DISEASE RESISTANCE IN THE *SENECIO VULGARIS* L./ *ERYSIPHE FISCHERI* BLUMER WILD PLANT PATHOSYSTEM.

by

JOSETTE R. BEVAN

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and

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To

Mum and Dad

and

Jonathan

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Summary.

An investigation was made of the frequency and distribution of race specific resistance in two populations of groundsel (Senecio vulgaris) to the powdery mildew fungus Erysiphe fischeri. Ten different powdery mildew isolates were tested on a sample of 98 plants from Glasgow and 151 from the National Vegetable Research Station (N.V.R.S.), Wellesbourne. Five of the isolates were collected at Glasgow and 5 from N.V.R.S. Additional plants were assessed using only 5 isolates from Glasgow. In total, 247 plants from Glasgow and 266 from N.V.R.S. were assessed. Plants were tested with several isolates at a time by using detached leaves on benzimidazole supplemented agar.

Generally the frequency of plants detected with race specific resistance to each isolate was low, ranging from 1% to 10%. However, the frequency of resistant plants in the N.V.R.S. population to one isolate from Glasgow, G9, was relatively high, at 35%. The majority of these plants originated from a small, intensively sampled plot of 17 m by 17 m. The frequency of resistance to the remaining 9 isolates was low and of a similar order in both groundsel populations. However, the tendency for more plants to have resistance at N.V.R.S. was significant.

Ten different resistance phenotypes were detected in groundsel plants from Glasgow, and 24 from N.V.R.S., 9 of which were common to both populations and included the phenotype susceptible to all isolates. This indicates that patterns of race specific resistance were essentially similar in each population, but there was greater variation at N.V.R.S. Partial resistance was more common at N.V.R.S. and natural frequencies of mildew infection were lower at N.V.R.S., possibly, the defence strategy was more effective here than at Glasgow.

The groundsel population was highly heterogeneous, 10 resistance phenotypes were detected, using 5 mildew isolates, in 75 plants from a 1 m by 1 m plot. This heterogeneity was reflected in the mildew population. Of 24 isolates characterized on 50 inbred groundsel lines, 18 were found to be different. A minimum of 14 avirulence genes could explain the observed reactions of the 18 races. The races were complex, having several virulence genes and usually only one avirulence gene, but races with up to 5 avirulence genes were also detected.

Studies of partial resistance began to reveal the complexity of the defence strategy deployed by groundsel. Partial resistance was very common in groundsel and could be race specific in some instances whilst non race-specific in others. Some individuals possessed both complete and partial resistance. Perhaps crop plant breeding programmes should aim to include both forms of resistance within a cultivar.

Plant age had a significant effect on all forms of resistance. Incubation temperatures could alter the susceptibility of some plant lines to some isolates. The effects of plant age and temperature could be considered race specific in nature, since some plant lines switched from susceptible to resistant under one set of conditions to a particular isolate, whilst under another set of conditions, and with another isolate, resistance switched to susceptibility.

An investigation of sporeling development, revealed that Erysiphe fischeri conidia commonly form several germ tubes on susceptible host tissue. Each germ tube has the potential to form an appressorium and initiate infection. Resistance was expressed at the haustorial and secondary hyphal stages of development.

CHAPTER 1. GENERAL INTRODUCTION.

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1.0. GENERAL INTRODUCTION.

A substantial proportion of the worlds food supply is lost each year as a result of crop disease caused by pathogens. The economic cost of such losses is difficult to assess, but when a nations food supply is severly affected the result is famine and starvation. An epidemic of Helminthosporium oryzae (Cochliobolus miyabeanus) in rice in 1942 resulted in the starvation of around 2 million people in Bengal the following year (Padmanabhan, 1973).

Resistance provides a promising form of crop protection, being more economical and environmentally acceptable than chemical control. At the turn of the century Biffen (1905) produced his classic paper showing that the resistance of wheat to yellow rust (Puccinia striiformis) was controlled by Mendelian genes. There were great hopes that the exploitation of this natural form of crop protection would be lasting and effective. Early workers did not believe that pathogens, notably the rusts, could adapt quickly enough to overcome the resistance of their new cultivars (Biffen, 1912 , Stakman et al, 1918). Unfortunately, they were to be proved wrong. This form of resistance which tended to be inherited by a single dominant gene of large effect, often only provided temporary protection. It appears that as a consequence of its comparative genetic simplicity, this form of resistance may be readily overcome by relatively small evolutionary steps in the pathogen (Fraser, 1985). As cultivars with resistance gained popularity and were extensively used, selection for virulent forms or races of the pathogen capable of colonizing these cultivars was great, leading to what is commonly known as the 'boom-and-bust-cycle'. Borlaug (1965) estimated that the average rust resistant cultivar of wheat lasted about four years in Mexico.

In many cases, genes for resistance have either been isolated directly from the wild relatives of crops or from existing crop varieties which presumably originated from the wild at some point in their ancestry. Yet there is little understanding of role of disease in the ecology of wild plant populations (Burdon, 1982a , Harlan, 1976), let alone the importance and function of disease resistance within a natural pathosystem. It is hardly suprising that disease resistance has often proved to be a disappointment as a control measure when so little is known about its function in the natural situation. The bulk of our knowledge originates from studies of various forms of resistance in crops in a man-made environment. The use of cultivar mixtures and multilines has sparked an interest in the study of the role of disease resistance in wild populations. It has been postulated that mixtures and multilines will provide a more durable form of resistance because they may resemble more closely the strategy employed by wild host populations (Burdon, 1978 , Browning, 1974). Yet we can only postulate as to the nature of the wild defence strategy from our knowledge of the artificial crop situation (Robinson, 1982b). The present study of the Senecio vulgaris/ Erysiphe fischeri plant pathosystem was undertaken in the hope that it would provide information that can be utilized in the breeding and management of crop systems.

1.1. Effects of pathogens on wild plant communities.

Pathogens by definition are generally assumed to cause some extent of damage to their hosts. Pathogens of crop plants tend to reduce marketable yield which has stimulated the study of the effects of various pathogens on factors such as plant growth and population size. Equivalent studies on the effects of pathogens on wild populations have rarely been studied and their importance as a factor in selection has

often tended to be overlooked (Harper, 1977).

Observations of wild host plant populations indicate that the host population tends to suffer relatively little damage as a result of colonization by pathogens (Harper, 1977 , Segal et al, 1980 , Dinoor and Eshed, 1984 , Burdon, 1982a). This suggests that efficient disease defence strategies have evolved in these pathosystems. Hypotheses on the mechanisms by which damage limitation is brought about are numerous but they have rarely been tested in practise.

The diversity of natural populations, both inter and intra specifically is postulated to reduce epidemic spread and is one of the reasons why it is hoped that resistance may be more effectively utilized by developing cultivar mixtures and multi-lines (Burdon, 1978).

It is not always the case that wild populations are left unscathed by epidemics. Kranz (1968a) found that the severity and progress of an epidemic in natural populations depends on the particular host and parasite associations concerned. Kranz (1968b) made a study of 59 different fungal parasite-host pairs. Disease progress curves tended to follow one of three different patterns.

A). The fungus completed its disease progress curve and the host survived.

B). The parasite caused sufficient damage to result in the disappearance of the host population.

C). The disease progress curve was halted by the disappearance of the host due to natural senescence, eg as with many annuals.

There are accounts of pathogens which do cause devastation in the wild. For example, Phytophthora cinnamomi the causual agent of die back in Eucalyptus spp. causes severe damage in some parts of Australia. In other areas, die back is seldom a problem (Blowes et al. 1982, Halshall, 1982). Where there have been serious epidemics of

die back it has been postulated (Pratt and Heather, 1973) that this may have been due to a recent disturbance in the environment causing an imbalance in the pathosystem and resulting in an epidemic. One may expect that such a potentially aggressive organism is in danger of eliminating its host and consequently itself. However, it has a wide host range including hosts found in association with Eucalyptus spp and this probably ensures its survival.

Pathogens recently introduced into an area may produce severe epidemics in wild host communities. Endothia parasitica, the causual organism of Chestnut blight, was introduced to North America in 1904, and within 50 years it had swept across America (Van Alfen, 1982). The American Chestnut (Castanea dentata) now survives as sprouts developing from infected tree stumps, with few mature trees remaining. In Europe, Chestnut blight also posed a severe threat to the sweet chestnut population, but within fifteen years of its introduction it was noticed that some trees were surviving attack. It is thought that either a virus infects the parasite thus restricting its aggressiveness or that a more competitive but less aggressive form of the pathogen has developed (Van Alfen, 1982). The development of a less aggressive strain of the pathogen would ensure the survival of the host and in turn the pathogen.

Few true examples of reduced pathogenicity have been observed in pathogens of plants, however, a classic example has been observed in the animal kingdom. Myxomatosis of rabbits originally devastated the Australian rabbit population. However, in due course, less aggressive strains of the virus became dominant in the population, probably because they were more readily transmitted by mosquitoes. Resistance in the rabbit population also started to increase and the situation is now moving towards an endemic balance (Harlan, 1976 , Marshall and Fenner, 1985 , May and Anderson, 1983).

1.2. Defence strategies.

Defence strategies identified in crop pathosystems include disease escape (or escape resistance), tolerance and various forms of resistance.

1.2.1. Disease escape.

Disease escape or escape resistance (Parlevliet, 1977) can be considered to be a passive form of resistance and the character that results in escape may or may not necessarily have this as a primary function. For example, Burdon (1982a) in studies with oat crown rust (Puccinia coronata avenae) showed that the more susceptible host Avena fatua developed earlier than the less susceptible A.barbata avoiding the impact of the disease. This situation could be merely fortuitous or could result from selection imposed by the rust fungus. It has also been postulated that the evolution of the annual and deciduous growth habits may not have evolved solely in response to unfavourable growing conditions, but may also serve to cause a break in the availability of host material for pathogens thus checking an epidemic. The pathogen would have to respond by evolving a method of colonising an alternative host for this period or developing a survival mechanism to maintain itself over this period (Price, 1980). The occurrence and importance of disease escape may have been underestimated in many pathosystems. It is obviously important that characteristics of the plant that confer disease escape are not accidentally bred out of a crop variety.

1.2.2. Tolerance.

Tolerance is difficult to define (Schafer, 1971 , Clarke, 1986) and this has often led to its confusion with disease escape and forms of resistance, especially partial resistance. There may be tolerance to

the pathogen itself or tolerance to the effects of the disease the pathogen causes. In this context, tolerance is said to occur where two individuals are equally colonized by a pathogen but one individual is less affected in terms of growth and/ or reproductive capacity. In fact, most accounts of tolerance in the literature are subjective. Studies on tolerance to rust fungi in cereal crops have perhaps been the most extensive. For example, Kramer et al (1980) worked with 15 cultivars of barley but found that only two cultivars showed evidence of tolerance to Puccinia hordei but that these tended to be the lower yielding varieties with a low harvest index. This implies that a lower proportion of assimilates is directed towards the ear so that when a tolerant plant becomes infected the pathogen utilizes assimilates not necessarily directed towards grain production, thus grain production is buffered from the effects of the rust.

There is good observational evidence (Tarr,1972) and experimental evidence (Ben-Kalio and Clarke 1979) that tolerance may play an important part in the survival strategy of wild plants to parasite attack.

