available and the need to gain a broad perspective of host resistance, only some of the genes isolated were located. However, the data available indicate several combinations of genes which may be used by breeders.

Four genes at the three loci clearly established were permanently designated *Ur-1*, *Ur-2*, *Ur-2*^{2} and *Ur-3*. The other 11 (*Ur-C*, *Ur-D*, *Ur-F*, *Ur-G*, *Ur-H*, *Ur-I*, *Ur-J*, *Ur-K*, *Ur-L*, *Ur-N* and *Ur-Red*), retain their temporary designations. The existence of *Ur-N* and *Ur-Red* has not been established beyond doubt. There is evidence for at least two additional loci.

Seven genes (*Ur-1*, *Ur-2*, *Ur-C*, *Ur-D*, *Ur-F*, *Ur-J* and *Ur-Red*) were identified with local cultures, but the remaining eight (*Ur-2*^{2}, *Ur-3*, *Ur-N*, *Ur-G*, *Ur-H*, *Ur-I*, *Ur-K* and *Ur-L*), were effective against all Australian races. Of this latter group, *Ur-L*, and at least in some tests, *Ur-K* may be distinguished from other genes by a characteristic white or purple area around the pustules on the lower leaf surfaces. Genes *Ur-I* from NEP 2 and *Ur-3* from Aurora which showed identical ITs may be either at the same locus or identical. Genes *Ur-G*, *Ur-H* and *Ur-J* may be either linked or at the same locus, but there is no evidence that they are identical.

In other host : pathogen systems, some genes which condition a low or intermediate IT in greenhouse seedling tests do not confer protection under field conditions (McIntosh and Luig 1973). One such gene, *Ur-L*, was detected in this study.

8.3. Surveys of accessions of *P. vulgaris*

As may be predicted from studies in other host : pathogen systems, resistance was most common in beans introduced from areas where rust is destructive. This finding applied both to seedling resistance and slow rusting, although relatively few of the Central American and South American beans trialed in the field were susceptible to Australian races. One notable exception to this trend was the presence of *Ur-C* in the majority of fleshy-podded beans, most of which were

selected in areas where rust rarely occurs. Other exceptions were from programmes where beans from Central America and South America were used as sources of particular features such as disease resistance. In the past decade, bean breeders have used more diverse germplasm, especially from Central America and South America. As a result, rust resistance may be more common in cultivar and germplasm releases in future, even where selection for rust resistance had not been a specific objective.

8.4. Influence of seedling resistance genes on slow rusting

There have been suggestions that seedling resistance genes whose effects have been overcome by a change of virulence in the pathogen may still operate in restricting disease development in the field in the presence of races with the corresponding genes for virulence. However, no evidence for such a dual role was found in four situations in this study:-

- Cultivars homogeneous for *Ur-1*, *Ur-2*, *Ur-C*, *Ur-D*, *Ur-Gol*, *Ur-Red*, singly or in combination.
- Cultivars heterogeneous for greenhouse reactions but homogeneous for field reactions with races virulent on them. Genes involved were *Ur-1*, *Ur-2*, *Ur-C*, and *Ur-Red*.
- Hybrids (F6) derived from Sanilac/Apollo (*Ur-C*) and Apollo/California White Kidney.
- Hybrids (F3) derived from Sanilac/Bonita (*Ur-L*).

8.5. Stability of slow rusting

Resistance expressed as slow disease development has commonly been assumed to be effective in all locations and to all races. In Eastern Australia, field experience is that the green fleshy-podded beans grown have a useful degree of resistance which has apparently been stable. While no major deterioration is known, there are no precise experimental or historical data on this resistance.

In all trials in this investigation, including some in which a race virulent for all component genes was present, Redlands Greenleaf C remained almost free

of rust. However, its resistance in winter plantings in the Gympie district of South-East Queensland during some seasons has been inadequate (P. Farlow and G. Johnson, personal communication). Beans are cropped continually at Gympie, so ample inoculum is available. The plants grow slowly during winter so are exposed to infection for long periods. The difference between the reactions in the two areas may be the result of different genotypes of the pathogen, some clones of which may overcome at least some of the resistance. Alternatively, it may be the result of environmental conditions which either favour the pathogen or reduce the resistance of the host. Field experiments over a period of years with a range of accessions in the Gympie and Sydney districts may give useful data. It is desirable that a more precise rating system such as those outlined by Chiarappa (1971) for other crops be developed and used for such work.

8.6. Inheritance of slow rusting

The results of the study of inheritance of slow rusting in Apollo and California White Kidney are generally in agreement with most other studies of slow disease development systems. Resistance did not appear to be controlled by a single gene in either cultivar, although the relatively high incidence of transgressive segregation in the crosses between the slow rusting cultivars suggests a relatively simple basis of resistance. The high correlations between resistance in the two seasons, and the identical or similar ratings in replicates of most lines indicate that the resistance in these cultivars is relatively stable and is suitable for use in a breeding programme. The field reactions of Apollo and California White Kidney in other geographic areas are not known, but the inbred populations together with the parents provide a base of materials for study in any geographic area.

8.7. Application of these results in breeding programmes

8.7.1. IN AUSTRALIA

Three approaches (p. 2) may be used to apply the results reported in this thesis. Multilines will not be considered as this study was not designed to

investigate such a strategy. A breeding strategy suitable for fleshy-podded beans may differ from that relevant to at least some types of dry beans. This is because of the distribution of the two forms of resistance within the agronomic and horticultural types of beans as well as the exacting commercial requirements of the fleshy-podded types.

(i) Combination of genes for seedling resistance

As indicated in Section 8.2 (p. 191) the combination of genes for seedling resistance is a suitable strategy for certain types of dry beans, provided that the component genes are not exposed singly. It is desirable that at least one gene be effective against all Australian races and against most races in other geographic areas, *i.e.* has a low frequency of races with virulence. As the recently characterized and postulated genes for virulence, *e.g.* virulence on j (*Ur-J*) and PR 22 which may possess *Ur-PR 22*, have occurred in races avirulent for *Ur-C*, the use of *Ur-C* in combination with others may assist in improving the stability of rust resistance.

Three methods of selecting plants with combinations of genes are:-

(a) Select for rust resistance on an empirical basis in the field, from crosses involving rust resistant beans with a minimum of two genes, preferably either segregating independently, loosely linked in repulsion or linked in coupling. Test the most promising selections with a range of races to detect mixtures and to ensure that at least some plants are resistant to all known local races. Carry out conventional genetic analyses on single typical plants resistant to all races, to determine if one or more genes are present.

In this study, selections of Actopan/Sanilac bred by the Queensland Department of Primary Industries were subjected to such testing. The results indicated that elimination of plants carrying only *Ur-F* from Actolac (phenotypes *Ur-F*, *Ur-2*^{2} *Ur-F*) and the production of a stock of Actosan increased from B2093 shown to possess three genes (phenotype *Ur-2*^{2} *Ur-F* plus *Ur-J* or *Ur-Ver*) should prolong the resistance of these cultivars. It should be noted that cultivars

selected only on the basis of field reactions may be heterogeneous in such a way as to favour the progressive accumulation of virulences. The mixtures in Actolac and Gallaroy illustrate this.

(b) Use rare cultures with particular combinations of virulence to ensure that only plants with combined resistances are selected. Some cultures from local populations may be suitable, or mutant cultures may be obtained. For example, a strategy for developing a rust resistant pinto bean based on the use of certain rare races such as (a)cdhi to incorporate a gene effective against all Australian races into B1553 (*Ur-2 Ur-Gol*) was outlined in Section 6.2.10.2. (p. 138).

(c) Use a combination of genes which results in a lower IT than the IT produced by the individual component genes. Such an interaction occurred in the cross Bonita (*Ur-L*)/B2052 (*Ur-K*), but the precise genotypes of seedlings with the lower IT was not known. While the simplest hypothesis is that *Ur-K* was interacting with *Ur-L*, an alternative hypothesis is that *Ur-K* was interacting with the factor(s) governing slow rusting in Bonita.

(ii) Improvement of slow rusting

In the bush fleshy-podded beans, slow rusting was widespread whereas seedling resistance effective against all Australian races was rare. For this reason, selection for slow rusting appears to be a more promising strategy than use of seedling resistance, at least in the short term. The quality requirements of fleshy-podded beans are very exacting and other disease resistances need to be included. Thus transfer of more than one gene for seedling resistance to beans with suitable horticultural features would be a major undertaking. Crosses between beans with the highest levels of resistance could be expected to give some very resistant progeny. However, as resistance has been recovered from crosses involving at least one susceptible parent in some other systems (Gallegly and Neiderhauser 1959, Pope 1968) routine field screening for rust resistance of hybrid progeny from a range of crosses may be a valuable breeding procedure.

In the field plots slow rusting was obvious in certain lines even where

adjacent plots were very severely rusted. Hence selections may be made from simply designed breeding nurseries using high disease incidence. This contrasts with some other systems where more specialized designs and controlled disease levels have been advocated to avoid underestimation of the resistance (Parlevliet and Van Ommeren 1975, Van der Plank 1963).

When selecting for slow rusting it is essential that the effect of any seedling resistance genes be removed to ensure that the transfer of genes for slow rusting has been achieved. For example, if Wis HBR 72, found to develop rust slowly, is used as a parent, a race virulent for *Ur-Epi* should be used in field selection plots. This accession carries *Ur-Epi*, so in the absence of races virulent on this gene, selection may be made for seedling resistance, conditioned by *Ur-Epi*. Similarly, if Redlands Greenleaf B, or Redlands Pioneer, shown to develop rust very slowly are used as parents, the dh group of races should be used since it is virulent for all genes conferring seedling resistance in these cultivars.

The identification for the first time of slow rusting in Small White beans in this study should enable breeders to broaden the base of resistance. The unreleased breeding lines with such resistance, *viz.* 1C-93 from the Ferry Morse Seed Company, the NB group from a New South Wales Department of Agriculture breeding programme and the 227.25 group from Sanilac/Bonita are either of commercial standard or closely approach it.

(iii) Combinations of both forms of resistance

The use of sib-selection as outlined by MacIndoe (1949) would permit the combination of both forms of resistance. Within the Small White group, the lines outlined above as slow rusting and the germplasm of the Small White type with genes *Ur-3*, *Ur-G* and *Ur-H* singly, as generated in this study.