1.2.3. Resistance.

The term resistance may be applied where a plant allows no infection, reduced infection, does not allow reproduction or reduces the rate of reproduction of the pathogen. The majority of plant species are resistant to the majority of pathogen species ie they are non-hosts. Within a host species some potential hosts may show resistance to a pathogen normally capable of infecting that host species. This may be termed intra specific resistance. Where resistance is expressed to some strains or races of the pathogen but not others, this can be called race specific resistance (Parlevliet, 1981). Where resistance is expressed to the total pathogen population

this can be called race non-specific resistance. Race specific resistance in crop pathosystems tends to be associated with clear cut reactions of resistance or susceptibility and tends to be controlled by single genes. Resistance of this type is easy to observe and its simple inheritance has meant that it is the most commonly used form of resistance in crop breeding (Robinson, 1982b). Unfortunately, race specific resistance has so often proved to be highly successful initially, only for the pathogen population to quickly render the resistance ineffective.

Race non-specific resistance is often less complete than race specific resistance. It is often, but not necessarily, thought to be under polygenic control and is said to be more durable, possibly because its relative complexity makes the chances of all mutations required for virulence less likely within a pathogen isolate (Fraser, 1985).

1.3. Resistance in wild pathosystems.

Race specific resistance has been demonstrated in some wild pathosystems. Heather and Chandrashekar (1982a) found race specific interactions between isolates of Melampsora larici-populina and M.medusae and collections of Populus spp. Blowes et al (1982) also found indications of race specific resistance in the Phytophthora cinnamoni/ Eucalyptus spp pathosystem.

In many cases race specific resistance has been identified in the search for novel resistance for use in crop plants. For instance, Crute et al (1980) found race specific resistance to lettuce downy mildew (Bremia lactucae) in a wild relative Lactuca serriola of cultivated lettuce (Lactuca sativa). Lebeda (1984) also identified race specific resistance in wild relatives (Cucumis spp) of the cultivated cucumber to powdery mildew fungi (Erysiphe cichoracearum and Sphaerotheca fuliginea).

Dinoor (1977) demonstrated the presence of race specific resistance in the wild oat/crown rust pathosystem (Avena sterilis and A.barbata/ Puccinia coronata avenae). In race surveys of P.coronata avenae in various localities in Israel over a period of three years he detected 91 different physiological races of the pathogen, indicating that there was a tremendous amount of variability in the pathosystem. Work in Israel, by Segal et al (1980) in the search for resistance in barley to powdery mildew Erysiphe graminis hordei detected the presence of race specific resistance in wild barley (Hordeum spontaneum) to the same powdery mildew.

The frequency of H.spontaneum plants in several natural populations, showing various levels of infection to E. graminis was also reviewed by Segal et al (1980) and compared with the frequency of infection types produced by Puccinia coronata avenae on natural

stands of Avena sterilis. High infection types predominated in the A. sterilis populations in comparison with those found in the H. spontaneum populations. Thus, differences in both the levels of resistance and the frequency of race specific resistance were identified in the different wild plant pathosystems depending on the pathogen and host involved in the association.

There have been very few attempts to actually map the distribution of various resistance phenotypes in wild plant populations. Burdon (1980a,b) studied a natural population of Trifolium repens. The 50 plant clones studied showed considerable variability in their resistance to two foliar pathogens Cymadothea trifolii and Pseudopeziza trifolii. However, only one isolate of each of the pathogens was used so that race specificity could not be examined.

Dinoor (1970) used two isolates of Puccinia coronata avenae to survey race specific resistance in Avena barbata and A. sterilis in various locations throughout Israel. It was found that approximately 7.5% of the A. sterilis plants were resistant to one or other of both the rust isolates. Different locations revealed different frequencies of resistant plants, indicating diversification between populations. The differences between the populations were thought to be a reflection of the different environmental conditions of north and south Israel.

Disease may also be the cause of diversity. Burdon and Chilvers (1974) in a study of pathogens of Eucalyptus spp. suggested that even where saplings were not badly affected by insects and fungi if 20% of the leaf surface was damaged, this was enough to reduce plant stand and hence lead to a diversity of species. Variation for race specific resistance may be present in plant populations but the question as to whether or not it has occurred as a result of selection by pathogens

remains unanswered.

The importance of partial resistance in host defence has sometimes been overlooked in both crop and natural populations. Partial resistance is recognised when infection of a host is significantly reduced in comparison to a highly susceptible host although colonization by the pathogen is not completely halted as in a completely resistant response. There has been a tendency to consider partial resistance to be race non-specific but this need not necessarily be the case. Partial resistance that is race specific can also occur. Partial resistance was commonly found by Burdon and Marshall (1981) in four native species of Glycine to the leaf rust fungus Phakopsora pachyrhizi. Wahl (1970) also reported that partial resistance to Puccinia coronata avenae was common in wild populations of Avena sterilis. Variation in susceptibility of the cruciferous weeds, shepherds purse (Capsella bursa-pastoris) and hedge mustard (Sisymbrium officinale) to club root (Plasmodiophora brassicae) was studied by Buczacki and Ockendon (1979). Race specific resistance was identified in shepherds purse and there was evidence that hedge mustard showed various levels of partial resistance.

1.4. Reasons for studying the Senecio vulgaris/Erysiphe fischeri wild plant pathosystem.

Senecio vulgaris is a common annual with a short life-cycle, therefore, it is easy to collect and many experiments can be completed in a relatively short period. It tends to inbreed (Haskell, 1953) so that progeny from the same plant are likely to be of similar genotype. Inbreeding would also tend to cause divergence (Loveless and Hamerick, 1984) between populations of groundsel thereby increasing the chances of finding differences in the resistance phenotypes between populations.

Erysiphe fischeri is an obligate pathogen frequently found colonizing groundsel in late summer. Isolates of the pathogen are, therefore, relatively easy to obtain. Groundsel powdery mildew is unknown on crop plants, and so its race structure is unlikely to be affected by pathogen races that build up on crop populations, which in turn could affect the survival strategy in the wild host population. Studies of resistance in wild populations in the past have generally involved resistance to pathogens that also colonize crop plants and, therefore, may have revealed artifacts that would not normally occur in natural pathosystems without the interference of man's activities. Although the pathogen is relatively common on groundsel it rarely kills its host or devastates its host population. This indicates that an efficient defence strategy may have evolved in the host which could provide information of value in the efficient use of resistance in crops.

Work has already been carried out on the pathosystem enabling existing information and techniques to be utilized. Ben-Kalio (1976), and Ben-Kalio and Clarke (1979) found evidence of tolerance to powdery mildew in groundsel. This was also supported by the work of Harry (1980). Dry matter production in groundsel showed no signs of reduction until there was a 30% leaf cover by mildew. The distribution of dry matter was not significantly affected until 90% of the above ground parts of the plant were covered with mildew. Roots, stems and leaves were equally affected. In contrast Last (1962) found that barley leaves rarely survived infections where more than 50% of their area was affected by Erysiphe graminis. Root growth in barley was severly affected when 30% of the aerial parts of the plant were colonized with barley powdery mildew.

Groundsel heavily colonized by mildew remains capable of setting viable seed. Harry (1980) found that eight week-old groundsel plants

with 75-100% of their leaf area affected by powdery mildew produced half as many flowers as uninfected, benomyl treated controls. Once the plants were 9-10 weeks old, infected plants produced a third of the flowers of the healthy controls. Although the groundsel set seed the reduction in seed output was obviously sufficient to produce genetic feedback if some individuals were more affected than others. Susceptibility and resistance would then play an important role in determining the frequency of various phenotypes in groundsel populations.

Race specific resistance was demonstrated in the pathosystem by Harry (1980), and Harry and Clarke (1986). The responses of approximately 250 groundsel lines to 8 mildew isolates indicated a 'gene-for-gene' type of relationship (Flor, 1956 , Person, 1959). The results also indicated differences in the distribution of resistance phenotypes between groundsel populations at different sampling sites. However, the numbers of plants tested from each location were low and the isolates used were not selected randomly so it was difficult to make comparisons between the groundsel populations.

1.5. The aims of the present project.

The main aims of the project were to:

1. Confirm the presence of race specific resistance in the *Erysiphe fischeri/ Senecio vulgaris* pathosystem.
2. Determine the number, frequency and spatial distribution of various race specific resistance phenotypes in two geographically distant groundsel populations and determine how similar these two poulations were.
3. Determine the virulence characteristics of a range of isolates from two distant locations.

The knowledge gained could be of value for the planning of plant breeding and crop management programmes.

CHAPTER 2. GENERAL MATERIALS AND METHODS.

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2.0. GENERAL MATERIALS AND METHODS.

2.1. The production of mildew free plants at Glasgow.

Mildew free groundsel was successfully produced in growth rooms at Glasgow. The growth rooms were maintained at 18°C to 21°C, with a 16 h photoperiod. Illumination was provided by daylight flourescent tubes, giving a photon flux density of approximately 610 μmol m^{-2} s^{-1}. Groundsel seeds were sown in S.A.I. potting compost in 9 cm plastic pots; the seedlings were individually transplanted to 12 cm pots about three weeks after sowing. At six to seven weeks old the plants were usually in flower and producing ample leaf tissue for the testing and maintenance of mildew isolates.

2.2. The production of mildew free plants at the N.V.R.S.

At N.V.R.S., groundsel was sown and transplanted in the same way as at Glasgow. However, the plants were grown in several smaller cabinets, (See Plate 1.). Temperatures were maintained at 18°C ± 1°C. A 16 h photoperiod was provided by high pressure SOM/R sodium lamps giving an intensity of approximately 320 μmol m^{-2} s^{-1}. On two occasions mildew was detected at a low level in one of the cabinets. The single plant infected in each case was discarded together with the remaining plants in each cabinet. On each occasion this action eliminated the mildew infection.

2.3. The mildew isolates.

A collection of isolates of *Erysiphe fischeri* was made in Glasgow in 1983 and at the National Vegetable Research Station, Wellesbourne (N.V.R.S.) in 1984. Twelve isolates from each location were obtained. Infected plants from different areas within the two locations were

PLATE 1. Mildew free cabinets at N.V.R.S.

PLATE 2. Incubation room at N.V.R.S.

randomly sampled and maintained separately from one another by enclosing them individually in polythene bags. Conidia from each of these plants were dusted directly on to mildew free leaf segments of several groundsel lines in 9 cm Petri dishes on benzimidazole supplemented agar. The dishes were incubated at $15^{o}C \pm 1^{o}C$ at the N.V.R.S and $20^{o}C \pm 2^{o}C$ at GLasgow under lights providing a 12 h photoperiod and an intensity of approximately 100 μmol m^{-2} s^{-1}. Once a distinct colony was formed, single chains of conidia were picked off the colony, with the aid of a dissecting microscope, using a fine steel sewing needle. Each chain was then gently placed on a separate mildew free leaf segment and incubated as before. This was repeated at least twice to ensure that the resulting colony was the product of a single chain of conidia. Several chains of conidia were taken from the mildew from each plant and transferred to several different leaf segments to ensure that at least one colony of each isolate was successfully produced. At the end of the process a resulting colony was considered to be a pure isolate originating from a single conidial chain. Each isolate was multiplied up using mass conidial inoculations onto fresh leaf segments until enough inoculum was obtained for testing the isolates on several plant lines. Several plant lines were used for the 'bulking- up' of isolates to ensure that isolates did not become adapted to one particular line, and to ensure that no isolates were lost because they were cultured on unsuitable lines.

2.4. Inoculations.

Several methods of inoculation were attempted; in all cases inoculum was deposited on the upper (adaxial) surface of the leaf segments. The methods included:

1. Using a small paint brush to transfer conidia from a

sporulating colony to the leaf segment to be tested.

2. A steel needle was gently dabbed in a sporulating colony and the conidia gently deposited on the leaf segment to be tested.

3. A small piece of leaf was held with tweezers and dabbed in a colony and then gently dabbed onto the fresh leaf segment.

4. The leaf segment producing the colony was cut into small pieces if the colony was large, and held with tweezers and gently stroked across the leaf segments to be inoculated.

It was found that method 4., otherwise known as the 'direct dusting method' produced the most consistent reactions.

2.5. Incubation period.

Isolates C2, D7, G2, and P1 were used to determine the optimum period of incubation. Leaf segments from several susceptible plant lines were inoculated and examined daily until no further colonies developed. The number of leaf segments that produced mildew colonies each day are given in Table 2.1. It was found that with an incubation temperature between 15°C and 20°C, most of the segments started to produce visible colonies 5 days after inoculation. By the 10th day after inoculation all plant line/isolate tests giving susceptible reactions had produced colonies. The results indicated that the optimum time for assessment of infection types was 9 days, thus ensuring that susceptible reactions had adequate time to be expressed but limiting the time for the development of colonies that were a result of secondary infection.

2.6. Maintenance of mildew isolates in Glasgow.

Erysiphe fischeri is an obligate parasite and has to be maintained on host tissue. At Glasgow, mildew isolates were maintained on leaf segments on 0.5% agar supplemented with 30 ppm benzimidazole in 9 cm

Table 2.1 Mean % of susceptible leaf segments colonized with mildew

Days after inoculation	Isolate							
	C2		D7		G2		P1	
	Mean	Se	Mean	Se	Mean	Se	Mean	Se
1	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
2	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
3	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
4	2.8	2.78	0.0	0.00	0.0	0.00	0.0	0.00
5	2.8	2.78	2.6	1.35	2.0	2.02	0.0	0.00
6	9.7	6.45	13.7	4.90	4.0	4.04	18.5	0.33
7	18.1	8.89	26.5	7.29	25.3	8.08	32.1	11.48
8	20.8	10.80	33.3	7.55	34.3	9.41	46.9	11.33
9	25.0	10.01	35.9	7.02	-	-	59.3	9.91
10	25.0	10.01	-	-	43.4	10.10	69.1	10.96
11	-	-	39.3	7.29	49.5	9.16	-	-
12	27.8	10.07	59.0	6.08	57.6	8.71	80.3	10.96
13	29.2	9.61	-	-	58.6	8.62	80.3	10.96
14	29.2	9.61	-	-	59.6	9.02	-	-
15	29.2	9.61	-	-	61.6	9.16	-	-
16	31.9	9.02	-	-	-	-	-	-

- = Not examined

diameter clear plastic Petri dishes. This mediumwas used in all investigations using detached leaves. After 10 to 14 days incubation at 18°C to 20°C in a 12 h photoperiod, the Petri dishes were transferred to a cold room of approximately 5°C that was in continual darkness. In this way subculturing was necessary every five to six weeks.

2.7. Maintenance of mildew isolates at N.V.R.S.

Mildew isolates that were in continual use were maintained on leaf segments from mildew free plants as above. Incubation temperatures for the initial production of the colonies was lower being 15°C ±1°C with a photoperiod of 12 h (Plate 2.). After 10 to 14 days incubation, dishes were then transferred to a cold room held at a temperature of approximately 5°C and light for about 8 h each day.

Isolates that were not continually in use were successfully maintained on whole plants in an Isolation Plant Propagator (Burkard Manufacturing Co Ltd; Jenkyn et al, 1973) (Plate 3.). Seedlings of groundsel lines found to be susceptible to all isolates were grown in mildew free cabinets and then transplanted into the Isolation Plant Propagator when they were about three weeks old. Plants were inoculated with the respective isolates a week or so later. The Isolation Plant Propagator was situated in a temperature controlled glasshouse and maintained at about 15°C without supplementary lighting. Mildew isolates were maintained in this way for many weeks until the plant reached the end of its natural life span. Over winter when plants were growing slowly, isolates were maintained for up to four months in this way without subculturing.

PLATE 3. Isolation plant propagator.

PLATE 4. Mildew infection types

2.8. The source of the 50 inbred plant lines used to differentiate and characterize mildew isolates.

Fifty inbred groundsel plant lines used to characterize the mildew isolates were originally obtained by Harry (1980). The original seed were collected from several locations in the British Isles (See Fig 2.1 and Table 2.2.). Since all the lines had been maintained for many generations by inbreeding they were assumed to be true-breeding for resistance phenotype.

2.9 Designation of infection types.

Infection types were designated in all the experiments involving detached leaves based on the key developed by Harry (1980). This key was itself derived from a scoring system used by Moseman (1956) to categorise the reactions of barley to Erysiphe graminis hordei. Harry's key was as follows:

Infection type 0 = No germination was observed.
Infection type 1 = Slight mycelial development but no conidia were produced.
Infection type 2 = Moderate development of mycelium but with the production of very few conidia. Some necrosis or chlorosis was usually present.
Infection type 3 = Moderate to abundant development of mycelium, accompanied by moderate sporulation with some necrosis.
Infection type 4 = Extensive colony formation with abundant sporulation and no necrosis or chlorosis.
Infection types 0 and 1 were considered as resistant and types 2, 3 and 4 susceptible.

The scoring system used in this study was an adaptation of that used by Harry (1980). It was found that it was necessary to divide

Fig 2.1

Origin of the inbred groundsel lines.

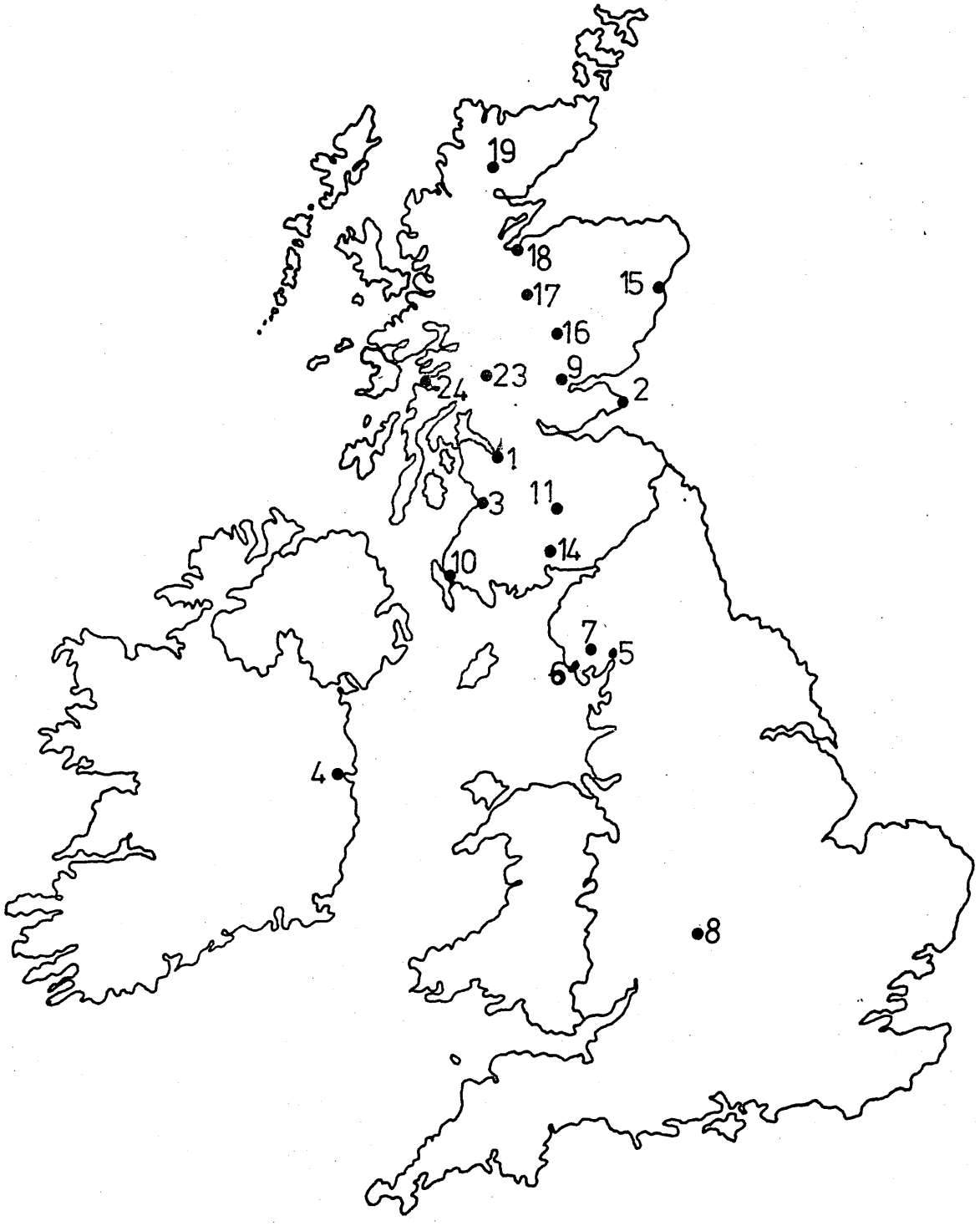


Table 2.2. The origin of the inbred groundsel lines collected by Harry (1980).

Origin	Line	Origin	Line
Glasgow	1c	Wellesbourne	8a
	1e		8g
	1f		
	1g	Perth	9a
	1h		9c
	1i		9d
	1m		9g
	1n		
	1p	Stranraer	10j
	1s		
Crail	2a	Abington	11a
	2d		11e
	2e		11i
	2i		
		Dumfries	14b
Ayr	3f		
	3g	Aberdeen	15c
			15j
Dublin	4a		
	4h	Pitlochry	16d
			16f
Far Sawrey	5a		
		Kingussie	17h
Ulverston	6b		
	6d	Inverness	18i
	6f		
		Lairg	19b
Coniston	7a		
	7b	Crianlarich	23f
	7c		23g
	7d		23i
	7f		
		Oban	24f
			24j

infection type 2 into two categories: 2- and 2+. This was because in many cases small colonies were observed producing very few conidia but were only visible with the aid of a dissecting microscope. These reactions were designated as infection type 2-. Infection type 2+ was assigned to reactions where the colonies were visible with the naked eye and produced a moderate number of conidia.

The infection types used in this study were as follows:

Infection type 0 = No mycelium or conidia produced.
Infection type 1 = Sparse mycelium produced but no conidia.
Infection type 2-= Mycelium produced a few conidia, but colony was only discernible with the aid of a dissecting microscope.
Infection type 2+= More abundant sporulation, colony visible with the naked eye.
Infection type 3 = Abundant sporulation giving rise to a moderately sized colony.
Infection type 4 = Very abundant sporulation and extensive colony produced.

Plate 4. shows examples of leaf segments with infection types 0, 2+, 3 and 4

2.10. Transformation of infection types to infection scores.

For the purposes of analysis the infection types 0, 1, 2-, 2+, 3 and 4 were transformed to numerical infection scores 0, 1, 1.5, 2.5, 3 and 4 respectively. Scores 1.5 and 2.5 were chosen to represent types 2- and 2+ since reactions giving a 2- type colony produced very few conidia and the reaction was considered to be closer to the resistant type reactions than the susceptible type reactions. Infection type 2+ colonies, on the other hand, produced quite abundant conidia estimated in the hundreds and capable of producing more substantial infections

than 2-, therefore being more similar to reaction type 3 than to type 2-.