8.7.2. IN OTHER GEOGRAPHIC AREAS

The studies reported here and the strategies outlined are directly applicable only in relation to the Australian cultures used. Quite different results may be obtained using isolates from other geographic areas. For example, the gene

detected in PR 5 during these studies may not be identical with the one detected in F2 plantings in Colombia (Anon. 1976a). It is possible that two different genes are involved, *viz.* *Ur-G* in Australia and *Ur-X* in Colombia. The Australian races may thus be genotype *P-Ur-G* ——— *p-Ur-X p-Ur-X* and Colombian races *p-Ur-G p-Ur-G P-Ur-X* ———. Testing of a range of lines of known reaction to Australian cultures with a range of Colombian cultures would indicate if the same gene was being detected with all cultures.

The New Zealand study of inheritance in Westralia may be applicable to the Australian situation. The populations of the pathogen in New Zealand were similar or identical to those occurring in Australia in the 1960's when the single gene was detected in the F2 and F3 Westralia progeny.

The relationship of the gene R_{B11} (Augustin *et al.* 1972) to genes isolated in this study is not known. This gene was reported as the sole determinant of resistance in Great Northern US 1140 on the basis of F2 tests. The critical experiment would be to test Great Northern US 1140 (hypothetical phenotype *Ur-1 Ur-2*), Sanilac and single gene stocks B1627 (*Ur-1*) and B2090 (*Ur-2*) with the culture of Brasilian race B11 used by Augustin *et al.* (1972)

The host stocks analysed in this study may not be identical with those accessioned elsewhere under the same name. The differences in reaction of a number of such stocks received from different collections in the germplasm survey and the heterogeneity of many accessions illustrate this.

However, the results of this study can be applied in other areas. The data on ITs, locations and other features (*e.g.* temperature sensitivity) of the genes available in single gene stocks provide basic reference points. The hypothetical phenotypes assigned to several hundred accessions are a further adjunct in work on seedling resistance. The identification of slow rusting accessions in trials with high levels of disease and known races may provide a basis for comparison in other areas.

Similarly, the accessions and hybrids described here as slow rusting may

have this resistance only in relation to the Australian cultures used in this study. Some groups of cultivars, for example, pole Blue Lake fleshy-podded beans, Great Northern, Pinto, Red Mexican and some Small White dry beans are severely affected in many geographic areas. However, others, for example many bush green beans and the Red Kidney and White Kidney dry beans, are less severely rusted in a wide range of environments (Ballantyne 1974b). Nevertheless, further detailed information is needed to determine if the slow rusting is non-specific.

Overseas breeders selecting for rust resistance may be guided by:-

(i) Results of race surveys from a number of geographic areas and International Virulence Gene Surveys. These indicate some of the most valuable host genes, *i.e.* those for which the corresponding gene for virulence in the pathogen is rare. In addition, in continuing and comprehensive surveys, the combinations of virulences in the pathogen may be predicted. If particular combinations do not occur, *e.g.* race fgh, which was predicted but not detected in this survey, the corresponding host resistance genes may give stable resistance. While there have been suggestions (Van der Plank 1975, Wolfe *et al.* 1976) that such an approach may be useful, there are no reports of its successful application.

(ii) Results of germplasm screening with a range of races, as carried out in this investigation. Such results permit allocation of hypothetical phenotypes which have a value equivalent to data from testing F2 populations in genetic analyses.

(iii) Results of IBRNs. However, without data on greenhouse reactions to the races present, only limited conclusions can be made. For example, an accession which develops only a slight amount of rust in such a trial may have

- Seedling resistance expressed as low or intermediate IT to all races present,
- Seedling resistance to the prevalent races. However, in other trials with different races, or different incidences of races, such an accession may be severely rusted, or
- Resistance expressed as slow rusting, *i.e.* susceptible in the seedling stage

the prevalent race(s). Identification of accessions which are slow rusting in relation to rust populations in different geographic areas and in a range of environments would be valuable.

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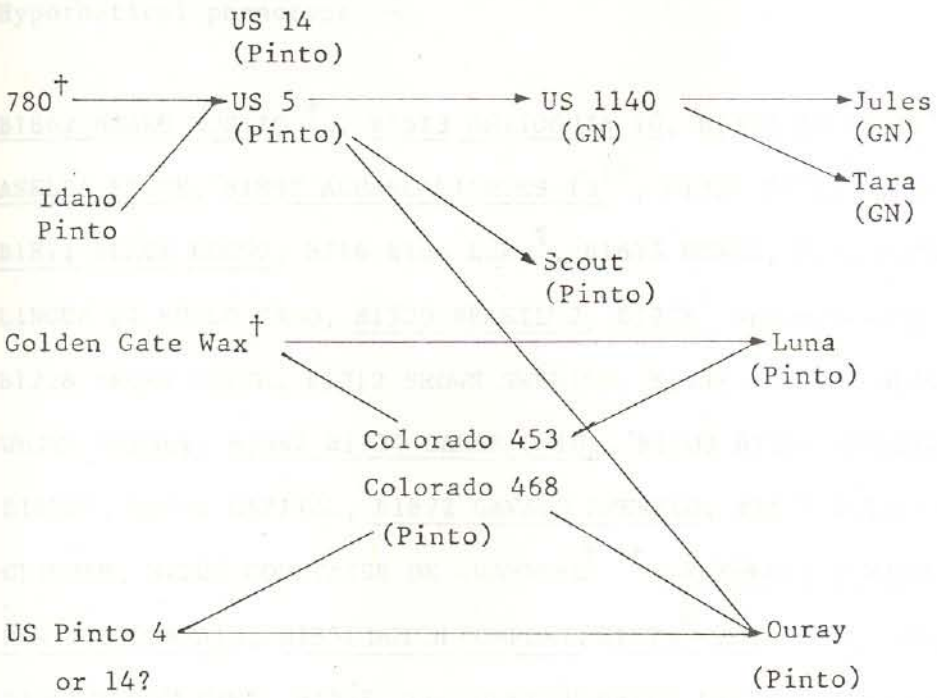
Appendix 1 Dry beans selected for rust resistance in El Salvador and Puerto Rico by Dr. N. Vakili, USDA, Mayazuez, Puerto Rico

Abbreviation	Complete designation
PR 1	(1) PR-70-15R-42
PR 2	(2) PR-70-15R-52
PR 3†	(3) PR-70-15R-55
PR 4	(4) PR-70-15R-57
PR 5	(5) PR-70-15R-87
PR 6	(6) PR-70-15R-148
PR 7	(7) PR-70-15R-167
PR 8	(8) PR-70-15R-180
PR 9†	(9) PR-70-15R-189
PR 10	(10) PR-70-15R-193
PR 11	(11) PR-70-15R-194
PR 12†	(12) PR-70-15R-227
PR 13	(13) PR-70-15R-287
PR 14	(14) PR-70-15R-292
PR 15†	(15) PR-70-15R-210
PR 16†	(16) PR-70-15R-66
PR 17	(17) PR-HYB-71-1R-63
PR 18	(18) PR-HYB-71-1R-69
PR 19	(19) PR-HYB-71-1R-101
PR 20	(20) PR-HYB-71-1R-103
PR 21	(21) PR-HYB-71-1R-113
PR 22	(22) PR-HYB-71-1R-136

† possible slow ruster.

Appendix 2. A simplified pedigree of certain Great Northern (GN) and Pinto beans, showing apparent sources of rust resistance



† Rust differentials in North American set.

References.
Coyne and Schuster (1969, 1970); Minges (1972); Quinones (1963); Wood (1971 and personal communication); Zaumeyer *et al.* (1960) and Zaumeyer *et al.* (1946)

Appendix 3. Numbers and names of accessions in hypothetical phenotype groups outlined in Table 53 (cont.)

GROUP 26

I (cont.)

c Genotype *Ur-F Ur-J Ur-I Ur-K*

Type accession B1666 NEP 2

d Genotype *Ur-G*

Type accession B1667 PR 5[†]

e Genotype *Ur-H*

Type accession B1672 CORNELL 49-242

II Hypothetical phenotypes assigned on the basis of tests on Actopan/Sanilac Selection 37 tests above and F2 populations

a Hypothetical phenotype *Ur-2*^{2} *Ur-F*

Type accession B2092 ACTOLAC

B982[#] ACTOLAC

b Hypothetical phenotype *Ur-2*^{2} *Ur-F + Ur-J or Ver*

B2093 ACTOSAN

III Unknown genotype

B1844 AGUASCALIENTES 13[††], B1477 ANTIOQUIA 8, B1514 ANTIOQUIA 12, B1516 ATLANTICO 1, B1517 ATLANTICO 6, B1680 B1819 BLACK TURTLE SOUP, B1659 BLACK TURTLE SOUP TA31, B1521 BRASIL 4, B1623 CARAOTAS, B1881 CHIAPAS 36-2, B1750 COMPUESTO CHIMALTENANGO 2, B1754 COMPUESTO CHIMALTENANGO 3, B1478[#] COMPUESTO COTAXTLA, B1479 COMPUESTO NEGRO CHIMALTENANGO, B1785 CUILAPA 72, B1400 B1786 CUVA 168-N, B2025(P771) CUARENTO, B1658 ECUADOR 66, B1724 ECUADOR 299, B1893[#] FRIJOL NEGRO CAHABON, B1854 GLOSSY WONJI, B1444[#] G71.1318A[¶], B1895 GUATAMALA 2226-B-21-N, B1822 GUERRERO 6, B1855 HIRNA BEAUTY, B1788 ICA TUI, B1846[#] JALISCO 33, B1779 JAMAPA[††], B1600 LA VEGA, B1756 LINEA 34, B1757 LINEA 37[††], B1532 MEXICO 11, B1533 MEXICO 53, B1534 MEXICO 112, B1537 MEXICO 120, B1538 MEXICO 122, B1663 MEXICO 142,

Appendix 3. Numbers and names of accessions in hypothetical phenotype groups outlined in Table 53 (cont.)

GROUP 26

III (cont.)