2.11. General testing procedures using detached leaves.

Mildew free plants were grown at N.V.R.S and Glasgow as described earlier. The plants were then tested for resistance to particular isolates using the detached leaf method developed by Harry (1980). Wherever possible, fully expanded but not senescent leaves were cut from the sixth node upwards from the mildew free plants to be tested. Immature leaves were avoided. For each test, four leaf segments from each plant line for each isolate were placed in each of two 9cm plastic Petri dishes on 0.5% agar supplemented with 30ppm benzimidazole. Three leaf segments in each dish were dusted directly with conidia from a culture of the isolate to be tested and the other leaf segment was marked by clipping it for identification and left uninoculated. The uninoculated segment acted as a control to check for possible cross-contamination with other isolates and to determine whether any symptoms of necrosis or chlorosis on the inoculated leaves were due to mildew infection. Thus, for each compatibility test, six leaf segments originating from two to four leaves from a single plant were inoculated. The Petri dishes were then incubated for 9 days. At Glasgow the Petri dishes were incubated in an incubation room at $18^{o}C \pm 2^{o}C$ with a 12 h photoperiod provided by strip daylight flourescent tubes providing an intensity of approximately $120 \mu mol^{-2}\ s^{-1}$. At N.V.R.S. the Petri dishes were incubated in an incubation room at $15^{o}C \pm 1^{o}C$ with a 12 h photoperiod produced by warm white flourescent tubes with an intensity of approximately $100 \mu mol\ m^{-2}\ s^{-1}$. After 9 days of incubation the infection types produced were recorded. Where all or most of the reactions were resistant (0 or 1) the test was repeated using the same plant about two weeks later.

CHAPTER 3. EFFECTS OF ENVIRONMENTAL FACTORS ON SUSCEPTIBILITY.

CONTENTS.

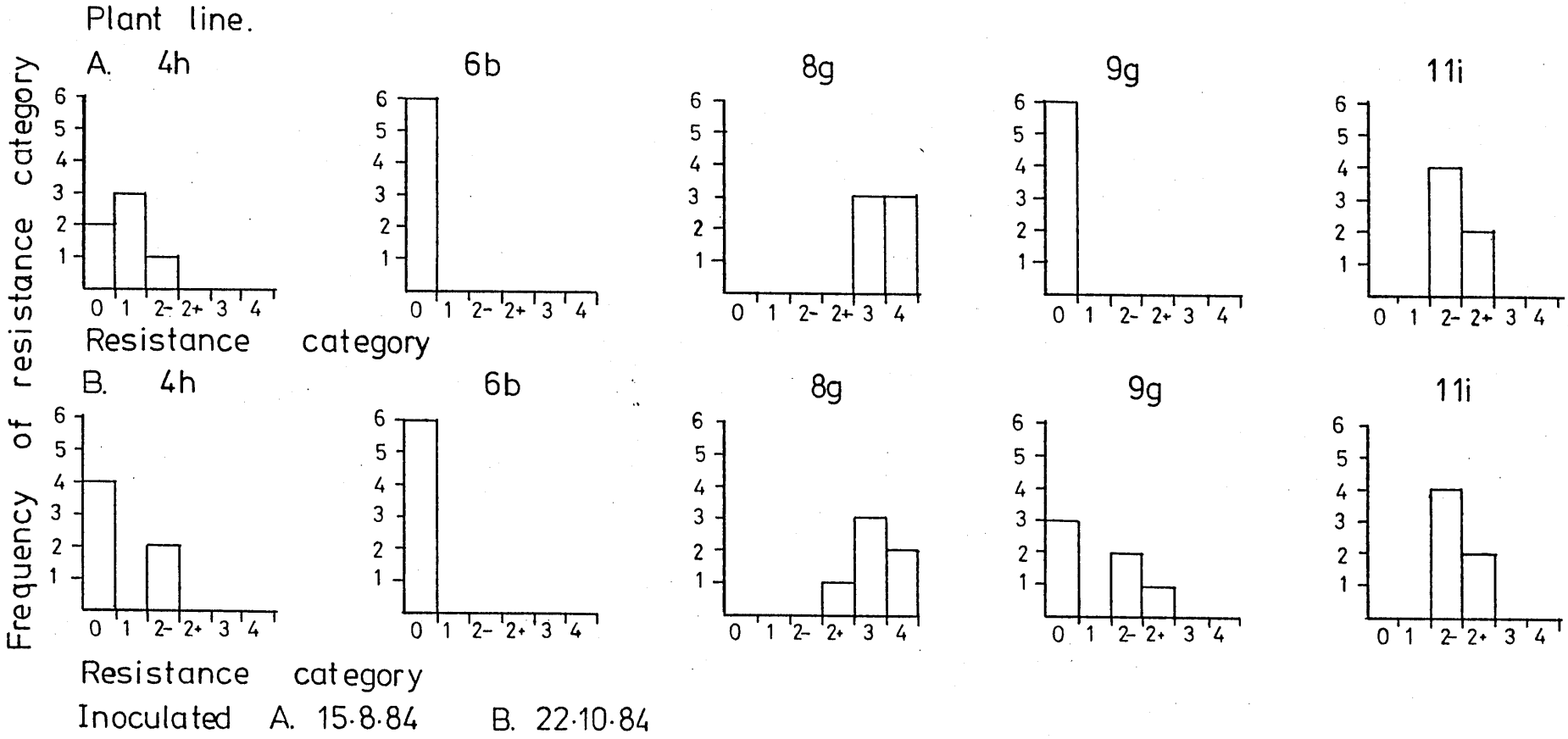
3.0. Effects of environmental factors on susceptibility.

3.1 Introduction.

Preliminary observations during the early stages of the work indicated that some groundsel line/isolate interactions produced variable infection types on different testing dates and to a lesser extent between leaf segments tested on the same date. The histograms presented in Figs 3.1 to 3.5 represent the infection types obtained for 5 groundsel lines (4h, 6b, 8g, 9g and 11i) when they were inoculated with 5 mildew isolates (G8, G9, G10, G11 and G12) on two separate occasions. It can be seen from the histograms that every leaf segment of plant line 6b tested was completely resistant to all 5 isolates. The infection scores obtained for line 8g showed some degree of variation, but generally its infection scores were fairly high making it easy to categorise as highly susceptible. However, plant lines such as 11i showed highly variable results, especially between testing dates. Tests made on the 13.8.84 showed that it was completely resistant to isolate G10 but tests on the 22.10.84 indicated a low level of susceptibility to that isolate. The infection types obtained with line 9g were also difficult to categorise as it tended to show a low but variable level of susceptibility to many of the mildew isolates.

This variation between tests made some plant line/isolate combinations difficult to classify. Experiments were carried out to determine whether any environmental factors were responsible for the variation that could be controlled in future experiments. The following are probably the more important sources of variation.

Isolate G8.- VARIABILITY OF INFECTION TYPE Fig 3·1

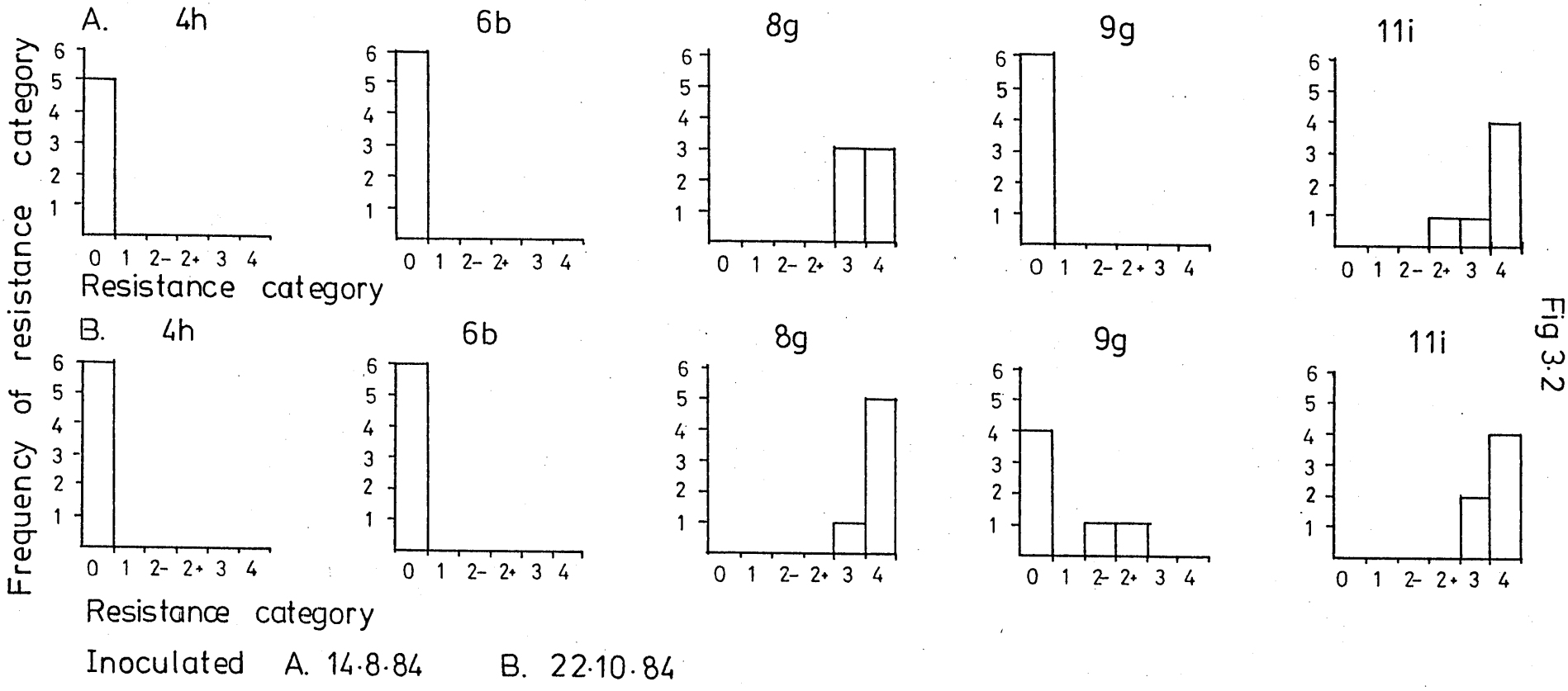


Inoculated A. 15·8·84 B. 22·10·84

Fig 3.1

Isolate G9. - VARIABILITY OF INFECTION TYPE

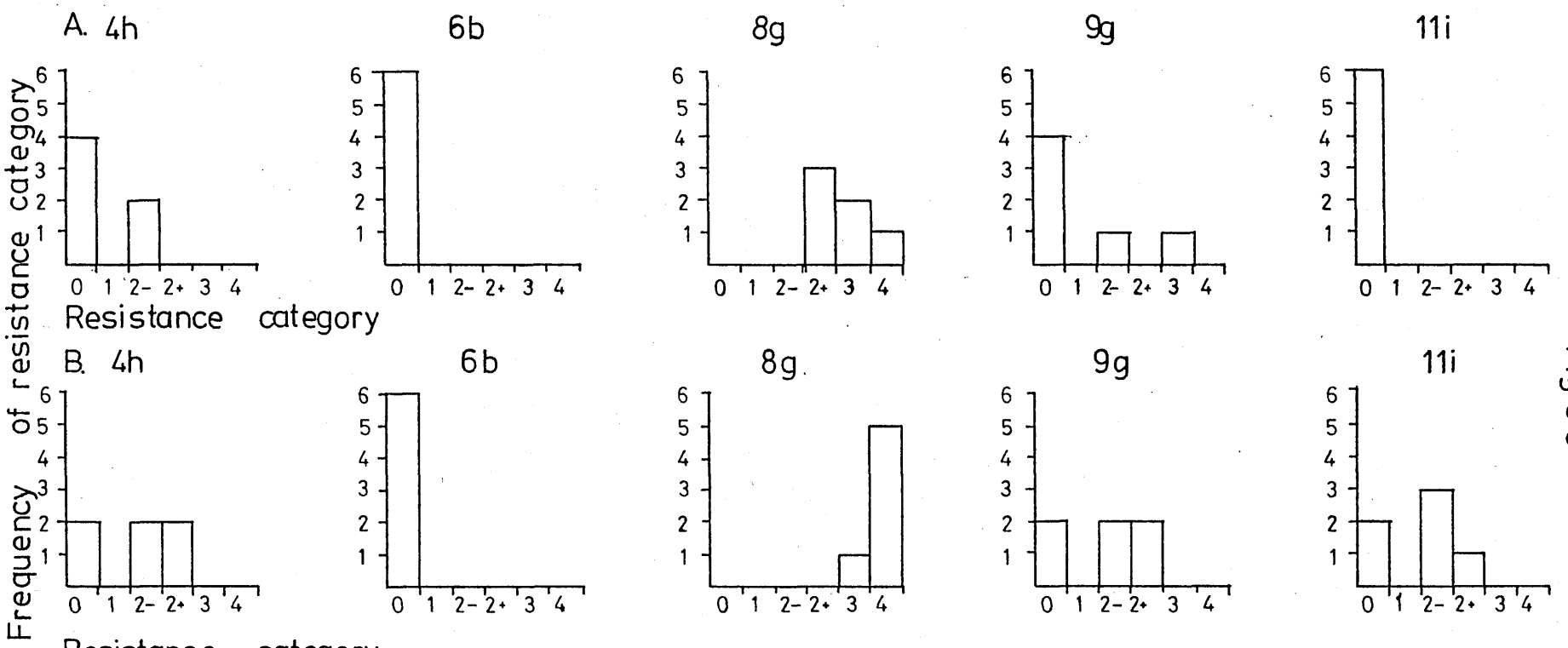
Fig 3.2



Inoculated A. 14·8·84 B. 22·10·84

Fig 3.2

Isolate G10 - VARIABILITY OF INFECTION TYPE. Fig 3·3



Inoculated A. 13·8·84. and B. 22·10·84

Fig 3·3

Isolate G11 - VARIABILITY OF INFECTION TYPE Fig 3.4

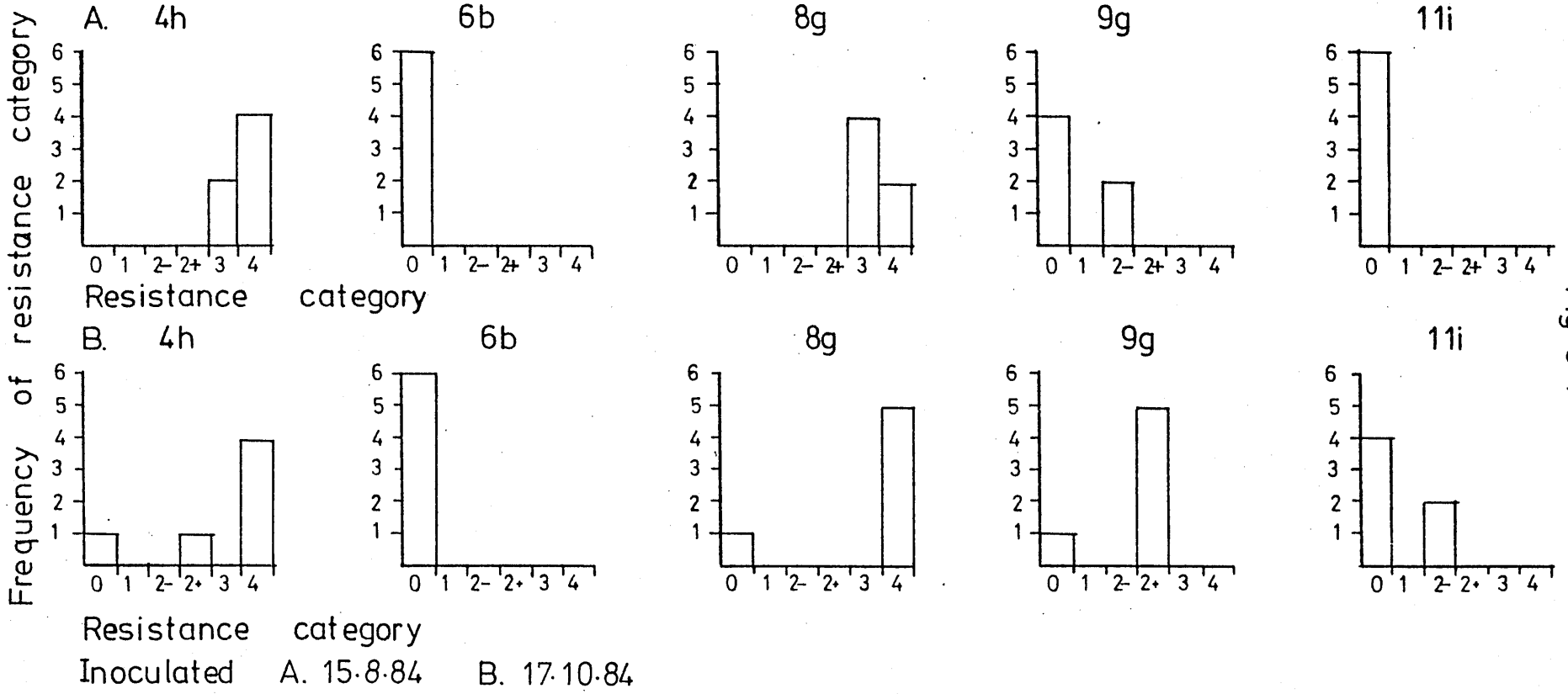
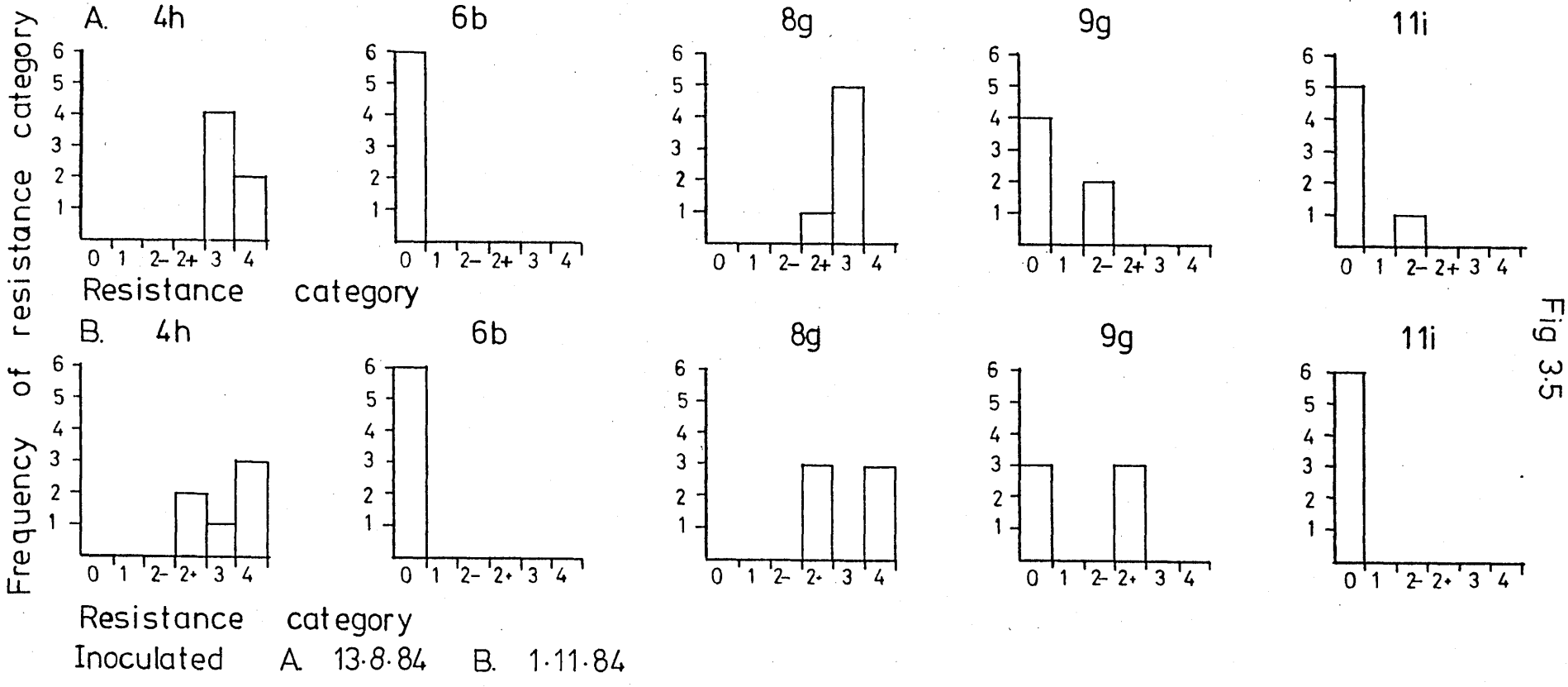


Fig 3.4

Isolate G12 - VARIABILITY OF INFECTION TYPE. Fig 3.5

Plant line.



Inoculated A. 13·8·84 B. 1·11·84

Fig 3.5

3.1.1. The conditions used to raise the plants.

The plants used for testing were raised in growth rooms where temperature and light regimes were controlled, and consequently it is unlikely that these factors were causing variation. It was thought possible that the spacing and size of pot used for plant raising could affect susceptibility. However, preliminary experiments investigating the effects of spacing and transplanting date suggested that these factors only affected susceptibility indirectly. For example, high sowing densities or delayed transplantation retarded the rate of development of the plants consequently making them less susceptible (See Section 3.2.2.). It was decided that for future experiments, all plants would be transplanted 3 weeks after sowing to individual plastic pots of at least 12 cm diameter.

3.1.2. The testing procedure.

The composition of the agar medium was always the same being 5g of agar per litre of water, with a supplementation of benzimidazole of 30mg per litre of water. Approximately 25 cm^3 of this medium was poured into 9 cm sterile plastic Petri dishes. Leaves were harvested between 9 am and 12 am and inoculated approximately 2-3 h later.

3.1.3. The inoculation procedure.

This involved dusting conidia from leaf segments supporting sporulating colonies directly on to the leaf segments to be tested. The number of conidia actually placed on the leaf segment could, therefore, vary considerably. The amount of inoculum is known to affect susceptibility in other pathosystems. For example, increased amounts of Puccinia coronata avenae inoculum on oats (Luke et al, 1972) and Puccinia hordei inoculum on barley (Clifford, 1974) produce increased amounts of rust infection. It is likely that the same

relationship applies to groundsel powdery mildew infection. In all tests the level of inoculum applied (100 or more conidia) was considered capable of producing a susceptible reaction on an appropriate host line. The conidia applied were just discernable with the naked eye as a white dust on the leaf segment surface; excessive amounts of conidia were gently removed.

The age of conidia and the age of colony from which conidia are obtained are known to be important factors related to the viability of Erysiphe graminis tritici on wheat (Nair and Ellingboe, 1962). There appeared to be no benefit obtained where the older conidia were blown from Erysiphe fischeri colonies 24 h before they were to be used for testing. This was probably because the excess of conidia used in the tests were adequate to produce susceptible infection types on susceptible lines.