B1432 MEXICO 142-N, B1669 MEXICO 227, B1806 MEXICO 235, B1539 MEXICO 254, B1640 MEXICO 309, B1543 MEXICO 492, B1758 MISS KELLY, B1399 MULATINHO††, B1544 NARINO 11, B1790 NEGRO JALPATAGUA, B1791 NEGRO SAN RAMON, B1886 N283-50689, B1830 PANAMITO CORRIENTE, B1778 PINTO SERRANO, B2083 PORILLO NO.1, B1405 B1792 PRETO 897, B2020(P498) B2024(P758) PUEBLA 152, B1869 PURPLE ALEMAYA, B1858 PURPLE AWASA, B1859 PURPLE BEDELLE, B1487 PI 152 326†, B1281 PI 165 426 (WHITE-SEEDED), B1282 PI 165 426 (BLACK-SEEDED), B1298 PI 165 435†, B1491 PI 194 587, B1883 PI 196 299†, B1493# PI 196 467, B1495# PI 207 409, B1727# B1801# PI 307824†, B1901 PI 311 930, B1365 PI 313 524†, B1803 PI 319 649, B1410 PR 1†, B1411 PR 2†, B1412 PR 3†, B1413 PR 4†, B1415 PR 6†, B1416 PR 7†, B1418 PR 9†, B1419 PR 10†, B1420 PR 11†, B1422 PR 13†, B1428 PR 19†, B2047 *P. vulgaris/P.coccineus*, B1408 RICOBAIO 1014, B1406 B1794 RICOPARDO 896, B1781 SAN PEDRO PINULA, B1480 S-19-N†, B1483 S-219-N†, B1593 S-18 (VENEZUELA 39), B1885 S434-AR(R239)†, B1828# SB-30-iI-pM-PI†, B1782 TURRIALBA 1, B1897 TURRIALBA 2, B1484 TURRIALBA 4, B1571 UBERABINHA, B2014(P539) VENEZUELA 2, B1773 VENEZUELA 54, B1783 VILLA GUERRERO, B759 Westralia, B1407 B1796 VI 1013, B1725 B2081# 814¶, B1831 142-mI-pM-pI†, B1832 173-mI-pM-pI†, B1882 51051 (I-1138)†.

GROUP 27

All plants resistant to all races, but heterogeneous in tests with single races

B1049 COSTA RICA 1031, B1755 GUATAMALA 416, B1481 JAMAPA††, B2020(P640) MICHOACAN 46, B2008(P436) NEGRO 324, B1496 PI 208 769, B1740 PI 310 814, B1739 B1802 PI 310 878, B2012(P512) S-166-A-N†, B1549 VENEZUELA 63.

Appendix 3. Numbers and names of accessions in hypothetical phenotype groups outlined in Table 53 (cont.)

GROUP 28

Heterogeneous accessions; resistant and susceptible plants present in no discernible pattern

B1639 B1768 CANARIO DIVEX 8120, B1525 CAUCA 31, B1524 CAUCA 47, B1530 HUILA 21, B1531 MAGDALENA 3, B1535 MEXICO 114, B1562 MULATINHO††, B1488 PI 155 212†, B1606 PI 163 583†, B1490 PI 189 227†, B1492 PI 195 371†, B1941 PI 206 980†, B1548 VENEZUELA 45.

GROUP 29

Infection type varied from intermediate to high, depending on environment and not on race

B1729 Dade, B2026(P776) TORTOLAS, B1940 PI 177 499, B1902 PI 313 654.

Key

† Upper case of this symbol indicates dry beans.

ǂ Lines underscored were selected in rust-liable areas.

§ Lines not underscored were not known to be selected in a rust-liable area.

¶ Lower case of this symbol indicates fleshy-podded beans.

Some plants only; heterogeneous accession.

†† Another accession with this name reacted differently.

ǂǂ Numbers prefixed by P are accession numbers given in Anon. 1976a.

§§ Numbers prefixed by B and bracketed, *e.g.* (B 3088) refer to germplasm releases from the United States Department of Agriculture Vegetable Breeding Laboratory, Charleston, South Carolina, U.S.A.

Appendix 4. Final rust ratings in summer-1974 trials of 170 accessions classified according to hypothetical phenotype for rust reaction

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
-	B	Redlands Autumncrop†	1	1
		PI 226895	1	2
		Royal Red	2	2
		White Kidney	2-3	2
		Saluggia B1226	2-3	2
		Borlotto	2-3	2
		California White Kidney	2-3	2-3
		Saluggia B1147	2-4	2
		Brown Beauty†	2-4	3-4
		Manteigao Preto 20	-	3-4
		Nr 66	4	3
		Goudkorrell	4-5	3
		Berna	4-5	3-4
		Borlotto Fuoco di Lingua nano	3-5	3-5
		Brown Swedish	4-5	4
		Cannellino Bianco	4	4-5
		Taisho Kintoki	4-5	4
		Small White 1C-101	4-10	2
		Brown Dutch	5-6	4
		Small White 1C-102	7-8	2-4
		Small White 1C-111	6-7	4
		Oregon 3044	5-6	4-6
		Cannellino B1209	5	6
		Small White 1C-98	6-10	4
		Nori A 25	7-8	3-6
		Nori B 30	7	5-6
		Beka	6-7	5-7
		Small White 1C-94	8-10	4
		Small White 1C-97	6-10	5-6
		Small White 1C-106	6-7	6-8
		Oregon 3596	6-7	6-8
		Small White 1C-100	7-10	5-6
		Flageolet Roi Des Verts	9-10	5

Key on p.227.

Appendix 4. Final rust ratings in summer-1974 trials of 160 accessions classified according to hypothetical phenotype for rust reaction (cont.)

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
-	B	Small White 1C-114	7-8	7
		Oregon 3597	8	7
		Clipper	8	7-8
		Small White 1C-95	8-10	6-7
		Small White 1C-110	8-10	6-7
		Small White 1C-112	7-9	7-8
		Small White 1C-116	7-9	7-8
		Oregon 2217-23	8-10	6-7
		Flageolet Chevrier	9-10	6
		Small White 1C-99	8-10	6-8
		Small White 1C-108	8-10	6-8
		Small White 1C-109	8-10	7
		Corvette	8-10	7-8
		Small White 1C-92	9-10	7
		Small White 1C-103	8-10	8-9
		Small White 1C-105	10	7-8
		Little Navy	8	9
		Capital	10	7
		Small White 1C-107	8-10	7-9
		FVE	10	7-8
		Sanilac	8-10	8-10
		Seaway	8-10	8-10
		Seafarer	9-10	8-9
		Small White 1C-104	10	8
		Small White 1C-115	9	9
		FVX	10	8-9
		Harkell	10	8-9
		Otenashi	10	8-9
		Purple King	9-10	9
		Small White 1C-113	9-10	9
		Blue Lake	10	9
		Great Northern UI 31	10	9
		Great Northern UI 59	10	8
		Pinto UI 114	10	9

Appendix 4. Final rust ratings in summer-1974 trials of 160 accessions classified according to hypothetical phenotype for rust reaction (cont.)

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
-	B	Red Mexican UI 36	10	8-10
		Kentwood	10	9-10
		Small White 1C-117	10	9-10
		Chilean White Pea	10	10
		Banat	10	10
		Cannellino B1003	10	10
		Limelight	10	10
		Sitan Beli	10	10
	C	Bonus	9-10(1)[‡]	9(1)-9(6)
C	B	Redlands Autumncrop[†]	1	1
		Ormiston[†]	1	1-2
		Borinquena	1-3	1-2
		Valgold	1-2	1
		Charlottetown	2-3	1-2
		PR 15	2-3	1-2
		Sunbeam	2	2
		Goldcrop	2-3	2
		Resistant Kinghorn Wax	2-4	1-3
		Canyon	3-4	2
		Apollo	4	2
		Gallatin 50	4	2
		B 3088	2-4	2-4
		Brown Beauty	2-4	3-4
		Tendercrop	3-4	2-4
		Bush Blue Lake 274	4-6	2
		Goldcoast	4	3
		Coram	3-6	3
		G4	5-7	5-6
		Oregon 3887	5-6	5-6
		B 3787	5-7	5-6
		Oregon 3603	6-8	5-6
		Oregon 3664	7-8	5

Appendix 4. Final rust ratings in summer-1974 trials of 160 accessions classified according to hypothetical phenotype for rust reaction (cont.)

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
C	B	Oregon 4022	7-9	2-6
		Wis HBR 40	7	5-6
		Oregon 58.2	8	5
		Oregon 2832-B	8	5
		Oregon 3634	8	5-6
		GCW199	6-8	7
		Oregon 3785	8-9	5-7
		Sungold	8	6-7
		Lake Shasta	8	7
		Oregon 1604-4	8	7
		Oregon 3800	8	7
3	B	Oregon 3948	7-8	7
		Oregon 2665	8	7-8
		Oregon Complex 1-1	9	7
		Oregon 3953	7-10	7-8
		Oregon 3792	9-10	7
		Bush Blue Lake 290	7	10
	C	Provider	4-5	1-2(5)
		G1318A†	2-8(0)	1(0)-8(0)
1	C	Gallaroy†	7(2)	1
		Small White FM53†	4-6	3-6(1)
1 2	B	Great Northern US 1140	9-10	5
		Small White 59	1-2	1-2
		Small White 643	1-2	3
		Small White FM51	4	1-5
		Small White FM52	4-6	3-5
		Small White 38	2-3	3-6
	C	Gallaroy	7(2)	1
		Chief	4(10)	4(10)-5
		Archer	5-7	6-8(4)

Appendix 4. Final rust ratings in summer-1974 trials of 160 accessions classified according to hypothetical phenotype for rust reaction (cont.)

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
1 2	C	Small White FM53†	4-6	3-6(1)
2	B	Small White 1C-93	1	1
	C	Small White 1C-91	3(8)-4	3-6
D	B	Small White UI 40	8-10	7-8
		Small White UI 74	9	9
2 C Red	B	Redlands Greenleaf C	2	1
C D Red	B	Redlands Greenleaf B	1	1
		Ormiston†	1	1-2
		Redlands Pioneer	1-2	1
Gol	B	Roman Cranberry	6-7	3-4
		Ouray	7-8	5-6
Epi	B	Wis HBR 72	2	2
Epi Ver	A B	PR 16	1	0-1
F	A	Rico 23	0	0
		Actolac†	0	0
L	B	Bonita	1	1
Epi +	A B	PI 203 958	1	0
2^2 *F*	A	Actolac†	0	0
		Actopan/Sanilac Selection 37	0	0
2^2 *F + J or Ver*	A	Actosan	0	0

Appendix 4. Final rust ratings in summer-1974 trials of 160 accessions classified according to hypothetical phenotype for rust reaction (cont.)

Hypothetical phenotype *Ur-*	Group	Accession	Final rust ratings	
			Rydalmere	Castle Hill
3 N	A	Aurora	0	0
H	A	Cornell 49-242	0	0
F I J K	A	NEP 2	0	0
unknown	A	Black Turtle Soup	0	0
		Costa Rica 1031	0	0
		Cuva 168-N	0	0
		Mulatinho	0	0
		PI 165 426 (white-seeded)	0	0
		PI 313 524	0	0
		Porillo No. 1	0	0
		PR 3	0	0
		PR 9	0	0
		Preto 897	0	0
		Ricobaio 1014	0	0
		Ricopardo 896	0	0
		Vi 1013	0	0
		Westralia	0	0
		G1318A†	2-8(0)	1(0)-8(0)

† some plants only.