Conidia used to conduct the tests were obtained from relatively young colonies about 10 to 12 days after initial inoculation. Older conidia were not removed from colonies prior to their use in testing. Leaf segments bearing older colonies tended to become contaminated with bacteria or fungi such as Botrytis spp. Dr B.L. Brady, of the Commonwealth Mycological Institute, identified Penicillium spinulosum on the agar and Trichoderma viride and Verticillium lamellicola were associated with the groundsel leaf pieces. Trichoderma viride is a known antagonist of several parasitic fungi such as Venturia inaequalis on apple (Andrews et al. 1983), Botrytis spp on strawberries (Tronsomo and Dennis, 1977) and Rhizoctonia solani (Martin et al, 1985., Papavizas and Lumsden, 1980). Penicillium spp are also known to be antagonistic to Botrytis cinerea on cyclamen (Blakeman and Fokkema, 1982) and to Verticillium spp on eggplant (Martin et al, 1985). However, it is unlikely that Penicillium spinulosum was responsible for any variation in levels of Erysiphe fischeri since it was rarely

found on the leaf segments. Although Verticillium lamellicola is not reported to be a hyperparasite of parasitic fungi it appeared to have a very close association with E. fischeri. Other species of Verticillium are known to be hyperparasites (Blakeman and Fokkema, 1982). For example, Verticillium lecanii is a parasite of scale insects and aphids. It is also reported to be a hyperparasite of fungi such as Puccinia graminis tritici (McKenzie and Hudson, 1976) and Uromyces dianthi on carnation (Spencer, 1980). It is highly likely that these contaminants found on the leaf segments contributed to the variability of the infection types of E. fischeri on groundsel but little could be done to eliminate them. It was considered that the contaminants were not responsible for inducing resistant reactions since all reaction types could be affected, including highly susceptible ones. Contaminants were kept to a minimum by regular subculturing.

3.1.4. Incubation conditions.

Inoculated segments were incubated for a constant period of 9 days; therefore, it is unlikely that the period of incubation was a source of variation. During the early stages of the work environmental factors such as light and temperature were not strictly controlled during the incubation period because facilities to do so were not available. Eventually the tests were transferred to growth rooms and it was then possible to determine how such factors affected susceptibility. Erysiphe fischeri is capable of germinating and colonising groundsel within the temperature range 6°C to 28°C (Ben-Kalio, 1976). Within this temperature range there must be an optimum for mildew growth which would in turn affect infection scores. There is also the possibility that temperature may affect the expression of resistance in groundsel. Temperature is known to affect

the expression of certain resistance genes in some crop pathosystems. Resistance may be expressed at higher temperatures but not at low temperatures. For example, resistance gene Dm6 in lettuce to Bremia lactucae was only expressed completely at 20°C (Crute and Norwood, 1978). Some cultivars of wheat such as Maris Fundin give an incompatible reaction at 20°C and a compatible reaction at 9°C to Puccinia recondita tritici (Hyde, 1982 , Dyck and Johnson, 1983). The reverse may also occur as in some cultivars of spring wheat to Erysiphe graminis (Futrell and Dickson, 1980) and in oats to Puccinia coronata avenae (Simons, 1954). Experiments were therefore carried out to determine whether some groundsel line/mildew isolate interactions were affected by temperature.

3.1.5. Plant age.

The ages of plants from which leaf segments were harvested varied between sampling dates, probably causing some variation. The effects of plant age on the expression of resistance to powdery mildews is well known in crop pathosystems. Some resistance is only expressed at the adult stage, for example, some cultivars of oat only express resistance to Erysiphe graminis avenae (Jones, 1975) at this stage. The same is true of some wheat cultivars in response to E. graminis tritici (Roberts and Caldwell, 1970 , Briggle and Scharen, 1961). In other cases resistance is only expressed in the seedling stage. Lettuce was generally found to be resistant to E.cichoracearum at the seedling stage but was susceptible at later growth stages (Schnathorst, 1959). Some cultivars of barley show seedling resistance but adult plant susceptibility to E.graminis hordei (Wright and Heale, 1984). Experiments were designed to examine the effect of plant age on susceptibility.

3.1.6. Leaf number or leaf age.

Occasionally it was not possible to use leaves from similar positions on different plants for testing as there were not enough. The age of leaf could have been a source of variation since it is known to be of importance in crop/powdery mildew pathosystems. For example, the lower leaves of lettuce are more susceptible to *E. cichoracearum* (Schnathorst, 1959). Likewise the lower leaves of oats are more susceptible to *E.graminis avenae* (Jones, 1975 and 1978). The older leaf cells of barley are reported to be more susceptible to *E.graminis hordei* (Lin and Edwards, 1974).

Preliminary experiments were carried out to assess the effects of incubation temperature, plant age and the relative developmental stage of leaf (leaf age or number). The experiments indicated that these factors may not only affect the general severity of mildew but they may also affect the resistance of some plant lines to particular isolates.

3.1.7. Experimental aims.

The first experiment described here was designed to determine in more detail the effect of plant age on infection type. The second experiment was a multi-factorial experiment using 5 plant lines and 4 isolates to determine whether there were any interactive effects of incubation temperature, plant age and leaf age on certain plant line/isolate combinations.

3.2 Experiment 1. The effect of plant age on susceptibility.

3.2.1. Materials and methods.

This experiment was carried out using the facilities at Glasgow. Plant lines 1c, 1e, 1f, 1g, 1h, 1i, 1m, 1n, 1p, 1s, 2d, 2e, 3g, 4h, 5a, 6d, 7c, 8a, 8g, 9a, 9g and 10j were tested with isolate G2 at five different ages of approximately 7 days, 14 days, 21 days , 32 days and 46 days after sowing. The seeds were sown at intervals such that all ages of plant were ready for testing at the same time. Seeds were sown in pots 6 cm in diameter, and plants that were required to be over 21 days-old were individually transplanted to 12 cm pots approximately 21 days after sowing.

The 7 day-old plants possessed cotyledons, while the 14-day old plants had also developed first and second true leaves. Whole leaves and cotyledons of these seedlings were tested because they were too small to cut into segments. Twenty cotyledons from 7-day old plants of each line were placed in a set of five dishes. This procedure was repeated with the cotyledons of the 14 day-old plants. A random mixture of first and second true leaves from the 14 day-old plants were placed in another set of five Petri dishes. Twenty leaf segments were cut from the 21 ,32 and 46-day old plants of each line and placed in further sets of five Petri dishes. Each Petri dish contained four pieces of leaf material. One leaf or leaf segment in each dish was marked by clipping it and left uninoculated as a control, the other three leaves or leaf segments were inoculated with isolate G2 using the direct dusting method. The Petri dishes were then incubated for 9 days at 18^{o}C with a 12 h photoperiod. The infection types which developed were recorded.

3.2.2. Experiment 1. Results.

The contingency Chi squared table of the frequency of the different infection types obtained in each treatment is given in Table 3.1. Infection types 0 and 1 were combined since infection type 1 occurred at a frequency of less than five in some treatments. Infection types 3 and 4 were also combined due to the low frequency of infection type 3. The χ^2 value was found to be highly significant so analysis of variance was carried out on the data after transforming the six infection types to 0, 1, 1.5, 2.5, 3 and 4 respectively.

Analysis of variance indicated that there were significant differences between plant lines and plant age, and a smaller but still significant, plant line x plant age interaction (Table 3.2). The mean infection scores for each plant line at each age are given in Table 3.3. There were some obvious differences in susceptibility between plant lines. For example line 1g was generally more susceptible than 1e, 1m, 2e etc. This indicated that partial resistance (ie the plants were colonized but to a reduced extent) to isolate G2 may be operating in some lines.

Within plant lines, there were no significant differences in susceptibility between cotyledons and first and second true leaves of the 14 day old plants or between these and the 7 day old plants. However, there were significant differences between each of the four older age categories of some plant lines and this was due to a gradual increase in susceptibility with age, eg lines 8a and 8g. Other plant lines, for example, 9a, remained totally resistant at all ages. A few lines gave unexpected results which were probably due to experimental error. For example, susceptible reactions were recorded on 7 and 46 day-old plants of line 1m but resistant reactions were recorded for intermediate ages.

Table 3.1 Plant age and susceptibility (Experiment 1)
Contingency χ^2 table
Number of leaf segments of each infection type.

Infection Type	Plant age: Cotyledons 7 days	Cotyledons 14 days	True leaves 14 days	True leaves 21 days	True leaves 32 days	True leaves 46 days	Total
0 and 1	223	228	254	187	134	104	1130
2-	42	40	26	58	35	27	228
2+	48	55	27	36	71	77	314
3 and 4	17	7	23	49	90	122	308
Total	330	330	330	330	330	330	1980

χ^2 value = 201.7 Expected χ^2_{15} at P 0.05 = 25

Table 3.2 Analysis of variance for plant lines and plant age (Experiment 1)

Source of variation	DF	SS	MS	VR	FPR
Lines	21	967.243	46.059	38.686	<0.001
Ages	5	530.624	106.125	89.136	<0.001
Lines x Ages	105	589.237	5.612	4.713	<0.001
Residual	528	628.633	1.191		
Total	659	2715.737	4.121		
Within dishes error	1320	851.167	0.645		
Grand total	1979	3566.904			

Co-efficient of variation = 58.0%

STANDARD ERRORS: Ages = 0.0849
Lines = 0.1627
Ages x Lines = 0.3984

Table 3.3 Mean scores for ages and lines combinations (Experiment 1)

Plant Lines	Plant age: Cotyledons 7 days	Cotyledons 14 days	True leaves 14 days	21 days	32 days	46 days	Mean Total
1c	0.10	0.50	0.00	1.50	0.50	2.33	0.82
1e	1.77	0.90	0.30	0.97	2.13	3.30	1.56
1f	2.20	0.27	2.40	2.30	2.40	3.10	2.11
1g	1.53	1.60	2.70	2.27	3.10	3.07	2.38
1h	1.40	0.00	0.07	0.77	1.10	2.50	0.98
1i	0.00	0.00	0.07	0.23	1.83	2.20	0.72
1m	2.17	0.17	0.47	0.80	0.73	2.17	1.08
1n	0.27	0.00	0.00	0.00	0.27	2.00	0.42
1p	1.80	0.80	1.70	2.30	2.53	2.80	1.99
1s	0.83	0.33	1.03	0.57	1.70	1.33	0.97
2d	0.40	1.57	0.90	1.50	3.30	2.87	1.76
2e	0.10	1.50	0.30	0.43	3.00	1.67	1.17
3g	0.57	0.93	0.20	0.43	2.10	1.70	0.99
4h	0.10	0.00	0.00	0.00	0.00	0.00	0.02
5a	0.00	0.20	0.00	0.27	0.57	1.10	0.36
6d	1.17	0.83	0.33	1.67	1.47	0.60	1.01
7c	0.37	2.03	0.63	2.43	2.93	2.90	1.88
8a	0.20	0.87	0.00	1.63	2.30	3.40	1.40
8g	0.53	1.77	1.17	2.20	2.60	3.00	1.88
9a	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9g	0.00	0.00	0.00	0.00	0.10	0.00	0.02
10j	0.30	0.30	0.10	0.40	0.00	1.23	0.39
Mean total	0.718	0.662	0.562	1.030	1.576	1.967	

LSD at P = 0.05 for: Plant age = 0.166
Plant lines = 0.319
Ages x Lines = 0.781

Seedling resistance and adult plant susceptibility to isolate G2 was indicated in some plant lines, eg 1c, 1i, 1n, 5a and 10j, but the age of plant at which resistance was reduced was different for different plant lines. Groundsel line 1c had lost seedling resistance by 21 days after sowing, line 1i did not loose resistance until 32 days and lines 1n, 5a and 10j did not become susceptible until 46 days of age. In the majority of cases, plants showed increased susceptibility once they were in flower, about 28 to 42 days after sowing. No clear cases of adult-plant resistance were found.