‡ the figure in brackets is the rating of a few plants which reacted differently to the majority in the plot.

Appendix 5. Formulae for the analysis of variance in a randomized complete block design

Source of variation	d.f.	Sums of squares (SS)	
		Definition	Working
Blocks	r-1	$t\Sigma_j(x.j - x..)^2$	$= \frac{\sum_j X^2.j}{t} - C$
Treatments	t-1	$r\sum_i(X_{i.} - x..)^2$	$= \frac{\sum_i X^2 i.}{r} - C$
Error	(r-1)(t-1)	$\sum_{ij}(X_{ij} - x.j - x_i + x..)^2$	(total-block-trt) SS
Total	rt-1	$\sum_{ij}(X_{ij} - x_{\circ}..)^2$	$= \sum_{ij} X^2_{ij} - C$

r = no. of blocks t = no. of treatments

X_{ij} is the observation from the jth block on the ith treatment, i = 1,..., t treatments and j = 1,..., r blocks.

x.. is grand mean

C is correction term = $\frac{X^2..}{rt}$

Mean square (MS) = $\frac{\text{Sums of squares}}{\text{d.f.}}$

F value = $\frac{\text{Treatment MS}}{\text{Error MS}}$ for Treatments

or

$= \frac{\text{Blocks MS}}{\text{Error MS}}$ for blocks

Appendix 6. Relationship between hypothetical phenotype and rust ratings of 72 entries in spring-1974 trial at Castle Hill

Hypothetical phenotype	Group	Entry	Mean final rust rating
-	B	Redlands Autumncrop†	1
		California White Kidney	1.5
		Tweed Wonder, Small White 1C-111	2
		Small White 1C-100, Small White 1C-101, Small White 1C-116	2.5
		Small White 1C-94, Small White 1C-108	3
		Small White 1C-106, Small White 1C-114	3.25
		Small White 1C-92, PI 226 895	3.5
		Small White 1C-99, Small White 1C-102	3.75
		Brown Beauty†, Small White 1C-110	4
		Small White 1C-107	4.5
		Small White 1C-105, Small White 1C-109	4.75
		Small White 1C-104, Small White 1C-112	5
		Small White 1C-98	6.5
		Capital, Small White 1C-95	6.75
		Sanilac, Small White 1C-103, Small White 1C-113	7
		Small White 1C-115	7.25
		Bonus	8.25
		Small White 1C-117	8.5
		Pinto US 14†	10
	C	GL1†	1
		Small White 1C-97	4.5

Key on p. 231.

Appendix 6. Relationship between hypothetical phenotype and rust ratings of 72 entries in spring-1974 trial at Castle Hill.

Hypothetical phenotype	Group	Entry	Mean final rust rating
Ur-C	B	College Pride	1
		Ormiston†	
		Redlands Autumncrop	
		Redlands Belle	
		Spartan Arrow	
		Valgold	
		PR 15	1.25
		Windsor Longpod	2
		College Early	2.5
		Hawkesbury Wonder	2.75
		Apollo	3.25
		Gallatin 50	3.5
		Brown Beauty†	4
Ur-1	B	Gallaroy B1627	5.75
		Gallaroy B1629	
		Small White FM53†	8
Ur-2	B	Small White 1C-93	2.75
		Small White 1C-91	8.5
Ur-1 Ur-2	B	NB2-S2	1
		NB3-S3	1.5
		Small White 59	
		643	1.75
		NB1-S1	2
		Small White 38	4.75
		Gallaroy B1630	5.5
		Gallaroy B1628	5.75
		Small White FM51	6.25
		Kerman	7
		Small White FM52	7.5
		Archer	8
		Chief	
		Scout	
		Small White FM53†	
		Pinto US 5	9.75

Appendix 6. Relationship between hypothetical phenotype and rust ratings of 72 entries in spring-1974 trial at Castle Hill

Hypothetical phenotype	Group	Entry	Mean final rust rating
Ur-1 Ur-2	C	Pinto US 14†	10
		Great Northern US 1140	

† Heterogeneous accession.

Appendix 7. Cultivars grown commercially in Eastern Australia: parentage, hypothetical phenotype, field reaction and period grown

Type/Cultivar	Parentage	Period grown	Hypothetical phenotype *Ur-*	Field reaction	References
FLESHY PODDED					
FRESH MARKET					
Brown Beauty (BB)		1940-1968	-, *C*	Slight-moderate†ǂ	Anon. 1939
Tweed Wonder (TW)	Selected from Canadian Wonder	1936-1968	-	Slight†	Anon. 1939
Hawkesbury Wonder (HW)	TW//Keeney's Stringless Green Refugee	1937-1968	*C*	Slight†	Anon. 1939
Wellington Wonder (WW) Syn. Peerless?		1937-1968	NT§	NT	Anon. 1950
Windsor Longpod	TW//Keeney's Stringless Green Refugee/WW	1946-1971	*C*	Slightǂ	Anon. 1948
Redlands Greenleaf A (RGA)	BB/643/BB17A	1960-1964	*C D Red*	NT	Ogle & Johnson, 1974

Key on p. 236

Appendix 7. Cultivars grown commercially in Eastern Australia: parentage, hypothetical phenotype, field reaction and periods grown (cont.)

Type/Cultivar	Parentage	Period grown	Hypothetical phenotype *Ur-*	Field reaction	References
FLESHY PODDED					
FRESH MARKET					
College Pride	HW/Granda	1960-1964	*C*	Slight†	Anon. 1960
College Supreme	HW/Granda	1960-1963	NT	NT	
College Early	HW/TW	1960-1963	*C*	Slight†	Anon. 1961
Redlands Belle (RB)	Langshaw Beauty/ Florida Belle	1964-1969	*C*	Very slight†	Groszmann 1963.
Redlands Greenleaf B	BB/643//BB17A	1965-1971	*C D Red*	Very slight†‡	Ogle & Johnson, 1974
Redlands Pioneer (RP)	RGA/Plentiful	1966-present	*C D Red*	Very slight†‡	Anon. 1967
Redlands Greenleaf C	RGB/Redlands 120-2B (sister line to RGB)	1972-present	*2 C Red*	Very slight†‡	Ogle & Johnson 1974
Redlands Autumncrop (RA)	Langshaw Beauty Florida Belle//BB/ 643	1965-present	- , *C*	Very slight†‡	Anon. 1967

Appendix 7. Cultivars grown commercially in Eastern Australia: parentage, hypothetical phenotype, field reaction and period grown (cont.)

Type/Cultivar	Parentage	Period Grown	Hypothetical phenotype *Ur-*	Field reaction	References
FLESHY PODDED					
FRESH MARKET					
Ormiston	Wx[¶](*P. vulgaris*/ *P. coccineus*)//RGA/ Plentiful/4/Diacol Nima/Corneli 14/3 Corneli 14// RGA/ Plentiful	not released	*C, C D Red*		Anon. 1973
Spartan Arrow	Tenderbest/Contender	1967-1970	*C*	Very slight[ǂ]	
PROCESSING					
Tendercrop (TC)	Topcrop (US 5 Refugee/Full Measure)// Tenderpod	1962-1965	*C*	Slight-moderate[†ǂ]	Minges 1972
Gallatin 50	Not disclosed	1963-1976	*C*	Slight-moderate[†ǂ]	Zaumeyer 1963
Apollo	white-seeded TC[*]4// TF3047/P-8-478	1969-present	*C*	Slight-moderate[†ǂ]	Minges 1972
Canyon	White Seeded TC[*]6// Curly top resistant line	1970/71-present	*C*	Slight-moderate[†ǂ]	Dean personal communication

Appendix 7. Cultivars grown commercially in Eastern Australia: parentage, hypothetical phenotype, field reaction and period grown (cont.)

Type/Cultivar	Parentage	Period grown	Hypothetical phenotype *Ur-*	Field reaction	References
FLESHY PODDED					
PROCESSING					
Cascade	TC/154-50	1969 present	*C*		Barnes 1970
Valgold	Not disclosed	1969-present	*C*	Very slight†‡	Barnes 1970
DRY					
California Small White (CSW)		1936-1968	*1 2 D*	Slight†‡	Vinning 1976
Sanilac (San)	Michelite X-Ray Mutant/Emerson 847// Emerson 53	Not known		Severe†‡	Minges 1972
Gallaroy	San/CSW	1966-present	*1, 1 2*	Moderate-severe‡	Groszmann & Gallagher 1966
Kerman	San/CSW	1968-present	*1 2*	Moderate-severe‡	Gallagher 1968
Seafarer	Michelite X-Ray mutant/Emerson 847/Trag/Florida Belle	1971-present		Severe†‡	Barnes 1970

Appendix 7. Cultivars grown commercially in Eastern Australia: parentage, hypothetical phenotype, field reaction and period grown (cont.)

Type/Cultivar	Parentage	Period grown	Hypothetical phenotype *Ur-*	Field reaction	References
DRY					
Borlotto		1970-present	-	Slight-moderate†	
Cannellino		1970-present	-	Very severe†	
Great Northern UI 31	Great Northern UI 59 /Common Red Mexican	1973-present	-	Very severe†	Minges 1972
Pinto UI 111	Red Mexican UI 34/ Common Pinto	1973-present	-	Very severe†	Minges 1972
Red Mexican UI 36	Great Northern UI 1 /Common Red Mexican	1973-present	-	Very severe†	Minges 1972

Key

† Rating in summer-1974 trials (Section 7.2.1.1.1., p.143).

† Rating in spring-1974 trial (Section 7.2.1.2. p. 162).

§ Not tested.

¶ Wx may be identical with B2047 (p. 137).

DEVELOPMENT OF A SET OF INTERNATIONAL DIFFERENTIAL VARIETIES AND A STANDARD NOMENCLATURE OF RACES

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The bean rust fungus, Uromyces appendiculatus (Pers.) Unger resembles many other rust fungi in having numerous physiologic races differing in virulence on a range of host genotypes (Table I).

History of Bean Rust Differentials and Race Surveys

The differentials which have been used in distinguishing races are listed in Table II. The race surveys carried out with these have generally not been done in conjunction with comprehensive rust resistance breeding programmes. The results have been presented with the numerical ratings in a tabular form, often without a clear indication of resistance or susceptibility on all differentials.

The present situation with bean rust differentials is unsatisfactory because:

1) The six differentials first used by Harter & Zaumeyer (1941) are inadequate. One of them, 181, is of doubtful value in distinguishing races. Additional differentials used by others have not always been generally available.

2) Evidence of differing genotypes in accessions with the same name. Ogle and Johnson (1974) have suggested that the 780 used by Waterhouse (1954) in his Australian survey differed from the one Mr Johnson obtained from Dr Zaumeyer and used in his later Australian survey. Dr Meiners recently advised that the 643 used by Mr Johnson and subsequently by me gave a susceptible reaction alongside a resistant reaction of his 643 in greenhouse inoculation tests. After Dr Zaumeyer's retirement, Dr Meiners became custodian of his differentials.

As differentials have been held in separate collections for many years it is possible that outcrossing and technical errors have lead to genotypic variation which could disrupt present comparisons of data obtained in different countries. We should therefore renew our stocks from a common source, or check our stocks against others derived from a common stock with a range of cultures in order to confirm that the same genotypes are being used in our separate studies.

3) The climbing habit, late maturity and extreme susceptibility to some rust races of several Harter & Zaumeyer differentials cause difficulty in maintaining seed stocks.

Race survey results will be more easily understood if the previously used system of presenting them is replaced by a method of expressing avirulence/virulence formulae, particularly if not all workers decide to adopt the same differentials.

General Principles of Host Differentials

Flor's gene-for-gene concept (Flor, 1955; 1971) that for each gene conditioning rust reaction* in the host there is a specific gene conditioning pathogenicity* in the parasite, provides the genetic basis for

* - see footnote page 2

an understanding of physiologic races differing in pathogenicity. Differentials are the host genotypes used in race differentiation. Results are generally expressed as avirulence*/virulence* formulae, coded as a number or letter designation.

Avirulence/virulence formulae may be given as a series of sequential numbers denoting differentials or named genes. The sequential numbers or the genes on which the culture is avirulent appear before a slash mark / and the numbers or genes on which the culture is avirulent, after the /.

Such formulae for the reported races of the bean rust pathogen are listed in Appendix I.

A continuing history of these formulae for the races present in an area and an understanding of the ways in which changes occur are essential for breeding programmes designed for rust resistance, particularly the race-specific form.

Variability Studies

One classical approach is that it is essential to have three groups of genotypes:

1) An internationally accepted group to provide a common language between breeders throughout the world.

2) A local group which is only valuable in specific regions to give information to the breeders. If genotypes in this group become commonly used they should be moved to group 1.

3) A group of unusually resistant genotypes whose resistance may be due to: (a) single genes effective against a wide range of races
(b) multiple genes

At present there is only limited information available on the specific genes controlling resistance to bean rust (Zaumeyer & Harter, 1941). However if particular genotypes in group 3 are shown to have a single gene they should be transferred to group 1 as they will be excellent differentials of the future.

Single gene differentials are theoretically desirable but are often impracticable. Single gene stocks show the highest resolving power whereas multiple gene hosts have low resolving power and fail to detect many races.

Changes in Avirulence/Virulence Formulae

The appearance of new virulence genes and new combinations of virulence genes are the most important changes in avirulence/virulence formulae.

Some of the ways in which these changes may occur are:

1) By sexual reproduction. The bean rust fungus is autoecious, forming all its spore stages (0, I, II, III & IV) on bean. There is little definite information on the role of the sexual stage in its propogation. Some races produce telia readily whereas others never form them (Harter et al., 1935). Australian experience with the cereal rusts is very similar. In

* Footnote: The following terms are used as defined by Loegering & Powers (1962): Bean rust results from the interaction of a host (Phaseolus vulgaris L.) and a pathogen (U. appendiculatus). Infection type is the character of this relationship, not of the host, nor of the pathogen; reaction and pathogenicity, respectively, designate the character of the host and pathogen. Infection type is a measure of these two characters. The phenotypic expressions of these characters are designed as low or high for infection type, resistant or susceptible for reaction and avirulent or virulent for pathogenicity.

some tropical and subtropical areas the fungus can survive solely by urediniospores on a succession of host plants, but in other areas telia are considered to be the means of carryover (Milbrath, 1944; Zaumeyer & Thomas, 1957). No difficulty has been reported in inducing germination although a rest period was found to be necessary (Andrus, 1931; Harter et al., 1935).

The aecia have not been frequently reported, but as they are small, white and inconspicuous they could be expected to be more common than the records listed in Table III indicate.

2) Asexual reproduction.

(a) Mutation. There is abundant evidence for mutation in the cereal rust fungi. Differences in mutation rates for particular genes in the pathogen are also well documented e.g. a particularly high mutation rate towards virulence in Puccinia graminis Pers. f. sp. tritici Eriks. & E. Henn. has been reported in wheat (Triticum aestivum L.) plants with Sr15 and a lower, but still relatively high rate rate with Sr5; this contrasts with a very low rate with SrTt1 (Luig & Watson, 1970).

(b) Other processes in which genetic change follows anastomosis of the hyphae (Watson, 1970).

3) By introduction of new genetic material. There is evidence that urediniospores may be carried considerable distances in wind currents. For example, new species and races of several rust fungi have been reported in New Zealand shortly after their appearance on the Eastern Coast of Australia, approximately 2,100 kilometres to the West. No such movement from New Zealand to Australia against the prevailing westerly winds has ever been detected (Luig & Watson, 1970).

Use of Differentials

In race surveys each field collection is given a survey number which may later become a culture number. It is inoculated directly, or after increase on a universal suscept, onto the differential set. Mixtures are common in surveys so care must be taken to detect this. In such instances, further inoculations should be carried out with cultures derived from single pustules on the differential(s) showing the mixed reaction.

Since environmental conditions influence disease reaction, as much standardization as possible is necessary. High concentrations of inoculum which produce abnormally small pustules should be avoided (Davison & Vaughan, 1964).

Some genes are temperature sensitive i.e. inoculation with one culture gives a resistant reaction at one temperature and a susceptible reaction at another. If possible, the use of stocks with such genes should be avoided; otherwise details of cardinal temperatures should be established and the temperature standardized.

Suggested Plan of Action

I. The following plan of action is suggested for choosing a set of differentials.

1) Ask all workers concerned which accessions he or she
(a) uses in race surveys and plans to use in future, distinguishing between those which (i) have been used directly or indirectly in the commercial situation and are being or will be used in breeding work; these have provided and will provide selection pressure, and (ii) have not been used in this way.

For example, of the Australian differentials used recently, Redlands Greenleaf strains B and C, derived from California Small White and Brown Beauty, have been widely used in Eastern Australia, whereas Epicure has only been used in home gardens and the other differentials - Golden Gate Wax, C.C.G.B. 44 and Veracruz 1 A 6 have not been grown outside Rust Nurseries. It is desirable to have both (i) and (ii) representatives in

a differential set; (i) are obviously very relevant in breeding programmes and the changes in the avirulence/virulence formulae and (ii) provide additional markers which can enable the changes in virulence of the races to be traced with greater accuracy.

2) Would like to have other workers use. Distribute seed of these and many other accessions of potential value to all workers interested in testing them in pure culture seedling inoculation tests. As mentioned earlier it is important that seed of currently used differentials be exchanged. The wider the range of races used the greater the number of resistances we can expect to distinguish. Field results are only of preliminary value in choosing differentials because of the likelihood of several races being present.

3) Examine results and choose differentials apparently with different genes for specific resistance (Loegering et al., 1971).

Where two accessions have identical reactions to all the races used, preference whould be given to those with small seed, bush habit and other features which facilitate the maintenance of seed stocks. There are advantages in having differential stocks which can be distinguished on phenotypic characters such as shape of seed and leaves and colour of seed, hypocotyl and pod.

It is inevitable that any set of differentials chosen on an international basis will contain accessions which would not be routinely used by all workers. For example, the differentials, 181, Pinto and 780 are susceptible to all races of rust recently distinguished in Australia, and except for Pinto which has served as a universal suscept I do not use them routinely, but only for characterizing each race detected on a more limited set. Since the interaction between host and pathogen is a dynamic one, additions will need to be made to the set from time to time.

It would be desirable for bean rust workers to choose initially a greater number of differentials than are necessary for their immediate needs. These would be available for an in-depth study of virulence changes in the bean rust fungus as well as to those associated with breeding programmes.

4) When differential genotypes have been chosen it is proposed that each be given a sequential number. Those likely to be most commonly and widely used would be listed first. New differentials would be assigned a number by Dr Meiners on request, provided that a minimum amount of justification data and a given quantity of seed are supplied. It is essential that all differentials be derived from the progeny of a single typical plant and show no sign of segregation for rust reaction. A given quantity of seed should be deposited with some established long term storage collection such as CIAT or the National Seed Storage Laboratory, Fort Collins, Colorado, U.S.A. Seed from the same original plant would be used for increase and distribution to interested workers.

II. The reaction needs to be examined to determine the cut-off point between avirulence and virulence. A system of naming the avirulence/virulence formulae is required as the formula is usually too long to be easily used as such. Day (1974) gives a detailed account of several systems. There are two main groups of systems; those in which results on a fixed set of differentials are required and those in which a more limited set may be used

1) Systems in which a fixed range of differentials are required to name races provide a comprehensive history of changes in the avirulence/virulence formulae and much valuable background information for breeders.

Watson & Luig (1963) added regional supplemental differentials to the International Differentials established by Stakman & Levine (1922) for distinguishing races of Puccinia graminis f. sp. tritici on Triticum spp. Watson & Luig used the chronological race number allocated by Stakman and co-workers e.g. 34, followed by the suffix ANZ (Australia and New Zealand) and the numbers of the differentials on which the strain was virulent e.g. 2,3,7 to give the final designation 34-ANZ-2,3,7.

This system could be adapted quite satisfactorily to the bean rust situation although a new key would need to be drawn up. The original set of six differentials of Harter & Zaumeyer would appear to be too limited as the basis for the first number so that the listing of numbers of the supplementals would soon become too long and cumbersome. In addition there is the likelihood of variation of some of the genotypes since the early work was done so that the old and recent results could not be validly compared. However, I doubt that we have at present the information needed to construct the new key.

Habgood (1970) outlined a naming system based on binary notation. A unique decanery or base 10 number is generated for each race from a binary number. This latter number is derived from the array or 0 or 1 alternatives, representing avirulent or virulent reactions on a set of differentials in a fixed linear order. For example, a pathogen culture inoculated onto a set of eight differentials (A to H) may be virulent on B,D,E, and F:

Differential host	H	G	F	E	D	C	B	A
Reaction	R 0	R 0	S 1	S 1	S 1	R 0	S 1	R 0
Binary value	(2^7)	(2^6)	2^5	2^4	2^3	(2^2)	2^1	(2^0)
Decanery value			32	16	8		2	

Designation of culture = 32 + 16 + 8 + 2 = 58.

When new differentials are needed, they may be added to the left of the current order. Intermediate reactions cannot be readily expressed in this system whereas they can be included in other systems. While the race designation is derived in a logical and automatic manner it would be at least initially more complex than the other systems.