3.3. Experiment 2. An investigation of effects of plant age, incubation temperature and leaf position on expression of resistance.

3.3.1. Materials and methods.

This experiment was carried out at N.V.R.S. Four isolates of powdery mildew, G8, G9, G10 and G11 were tested on five groundsel plant lines, 6b, 7d, 8a, 9c and 11i at three different temperatures 10°C, 15°C and 20°C. Plants were tested at two ages, 2 wks and 6 wks after sowing. The 2 week-old plants possessed cotyledons and true leaves which were tested together.

To investigate the effects of leaf number ie leaf age, on susceptibility, leaves from the 6 week-old plants were harvested from four different nodes. An excess of plants was grown for the experiment but there was such variation in the number of nodes on each plant even from within the same plant line that sampling from exactly the same node on each plant was impossible. Plants of similar size were chosen wherever possible and the leaves sampled from the nodes as shown in the Fig 3.6. Leaves were categorized as old, old-intermediate, young-intermediate and young. Leaves at the base of the plant (usually from the 3rd to 6th nodes) were classed as old leaves. Young leaves came from the highest node where leaves were sufficiently expanded to provide test material. The two intermediate categories were from the nodes approximately one third of the distance from the respective youngest or oldest nodes sampled.

The maximum number of segments of a reasonable size for testing (0.5 x 1 cm) that could be cut from most leaves, was six. Therefore, Petri dishes were prepared in blocks of six. Fig 3.7 represents the arrangement of the leaf pieces in a block of six Petri dishes. Each

Fig 3.6

Nodes from which leaves were taken.

For Experiment 2.

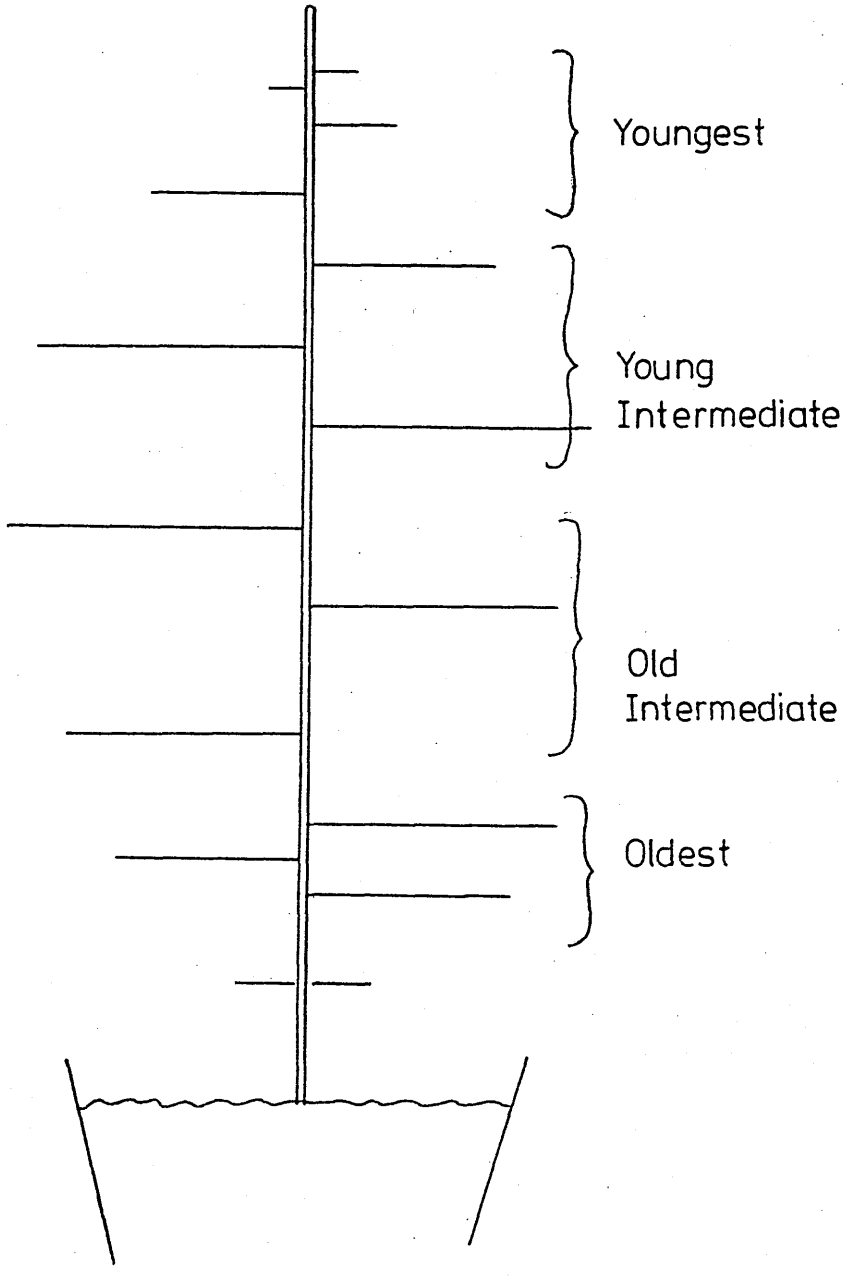
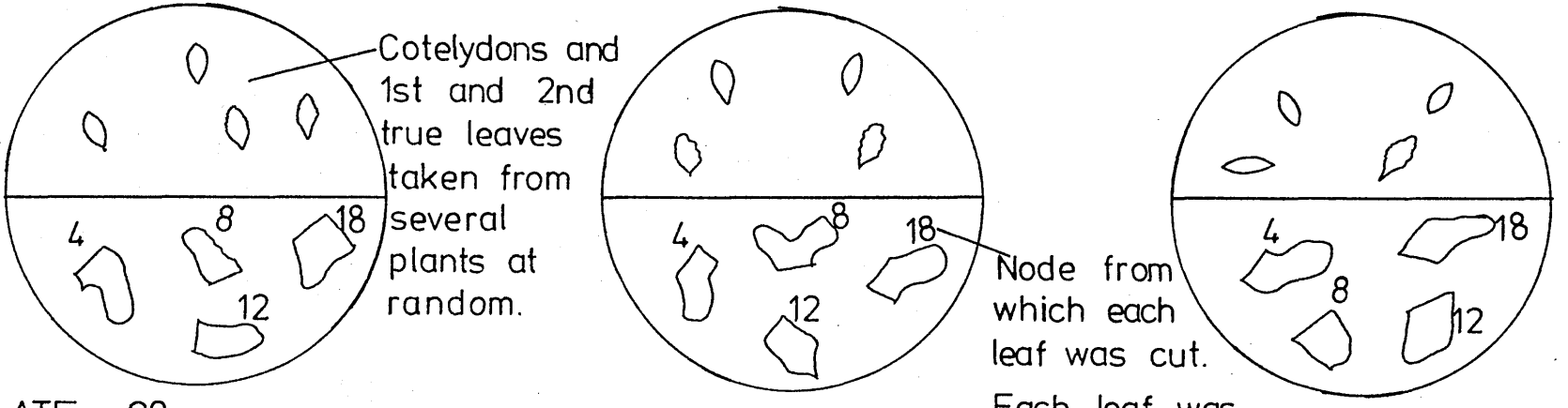


Fig 3.7 A BLOCK OF SIX PETRI DISHES FOR EXPERIMENT 2.

INOCULATED WITH LEAF MATERIAL FROM ONE PLANT LINE.

ISOLATE G10

TEMPERATURE 20°C 15°C 10°C

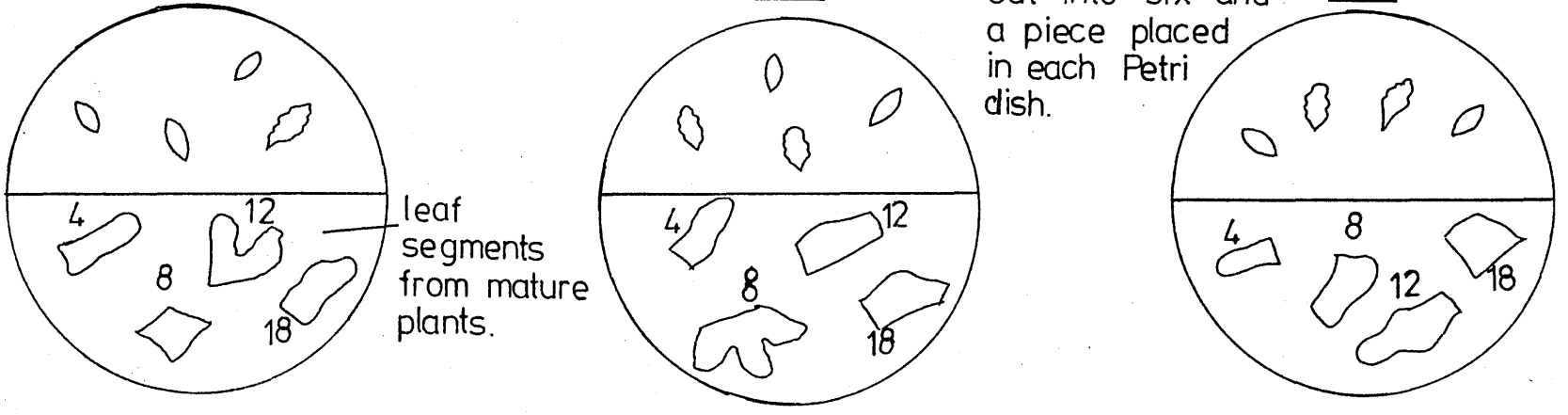


Cotelydons and 1st and 2nd true leaves taken from several plants at random.

Node from which each leaf was cut.

ISOLATE G9

20°C 15°C 10°C



leaf segments from mature plants.

Each leaf was cut into six and a piece placed in each Petri dish.

Fig 3.7

Petri dish in each block of six contained material from one mature plant and from several seedlings of one line. The oldest leaf from a mature plant was cut into six segments and one segment was placed into one half of each of six dishes. This was repeated for the youngest and two intermediate categories of leaf position. The node from which each leaf segment originated was marked on the base of the dish under the appropriate leaf segment. Two cotyledons, and the 1st and 2nd true leaves from the two-week old plants were placed in the other half of each Petri dish. The blocks of six dishes were divided into two sets of three. Each set was inoculated with a different isolate by the direct dusting method; in this case the isolates used were G9 and G10. One dish from each of the sets of three dishes was placed in a growth cabinet for incubation at 20°C, 15°C or 10°C. Other plants of each line were used to set up further blocks of 6 Petri dishes per line which were then inoculated with isolates G8 and G11. Five mature plants for each plant line were used for each isolate pair. Therefore, ten mature plants and several seedlings were used to test the four isolates. All the cabinets used for incubation were maintained with a 12 h photoperiod with a light intensity of approximately 280 μmol m^{-2} s^{-1} provided by high pressure sodium vapour lamps (SOM/R).

3.3.2. Results.

The six infection types were transformed to infection scores 0, 1, 1.5, 2.5, 3 and 4. The mean infection scores obtained are given in Table 3.4. A mean infection score of 0.6 or less was considered to be resistant, any score above 0.6 was susceptible. Analysis of variance was carried out on the data, and the results are given in Table 3.5.