Johnson et al. (1972) included Habgood's naming system in their proposal for naming races of Puccinia striiformis Westend. on wheat. They outlined a set of seven Internation Differentials and a set of European Differentials with a third set possibly being necessary to accommodate more localized specialization.

2) Because of the diverse origins of the pathogen races and differences in the varieties used in many countries there have been difficulties in deciding on International Differentials in some host pathogen systems and schemes have been devised to permit greater flexibility than those considered under 1).

Loegering & Browder (1971) proposed the allocation of sequential numbers and assignment of temporary codes to avirulence/virulence formulae of leaf rust caused by Puccinia recondita Rob. Ex. Desm. F. sp. tritici on wheat. The code included the year, possibly a State or laboratory number and a formula number e.g. 70 KS1 (1970, Kansas, formula 1).

Formulae	Formula codes
1,4,5/6,8	63KS1
1,4.5,6,8	63KS2

This system could also be used satisfactorily by bean rust workers. All these systems have their merits and problems according to the purpose for which they are used.

There is no right or wrong scheme for naming avirulence/virulence formulae. They all require some basic details of the system to be included in surveys and publications.

The basic requirements are:

(i) A language for communication between the race survey workers and the bean breeders.

(ii) A degree of permanence such that
(a) trends can be followed over years, and
(b) spore interchange between regions can be detected.

Results obtained in several seasons may be necessary before we can choose a system which suits the needs of bean rust workers and is easy for them to follow.

It is desirable that type cultures of important races be stored in liquid nitrogen (Cunningham, 1973).

III. It is desirable that genetic analysis be carried out on the differentials and that the genes for specific resistance be separated, where necessary, and named. Reference seed stocks and cultures of the pathogen used in such analysis should be placed in long term storage.

Value of Race Surveys

Race surveys on a well chosen set of differentials are a necessary guide to breeding programmes designed for resistance to bean rust. There is insufficient information in the literature at present to indicate which approach is likely to be most effective in reducing losses from bean rust in varieties released in future. Combinations of genes for specific resistance is one promising approach; such a strategy has been widely and successfully used to provide protection against wheat stem rust (Luig & Watson, 1970; Rajaram & Luig, 1972). However, the use of non-specific resistance which has been reported to give valuable protection against bean rust in at least some situations (Vieira, 1972; Ballantyne, 1974) is another possible approach. This has been employed by potato (Solanum tuberosum L.) breeders where the production of varieties with specific resistance to Phytophthora infestans (Mont.) de By. was very rapidly followed by the appearance of races virulent on them (Gallegly & Niederhauser, 1959). Incorporation of both types of resistance could be a desirable aim, although a clearer understanding of the nature of these resistances and their genetic control is probably necessary for this.

The details of the virulence genes present, the combinations in which they occur and a knowledge of the means by which changes in them occur will provide the basis for the decision of which approach bean breeders could most profitably adopt.

In a programme based on a strategy of many genes for specific resistance these details are of the utmost continuing importance. In such a programme accessions which remain resistant to all collections, or show only an occasional susceptible reaction in comprehensive surveys may be useful as a source of resistance.

In a programme aiming for non-specific resistance an understanding of the specific resistance in the material considered for use is necessary. The slow rusting character of some varieties thought to have non-specific resistance has subsequently proved to be specific (Browder, 1973).

When early work on race differentiation was carried out it was thought that breeding work carried out in one country could be of direct value in other countries. However, experience has now shown that cultures with the same avirulence/virulence formulae determined on a limited set of differentials in one country will not necessarily be identical with each other and these are even less likely to be identical with cultures having the same formula in another continent. While these cultures have

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TABLE I

Reported Races of Bean Rust

Country	Number of Races	Reference
United States of America		
mainland	35	Harter & Zaumeyer, 1941 Fisher, 1952 Sappenfield, 1954 Zaumeyer, 1960 Hikida, 1961 Goode, 1961 McMillan, 1972
United of America		
Hawaii	2	Parris & Matsuura, 1941
Mexico	31	Crispin & Dongo, 1962
Brazil, Minas Gerais	26	Netto et al., 1969
Brazil, Rio Grand do Sol	16	Augustin and da Costa, 1971
Costa Rica	11	Christen & Echandi, 1967
East Africa	8	Howland & Macartney, 1966 Leakey, 1971
Australia	3 8	Waterhouse, 1954 Ogle & Johnson, 1974
New Zealand	3 or 4	Yen & Brien, 1960
The Netherlands	2	Hubbeling, 1957

TABLE II

Bean Rust Differentials Used in Race Surveys Throughout the World

ACCESSION	COUNTRY AND RACE SURVEY REPORTS																					
	U.S.A.	Harter & Zaumeyer, 1941	Fisher, 1952	Sappenfield, 1954	Zaumeyer, 1960	Hikida, 1961	Goode, 1961	McMillan, 1972	BRAZIL	- Minas Gerais	Netto *et al.*, 1969	- Rio Grand do Sol	Augustin & da Costa, 1971	MEXICO	Crispin & Dongo, 1972	COSTA RICA	Christen & Echandi, 1967	EAST AFRICA	Howland & McCartney, 1966	AUSTRALIA	Waterhouse, 1954	Ogle & Johnson, 1974
3*		+	+	+	+	+	+	+			+		+	U.S. differentials susceptible to most species			+	?	6 unspecified U.S. differentials		+	+
181*		+	+		+		+						+					?			+	+
643*		+	+	+	+	+	+	+			+		+				+	?			+	+
650		+	+	+	+	+					+		+				+	?			+	
765*		+	+	+	+	+	+	+			+		+				+	?			+	+
780*		+	+	+	+	+	+	+			+		+				+	?			+	+
814*		+	+	+	+	+	+	+			+		+				+	?			+	+
Golden Gate Wax*			+	+	+	+	+	+			+							?			+	+
Z4			+																			+
Dade*								+														
Mulatinho*													+									
Cuva 168-N*													+									
Canario 101*													+									
Aquascientes 13															+							
Guerrero 6															+							
Guerrero 9															+							
Guanajvato 10A-5															+							
Mexico 6															+							
Mexico 12															+							
Veracruz 10															+							
Negro 150															+							
Tengeru Sel. 8																	+					
"Wonder var. e.g. Hawkesbury Wonder																					+	
Brown Beauty*																					+	
Redlands Greenleaf B*																						+
Epicure*																						+
C.C.G.B. 44*																						+
Veracruz 1A6*																						+

* Currently available

TABLE III

Reports of Aecia of the Bean Rust Fungus

Area	Reference	Remarks
NORTH AMERICA		
	Andrus, 1931	All spore stages (0,I,II,III & IV) obtained in green-house
Oregon, U.S.A.	Zaumeyer & Thomas, 1957	
Oregon, U.S.A.	McWhirter, quoted by Zaumeyer & Thomas, Milbrath, 1944	Uredial stage appeared three weeks after first occurrence of aecia
New York State, U.S.A.	Jones, 1960	
British Columbia, Canada	Eastman, 1943	
EUROPE		
Holland	Hubbeling, 1955	Aecia formed in all regions where climbing beans were grown on old poles contaminated with the fungus.
France	Guyot, 1957	Generally rare
Germany		
Switzerland		
England	Wilson & Henderson, 1966	Generally rare but recorded at five locations.
NEW ZEALAND		
Auckland	Brien & Jacks, 1954	

250

24 = 1,2,3,5,6,7/4

25 = 2,5/1,3,4,6,7

26 = 1,2,3,5/4,6,7

27 = 1,3,5/4,6,7

28 = 1,3,4,5,6,7,8/9

29 = 3,5/1,4,6,7,8,9

30 = 1,5,8,9/3,4,7

Sappenfield, 1954

31 = 1,3,4,5,6,7/2

Zaumeyer, 1960

32 = 1,3,4,5,6,7/2

Hikida, 1961

33 = 3,5/1,4,7 6?

Goode, 1961

34 = 1,3,4,5,6,7/2,8? ^{3}/10

McMillan, 1972 added Dade 10

35 = 6,8/1,3,4,5,7,10

BRAZIL - Rio Grand do Sol

Augustin & da Costa, 1971

11 Mulatinho

12 Cuva 168-N

13 Canario 101

assuming tentatively that 0,1,2,3 are avirulent
4,5 are virulent

B 1 = 3,5,7,12,13/1,2,4,11

B 2 = 3,7,12,13/1,2,4,5,11

B 3 = 2,3,5,7,12,13/1,4,11

B 4 = 5,7,12,13/1,2,3,4,11

B 5 = 3,4,12,13/1,2,4,7,11

B 6 = 3,12,13/1,2,4,5,7,11

B 7 = 7,12,13/1,2,3,4,5,11

B 8 = 5,12,13/1,2,3,4,7,11

B 9 = 2,5,7,12,13/1,3,4,11

B10 = 2,3,5,12,13/1,4,7,11

B11 = 5/1,2,3,4,7,11,12,13

APPENDIX I

Avirulence/Virulence Formulae of Reported Races of Bean Rust

U.S.A. - Harter & Zaumeyer, 1941

assuming tentatively that 0 - 7 is avirulent
8 -10 is virulent

(1) denotes U.S. No. 3
(2) " 181
(3) " 643
(4) " 650
(5) 765
(6) 780
(7) 814

Race 1 = 1,5,6,7/2*,3,4

2 = 5,6,7/1,2,3,4

3 = 3,5,6/1,2,4,7

4 = 1,3,5,6/2,4,7

5 = 5,6/1,2,3,4,7

6 = 1,5,6/2,3,4,7

7 = 1,5,6/2,3,4,7

8 = 1,3,5,6/2,4,7

9 = 5,6/1,2,3,4,7

10 = 1,3,4,5,6,7/2

11 = 5,6/1,2,3,4,7

12 = 5,6,7/1,2,3,4

13 = -/1,2,3,4,5,6,7

14 = 1,5/2,3,4,6,7

15 = 3,4,6/1,2,4,7

16 = 1,5/2,3,4,6,7

17 = 2,3,5,6,7/1,4

18 = 1,2,5,6/3,4,7

19 = 5,6/1,2,3,4,7

20 = 5/1,2,3,4,6,7

Fisher added
8 Golden Gate Wax & 9 Z4

21 = 1,2,5,6,7/3,4

22 = 1,2,5,6,7/3,4

23 = 1,2,5,7/3,4,6

* underscoring indicates the upper or lower limit of avirulence or virulence

B12 = 5,7,12,13/1,2,3,4,11

B13 = 1,2,3,5,7,12,13/4,11

B14 = 2,3,7,12,13/1,4,5,11

B15 = 3,5,7,12,13/1,2,4,11

B16 - 3,5,7,12/1,2,4,11,13

- Minas Gerais

Netto, et al., 1969

assuming tentatively that 1,2,3,4, are avirulent
5, is virulent

FM1 = 1,3,4,5,6,7/8

2 = 1,3,4,5,6,7,8/-

3 = 1,3,4,5,6,7,8/-

4 = 1,3,4,5,6,7/8

5 = 1,3,4,5,7,7/8

6 = 1,3,4,5,6,7,8/-

7 = 1,3,4,5,6,7/8

8 = 1,3,5,6,7/4,8

9 = 1,3,6,7/4,5,8

10 = 1,3,5,6,7,8/4

11 = 1,3,5,6,7,8/4

12 = 1,3,5,6,7/4,8

13 = 1,3,5,6,8/4,7

14 = 3,5,6,8/1,4,7

15 = 1,3,5,6,7,8/4

16 = 1,3,5,6/4,7,8

17 = 3,5,6,7,8/1,4

18 = 1,3,5,7,8/4,6,8,

19 = 1,3,5,6,7/4,8

20 = 1,3,5,6/4,7,8

21 = 1,3,5,6,7/4,8

22 = 1,3,5,6,7/4,8

23 = 3,5,6,7/1,4,8

24 = 1,3,5/4,6,8,8

25 = 1,3,6,7/4,5,8

26 = 3,6/1,4,5,7,8

AUSTRALIA

Waterhouse, 1954

14 = Brown Beauty

2 = 2,5,6,7,14/1,3,4,

17 = 2,3,5,6,7,14/1,4

17A= 2,3,5,6/1,4,14

Ogle & Johnson (in press)

15 Redlands Greenleaf B

16 CCGB 44

17 Vera Cruz 1-A-6

18 Epicure

A = 3,5,7,15,16,17,18/1,4,6,8,14

B = 3,5,7,8,16,17,18/1,4,6,14,15

C = 3,5,7,8,15,16,17/1,4,6,14,18

D = 3,5,7,15,17/1,4,6,8,14,16

E = 3,5,7,8,15,16,17/1,4,6,8,14

F = 3,5,7,8,17,18/1,4,6,14,15,16

G = 3,5,7,8,16,17,18/1,4,6,8,14,15

H = 5,7,8,16,17,18/1,3,4,5,6,7,14,15

Remarks

181 is of doubtful value in distinguishing races.

Several of the Harter & Zaumeyer races 1-20, and Fisher races 20-30 and the Netto, et al. are similar and may not be significantly different.
Similar formulae given different strain designations have been bracketed.

ESTABLISHMENT OF A GROUP OF BEAN RUST DIFFERENTIALS AND A SYSTEM OF RACE NOMENCLATURE FOR USE IN GREENHOUSE TESTING

CIRCULAR 1

INTRODUCTION

Race surveys based on the reactions of a carefully chosen set of host differentials are a useful guide for breeding programmes aiming for rust resistance. A knowledge of the virulence genes present, the combinations in which they occur, and of patterns by which changes occur, can provide the basis for decisions regarding plant breeding strategy. This applied both to resistance expressed as

(i) a fleck or small pustule (sometimes referred to as vertical resistance) and

(ii) slow development of rust in the field on genotypes susceptible as seedlings to all of the prevalent races present (sometimes referred to as horizontal resistance)

The value of a common group of host differentials used by all bean rust workers is that it provides a basis for application of results obtained in one geographic area to others. (see reference (1)).

The procedure is to:

- Compile a list of potential differentials including those used previously, genotypes being used as parents in rust resistance breeding programmes and lines resistant to a wide range of races or to all known races.
- Distribute from a common source.
- Inoculate the potential differentials (PD) with selected bean rust isolates.
- Apply the gene-for-gene theory to the results and allocate hypothetical genotypes.

These results will enable us to choose a suitable group of host lines, including

(i) accessions used directly or indirectly in the commercial situation and in breeding programmes and

(ii) accessions not used in this way.

It is desirable to have both (i) and (ii); (i) are obviously relevant to breeding situations and in monitoring important changes in the race situation, but (ii) provide additional markers enabling changes in virulence to be traced with greater accuracy and a rust isolate to be identified more precisely.

The value of a common rating (infection type) system is that it provides a basis for comparisons of data obtained by all investigators (see reference (2).

The value of a system of designating the avirulence/virulence formulae of isolates is that it provides:

(i) a language for communication between the race survey workers and bean breeders and

(ii) a degree of permanence such that

a. trends may be followed over years and
b. possible spore interchange between regions may be detected.

This circular -

A. Reviews the current list of potential differentials and suggests some additions

B. Discusses standardization of methods

C. Outlines some basic features of infection type reaction assessments

D. Suggests a rating system

E. Provides illustrations of various infection types

F. Proposes a naming system

A recent local race survey report is enclosed as an example.

A. LIST OF POTENTIAL DIFFERENTIALS

(i) Current List

Table I lists possible host differentials considered at the Bean Rust Workshop at CIAT in October, 1974. Please advise of any accessions which you have nc received either from me, CIAT or the originator. For example, Dr. Vakili has distributed seed of his lines and Dr. Coyne has distributed seed of Tara.

It is important that seed from a common source be used for the testing outlines in this circular. Where seed with the same name is received from severa sources, each lot should be maintained and treated separately, as each may differ in genotype. For example, 643 in Dr. Meiners' collection reacts differently in his tests to the 643 used in Australia, although in tests with Australian culture they behave identically. In the Australian collection the Meiners 643 is referre to as 643 - B1595 and the Australian 643 as 643 - B965.

(ii) Some useful additions include

a. Bonita. Reports in the literature, comments at the CIAT meeting and our own results here indicate that this has resistance in several geographic areas. In Australia Bonita behaves as a very slow ruster in the field, but in greenhouse tests with all known Australian races it produces a pustule which is slightly but significantly smaller than that characteristic of a susceptible reaction.

b. Cacahuete 72 which is listed as being used as a rust-resistant parent in the CIAT programme (3).

c. The following beans which are or have been rust resistant in your countr

- Widely grown cultivars
- Recently released cultivars
- Advanced breeding lines
- Parents in breeding programmes

Could you please advise of such lines and send small seed samples to me and other cooperators. It is particularly important to obtain this information now to ensure that the differentials chosen are relevant to the breeding being carrie out in various geographic areas.

The Australian differential set may be used to illustrate this. The cultiva Brown Beauty, Gallaroy, Redlands Greenleaf B and Redlands Greenleaf C were select for rust resistance and have been widely grown in rust-liable areas. Thus they may have exerted selection pressure on the bean rust fungus and influenced the

evolution of the Australian races. Actopan X Sanilac Selection 37 is an advanced breeding line selected for rust resistance. CCGB 44, Golden Gate Wax and Vera Cruz 1A6 have not been grown outside experimental plantings and Epicure is grown only on a limited scale in home gardens. These latter four possess additional markers which may enable changes in virulence of the pathogen to be traced with greater precision. Other accessions added after the 1974-75 survey are Aurora, Bonita, Cornell 49-242, NEP 2 and PR 5 which have resistance to all known Australian races and have been used in crosses for genetic analyses of rust resistance and possibly for breeding.

(iii) Some additions which are optional:

a. Table II lists those resistant accessions from the CIAT Annual Report, 1974 not already in the list of potential differentials (PD).

b. Entries in the International Bean Rust Nurseries not already in the PD.

c. Other promising accessions being used in the CIAT programme. While these may not have resistance to Colombian races, some may be resistant in other areas.

While results with these would be valuable, cooperators with only limited time or resources may not wish to include these.

Please advise if you need seed.

It is desirable but not essential that differentials have only a single gene for rust resistance. Single gene stocks permit the determination of more precise virulence genotypes. Our genetic analyses have shown that some differentials have more than one and as many as three genes for resistance; some differentials have genes in common. I am deriving homozygous single gene stocks* from recombinant F_3 populations which are segregating monogenically. These will be distributed as soon as possible.

If any other cooperator has carried out genetic analyses and derived single gene stocks, I would appreciate receiving details and seed.

B. METHODS OF TESTING AND ASSESSING REACTION

These points were discussed at the CIAT Workshop.

Isolates

Where cooperators already have rust isolates with considerable differences in avirulence/virulence formulae as determined on a limited set of differential hosts, they should attempt to select isolates showing the greatest range of variation.

It is desirable that each isolate should have originated from a single pustule.

Number of Plants

Seven to ten plants of each accession for each isolate would be satisfactory. Fewer plants could be used if the stocks were uniform but many lines are of mixed genotype for rust reaction with certain isolates. A record of the number of seedlings inoculated is often desirable to distinguish "escapes" (from late

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* These are from crosses between Sanilac (used as an Australian Universal Suscept and Redlands Greenleaf B (RGB), Gallaroy, Actopan X Sanilac Selection 37, and NEP 2. RGB and Gallaroy have genes for rust resistance from the Australian accession of 643

germination) from inoculated plants showing no signs of infection. If the same number of seeds is sown for each accession, the only record needed is for those where some seeds failed, or were slow to germinate. For example, if seven seeds are sown for each accession, record only where less than seven are inoculated. I write this number on the pot label so it is readily available at note taking.

Ensure that a known susceptible cultivar is included as a reference for each inoculation.

Stage of Plant Growth at Inoculation

Many workers inoculate when the primary or unifoliate leaves are 1/2 to 2/3 expanded and it would be desirable to standardize on this stage.

Spore Concentration

High concentrations of spores may produce abnormally small pustules and should be avoided (4). Some method of adjusting spore concentrations is desirable so that the pustule density 14 days after inoculation is approximately 8-10 pustules per square centimetre. My experience with using Odourless Mineral Spirit as a suspending agent, is that fresh spores need to be added to give a faint brown colour; when using spores 4-6 weeks old the suspension is adjusted to a slightly darker brown. A suitable rate is 7-10 mls to 45-50 4" pots in an area of 4800 square centimetres.

Conditions after Incubation

It is important that all plants be kept under similar temperature and ample light conditions. I remove apical growth 7-8 days after inoculation to improve light penetration and facilitate handling.

Results obtained using plants kept under growth chamber conditions with artificial light may differ from those obtained in the greenhouse with natural light. For this study greenhouse results are desirable.

A record of greenhouse temperatures is desirable.

C. REACTION ASSESSMENTS

As indicated in (2) reaction assessments are always made relative to a susceptible reference cultivar as the pustule size of susceptible reaction may vary from one set of conditions to another. Sanilac and Pinto UI 111 or 650 are suitable reference cultivars in the Australian situation.

About 14 days after inoculation is a satisfactory time for making assessments of infection type.

The pustule size ranges outlined by Davison & Vaughan (5) have been used by many workers. I have needed to add another category as the pustules on the lower leaf surface at this laboratory are much larger than the upper limit of this scale.

Enclosed with this circular are photographs illustrating infection types and a card with reference circles of indicated sizes which I have used. However, the size of the pustule relative to the susceptible control is the important feature rather than absolute size.

Infection types involving large pustules similar to the susceptible control are interpreted as susceptible (or high) reactions.

Where the pustule is slightly but significantly smaller than that on the

susceptible reference cultivar, the reaction may be termed intermediate. This should be regarded as a low reaction and described in terms of the infection type.

Infection types involving no sign of infection, a chlorotic or necrotic fleck or a small pustule are interpreted as resistant (or low) reactions.

It should be noted that pustule size in the greenhouse does not always give a good indication of the protection conferred under field conditions. While plants producing either no sign of infection, flecking or small pustules in the greenhouse normally show little or no rust under field conditions where the same isolates of rust are present, no such generalizations may be made for interactions where an intermediate sized pustule occurs. For example, some plants of Gallaroy produce an intermediate sized pustule with some races in greenhouse tests; they show little if any rust when these races are present under field conditions. However, other host cultivar/rust isolate interactions which produce an intermediate sized pustule in the greenhouse may result in considerable development of rust under field conditions.

D. RATING SYSTEM

The enclosed reference card with circles of specified sizes may be helpful. This is similar to that issued by Davison & Vaughan but is extended with a category larger than 800μ. I have also substituted ";" (fleck) as used by cereal rust workers instead of 1 in the Davison & Vaughan system.

The infection type rating system I have used is

0 No sign of infection

; Necrotic or chlorotic flecking

1 Pustules smaller than 300μ

2 Pustules 301 to 499μ

3 Pustules 500 to 799μ

4 Pustules larger than 800μ

Pustule sizes close to the lower size limit of each category are indicated by a minus (-) and those close to the upper limit by a (+). Necrosis may be indicated by N for small necrotic area and N+, N++, N+++ for larger lesions according to size. Readings are normally made on the lower leaf surface but where there is a significant difference between the upper and lower surfaces relative to the susceptible control the infection type on the upper surface is recorded before a slash mark (/) and that on the lower surface after the mark.

Where there is a range of pustule sizes on one leaf the more common one is recorded first followed by the less common e.g. ;1 indicates that many chlorotic or necrotic flecks and few very small pustules were present.

Where individual plants of one accession differ in reaction, the results and separated by a comma, e.g.;1, 2+3- means that within one variety some plants produced flecks and small pustules whereas other plants of the same accession produced intermediate sized pustules when inoculated with the same race.

E. ILLUSTRATIONS - separate sheets

F. RACE NOMENCLATURE

The results from cooperators will indicate which accessions react similarly

and which behave differently. A limited number of different genotypes may then be selected as International Differentials.

When submitting results please indicate

1. Which differentials you think should be included in the international group and the reasons for this decision. If this group is too small it will give only limited information; if it is too large it may become cumbersome and some people may be reluctant to use all genotypes. Two accessions which should be considered are PR 5 and Cornell 49-242; PR 5 is being used by CIAT as a source of rust resistance and is resistant to all known bean rust races; Cornell 49-242 is being used by CIAT as a source of resistance to anthracnose and is resistant to bean rust races present in some areas. Others to be considered are rust resistant parents used in breeding programmes and accessions with resistance to all known races.

2. Which differentials you would include in the regional supplemental group. This would normally include resistant varieties currently grown in your region, advanced breeding lines and parents in rust resistance breeding programmes, where these are not in the international group. Some other accessions with value as markers are also desirable.

3. Which differentials you find difficult to maintain because of such features as sensitivity to daylength, climbing habit and other features.

At the CIAT Workshop there appeared to be general agreement that the race system outlined by Ling & Ou* would be a suitable basis for naming bean rust races. This system is of designated avirulence/virulence formulae 256 races determined on eight differentials. It consists of a dichotomous arrangement of susceptible and resistant reactions of the differentials. The race numbers can therefore be determined without referring to a race chart. This system readily accommodates intermediate reactions.

While Ling & Ou used eight differentials giving 256 races, other numbers could be used e.g. seven differentials would give a theoretical limit of 128 races; nine differentials would give 512 races.

The following naming system agreed upon at the CIAT meeting is outlined for consideration:

1. Chose an international group as outlined above. Assign a sequential letter A,B,C,D,E,F,G, etc. as in Ling & Ou
2. Assign sequential numbers to other supplemental differentials
3. Assign code letters to different geographic regions
   e.g. NA for North America
   SA for South America
   A for Africa
   E for Europe
   ANZ for Australia and New Zealand

In some areas an additional letter may be needed to indicate the country e.g. CAN for Nicaragua, SAB for Brasil, SAC for Colombia, AK for Kenya, EG for Germany.

Race designation may then be made as in the following hypothetical example:

A North American collection is applied to the international set of eight and

*Ling, K.C. and Ou, S.H. (1959). Standardization of the international race numbers of Pyricularia oryzae. Phytopathology 59:339-342.

and a supplemental set of six which have been assigned the numbers 8, 10, 11, 18 and 20. On the international group it is avirulent on A,B,F,G, and H and virulent on C,D, and E. It is thus determined as IC - 8 (see p. 340 of the Ling and Ou paper). On the supplemental group it gives a resistant reaction on 8, 12 and 18 and a susceptible reaction on 10, 11, 12 and 20. It may then be referred to as IC-8 NA 8, 12, 18, 10, 11, 20 or more simply as IC - 8 NA 10, 11, 20 if all the supplemental differentials used and clearly indicated.

Grateful acknowledgement is made to Dr. R.A. McIntosh for discussion.

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1976

References

1. Ballantyne, Barbara 1974. Development of a set of international differential varieties and a standard nomenclature of races. Bean Rust Workshop, CIAT, Cali, Colombia, October 1974.

2. Oliveira, Eliane Augustin 1974. An infection type rating system for the bean rust. Bean Rust Workshop, CIAT, Cali, Colombia, October 1974.

3. Annual Report for 1974, CIAT, Cali, Colombia.

4. Davison, A.D. & Vaughan, E.K. 1964. Effect of uredospore concentration on determination of races of *Uromyces phaseoli* var. *phaseoli*. Phytopathology 54: 336-338.

5. Davison, A.D. & Vaughan, E.K. 1963. A simplified method for identification of races of *Uromyces phaseoli* var. *phaseoli*. Phytopathology 53: 457-459.

TABLE 1 - POTENTIAL DIFFERENTIALS

1.	643 - Meiners accession	U.S. differentials
2.	Pinto U.I. 111	
3.	814	
4.	Golden Gate Wax	
5.	Dade	
6.	Mulatinho	Brazilian differentials
7.	Cuva 168 - N	
8.	Canario 101	
9.	Brown Beauty	Australian Differentials
10.	Redlands Greenleaf B	
11.	Redlands Greenleaf C	
12.	CCGB 44	
13.	Vera Cruz 1A6	
14.	Epicure	
15.	643 - Australian accession	
16.	Actopan X Sanilac Sel. 37	
17.	Bayo	Peruvian Differentials
18.	Bayo Camana	
19.	Caraotas	
20.	Panamito Mejorado	
21.	Panamito Sanilac	
22.	Canario LM	
23.	Cochaco	
24.	Canario Divex 8120	
25.	V 1756 Ecuador 66	Cambridge accessions
26.	IV 1210 Puerto Rican	
27.	V 1154 Black Turtle Soup TA 31	
28.	Comptasse de Chambord	
29.	Aguascalientes 13	Mexican differentials
30.	Guerrero 6	
31.	" 9	
32.	Guanajuato 10A - 5	
33.	Mexico 6	
34.	" 12	
35.	Veracruz 10	
36.	Negro 150	
37.	Canario 101	
38.	15R - 87 (PR5)	Resistant to all known strains
39.	Mexico 309	
40.	Ecuador 299	
41.	Cornell 49-242	Others
42.	Aurora	
43.	NEP 2	
44.	15R - 277 (PR - 15)	
45.	Westralia	
46.	Jamapa (Venezuela)	
47.	Mexico 142	
48.	ICA Guali	
49.	Turrialba 4	
50.	" 2 (51051)	
51.	Porillo No 1	

52. El Salvador 184
53. Porillo Sintetico
54. " 70
55. Venezuela 54
56. La Vega
57. IAN 5091
58. Tara
59. Cacahuete 72
60. P.I. 152326
61. " 165426 white seeded
62. " 307824
63. " 310739
64. " 310814
65. " 310878
66. " 313524
67. " 207198
68. " 209805
69. Bonita

TABLE 2 - BEANS RESISTANT TO RUST AT CIAT AND NOT ON CANDIDATE DIFFERENTIAL LIST

Guatamala 487
Trigo E
73 Vul 3,215 (PI 313694)
" 3,231 (Puebla 87)
" 3,241 (Chiapas 2-A-3)
" 3,242 (" 3-A)
" 3,248 (Hidalgo 20)
" 3,285 (S-387-A-N)
" 3,287 (S-166-A-N)
" 5,506-1 (Frijol Chapin)
" 150-1-1 (51,051)
" 5375 (Line CH-11-198-6-C-3-C)
" 3,690 (Guatamala 209)

TABLE 3 - ENTRIES IN IBRN NOT ON CANDIDATE DIFFERENTIAL LIST

PR1
" 2
" 3
" 4
" 6
" 7
" 9
21
PI163372
" 165435
" 199044
" 203958
" 207262
" 226883
" 226895
" 313524
" 313664
" 313667
" 319649
A X S sel 39
" 51
Bush Romano 14
Caballero
Compuesto Cotaxtla
Costa Rica 1031
Cuilapa 72
Diacol Calima
Diacol Nima
Guatamala 416
Honduras 46
ICA Guali
ICA Pijao
" Tui
La Vega
Linea 34
" 37
Manteigao Preto 20
Mexico 142-N
Mexico 235
Miss Kelly
Mogul
Mountaineer H W R
Negro de Chincha
Negro Jalpatagua
Negro San Ramon 5
Panamito Corriente
Pinto Serrano
Preto 897
Redlands Autumncrop
" Pioneer
Portland Red
Portugal
Ricobaio 1014
Turrialba 1
Venezuela 54
Villa Guerrero
Vi 1013
4691-54.1
11.411
SB - 30 - iI - pM - pI
142 - MI - pM - pI
173 - ml - pM - pl