Table 3.4 Mean infection scores for each plant line with each isolate at each incubation temperature, plant age and leaf age

Isolate	Plant line	Temper-ture	Mature plant Old	Int Old	Int Young	Young	Seedling
G8	11i	20°C	0.0	0.0	0.0	0.0	0.2
		15°C	0.0	0.6	0.9	0.5	0.4
		10°C	0.0	0.0	0.0	0.6	0.1
G8	9c	20°C	0.0	0.0	0.0	0.0	0.3
		15°C	0.0	0.0	0.3	0.0	0.4
		10°C	0.3	0.0	0.0	0.0	0.5
G8	6b	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.0	0.0	0.0	0.0	0.2
		10°C	0.0	0.3	0.0	0.0	0.3
G8	7d	20°C	0.0	0.0	0.0	0.0	1.4
		15°C	0.0	0.3	0.0	0.0	1.0
		10°C	0.0	0.0	0.0	0.0	2.1
G8	8a	20°C	2.6	3.1	2.4	2.8	1.5
		15°C	1.7	2.2	2.6	2.7	2.0
		10°C	0.9	1.3	1.7	1.4	1.1
G9	11i	20°C	2.1	2.2	2.1	2.4	0.0
		15°C	2.1	3.0	2.3	2.1	0.9
		10°C	0.3	0.8	2.3	1.6	0.1
G9	9c	20°C	0.5	0.0	0.0	1.3	0.1
		15°C	0.9	0.9	1.8	1.5	0.5
		10°C	0.3	0.9	0.6	0.9	0.3
G9	6b	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.0	0.0	0.3	0.0	0.3
		10°C	0.0	0.0	0.0	0.0	0.1
G9	7d	20°C	0.0	0.0	0.6	0.5	0.2
		15°C	0.0	0.0	0.9	0.8	0.8
		10°C	0.0	0.0	0.8	0.6	0.3
G9	8a	20°C	1.1	2.0	3.2	1.4	0.5
		15°C	1.8	2.5	3.4	2.4	1.5
		10°C	0.8	2.1	1.7	2.0	0.3
G10	11i	20°C	0.0	0.3	0.3	0.5	0.1
		15°C	1.1	1.4	0.6	1.0	0.5
		10°C	0.3	0.6	0.6	0.3	0.2
G10	9c	20°C	0.0	0.0	0.0	0.0	1.3
		15°C	0.0	0.3	0.3	0.0	2.1
		10°C	0.3	0.0	0.0	0.0	1.3
G10	6b	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.0	0.0	0.0	0.3	0.4
		10°C	0.0	0.0	0.0	0.0	0.1
G10	7d	20°C	0.0	0.6	0.0	0.0	0.2
		15°C	0.0	0.0	0.6	0.0	0.6
		10°C	0.0	0.0	0.0	0.0	0.0

Table 3.4 continued

Isolate	Plant line	Temper-ture	Old	Int Old	Mature plant Int Young	Young	Seedling
G10	8a	20°C	2.1	3.0	3.3	3.5	1.1
		15°C	1.5	3.2	3.5	2.9	2.4
		10°C	0.6	1.9	2.3	1.4	1.2
G11	11i	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.3	0.3	0.3	0.3	0.2
		10°C	0.0	0.0	0.6	0.0	0.2
G11	9c	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.0	0.0	0.0	0.6	0.1
		10°C	0.0	0.0	0.0	0.3	0.0
G11	6b	20°C	0.0	0.0	0.0	0.0	0.0
		15°C	0.0	0.0	0.0	0.0	0.0
		10°C	0.0	0.0	0.0	0.0	0.3
G11	7d	20°C	0.0	0.0	0.0	0.0	0.1
		15°C	0.0	0.0	0.0	0.3	0.6
		10°C	0.0	0.3	0.0	0.0	3.0
G11	8a	20°C	1.1	2.3	3.0	2.1	1.6
		15°C	1.6	1.6	1.8	1.9	1.5
		10°C	0.6	1.4	1.7	1.6	1.3

Table 3.5 Analysis of variance for experiment 2

Source of Variation	DF	SS	MS	VR	VR2	P
Replicate	4	3.58	0.894	1.38	-	NS
Isolate	3	17.66	5.890	9.11	0.81	NS
Line	4	739.59	184.897	285.86	4.38	NS
Temperature	2	38.61	19.307	29.85	2.19	NS
Plant age	1	0.14	0.143	0.22	-	NS
Plant age, leaf age	3	21.21	7.071	10.933	2.87	NS
Isolate, line	12	144.33	12.028	18.60	1.53	NS
Isolate, temp	6	43.28	7.214	11.15	3.50	0.05
Line, temp	8	73.56	9.195	14.22	4.46	0.01
Isolate, plant age	3	75.89	25.29	39.11	3.21	NS
Line, plant age	4	172.78	43.195	66.78	5.59	0.01
Temperature, plant age	2	17.52	8.760	13.54	4.35	0.05
Isolate, (plant age + leaf age)	9	6.12	0.680	1.05	-	NS
Line, (plant age + leaf age)	12	30.62	2.552	3.95		0.001
Temperature, (plant age + leaf age)	6	0.72	0.121	0.19	-	NS
Isolate, line, temperature	24	42.07	1.753	2.71	0.95	NS
Isolate, line, (plant age)	12	94.28	7.857	12.15	3.81	0.01
Isolate, temperature, (plant age)	6	11.13	1.854	2.87	0.90	NS
Line, temperature, (plant age)	8	19.12	2.390	3.70	1.16	NS
Isolate, line, (plant age + leaf age)	36	14.62	0.406	0.63	-	NS
Isolate, temperature, (plant age + leaf age)	18	5.99	0.333	0.51	-	NS
Line, temperature, (plant age + leaf age)	24	5.87	0.245	0.38	-	NS
Isolate, line, temperature, (plant age)	24	49.44	2.050	3.19		0.001
Isolate, line, temperature, (plant age + leaf age)	72	25.45	0.354	0.55	-	NS
Error or residual	2096	1355.74	0.647			-
Total	2399	3009.35		1.254		-

Coefficient of variation = 128% DF = 2096 Se = 0.8043

SS = Sums of Squares, DF = Degrees of Freedom, MS = Mean Square, VR = Variance Ratio using error variation, VR2 = Variance Ratio using lower order variation for calculation

3.3.2.1 The effect of plant age.

Table 3.6 gives the mean infection scores for each plant line tested with each isolate for both ages of plant. Plant age was found to affect susceptibility or resistance in certain isolate/plant line combinations. Table 3.7 shows the resistant and susceptible reactions of each isolate/plant line combination for both plant ages and indicates which differences are significant.

There were instances of adult plant resistance in some lines to some isolates. Plant line 7d was susceptible to mildew isolates G8 and G11 as a seedling but resistant as an adult. This was also indicated in preliminary experiments. Groundsel line 9c also provided an example of adult plant resistance with isolate G10, but interestingly appeared to possess seedling resistance to isolate G9. The latter interpretation must be treated with caution as line 9c showed only a relatively low level of susceptibility as an adult plant to isolate G9. Line 11i gave a more definite example of seedling resistance to isolate G9. Age dependent resistance is probably quite common in the groundsel/powdery mildew pathosystem since it has been found in a relatively high proportion of the few plant line and isolate interactions examined in this experiment.

Plant line 6b was equally resistant to all four mildew isolates (G8, G9, G10 and G11), at both plant ages tested, suggesting that resistance of some plant lines is not age dependent.

Plant line 8a was susceptible to all four isolates at both ages of plant but the level of susceptibility of the seedling was significantly lower than that of the adult stage. This supports the evidence of experiment 1. that plant lines which are susceptible to most isolates tend to be less susceptible in the seedling stage. Field observations reported in Chapter 8 also indicated that many young plants in natural groundsel populations were relatively free of

Table 3.6 The effect of age of plant on infection score

Isolate	Plant line 11i		9c		6b		7d		8a	
	A	S	A	S	A	S	A	S	A	S
G9	1.86	0.33	0.80	0.32	0.03	0.13	0.35	0.43	2.03	0.74
G10	0.58	0.23	0.08	1.54	0.03	0.17	0.10	0.26	2.43	1.58
G11	0.15	0.13	0.08	0.05	0.00	0.09	0.05	1.23	1.73	1.43
G8	0.22	0.19	0.05	0.38	0.08	0.18	0.03	1.50	2.12	1.53

STANDARD ERROR: 0.1468

LSD at P = 0.05 = 0.288
" " P = 0.01 = 0.878
" " P = 0.001 = 0.733

A = Adult S = Seedling

Table 3.7 Summary of age of plant and its effect on resistance

(Including the probability of infection scores being significantly different)

Isolate	Plant Age	Plant line 11i	P	9c	P	6b	P	7d	P	8a	P
G9	Adult	S	0.001	S	0.001	R	NS	R	NS	S	0.001
	Seed	R		R		R		R		S	
G10	Adult	R	0.05	R	0.001	R	NS	R	NS	S	0.001
	Seed	R		S		R		R		S	
G11	Adult	R	NS	R	NS	R	NS	R	0.001	S	0.05
	Seed	R		R		R		S		S	
G8	Adult	R	NS	R	0.05	R	NS	R	0.001	S	0.001
	Seed	R		R		R		S		S	

NS = No significant different
R = Resistant = mean score of 0.6 or less
S = Susceptible = mean score of more than 0.6
P = Probability of difference between infection scores being significant
Seed = Seedling

mildew.

3.3.2.2. Effects of temperature and plant age on susceptibility.

The variance ratio for the isolate x plant line x temperature x plant age combination was significant. (see Table 3.5) This suggests that incubation temperature may affect susceptibility of certain isolate/plant line combinations at certain stages of development. Table 3.8 gives the mean infection scores for these combinations. Plant line 7d when inoculated with isolate G11 had a low level of susceptibility both as a seedling and as an adult plant at all temperatures except for the seedling stage incubated at 10°C where a high mean infection score of 3.0 was obtained. In other cases, for example, plant line 11i with isolate G9, temperature did not affect the expression of age dependent resistance.

Table 3.9 and Fig 3.8 show the general effects of temperature on the total mean infection scores of adult and seedling plants. In general, incubation at 15°C gave a uniformly high infection score at both plant ages. Raising the incubation temperature to 20°C tended to reduce the susceptibility of seedlings more than that of adult plants. Lowering the temperature of incubation to 10°C had the reverse effect; the susceptibility of the adult plants was lowered significantly more than that of the seedling.

3.3.2.3. Effects of temperature on the susceptibility of each plant line.

The effect of incubation temperature on the mean infection scores of the plant lines is illustrated in Table 3.10 and Fig 3.9. Four of the plant lines 6b, 8a, 9c and 11i, responded in the same way to temperature, being most susceptible at 15°C. Preliminary experiments also indicated that plant line 11i was more susceptible to isolate G12

Table 3.8 Effect of temperature and age of plant on mean infection score

Isolate	Temperature / Plant Line	20°C Adult	20°C Seedling	15°C Adult	15°C Seedling	10°C Adult	10°C Seedling
		Age of plant					
G9	11i	2.20	0.00	2.38	0.93	1.00	0.08
	9c	0.45	0.13	1.29	0.50	0.67	0.33
	6b	0.00	0.00	0.08	0.32	0.00	0.08
	7d	0.28	0.15	0.43	0.80	0.35	0.33
	8a	1.93	0.50	2.53	1.45	1.65	0.28
G10	11i	0.28	0.08	1.03	0.45	0.45	0.15
	9c	0.00	1.28	0.15	2.10	0.08	1.25
	6b	0.00	0.00	0.08	0.43	0.00	0.08
	7d	0.15	0.15	0.15	0.63	0.00	0.00
	8a	2.98	1.18	2.78	2.38	1.55	1.23
G11	11i	0.00	0.00	0.30	0.15	0.15	0.23
	9c	0.00	0.00	0.15	0.08	0.08	0.00
	6b	0.00	0.00	0.00	0.00	0.00	0.28
	7d	0.00	0.08	0.08	0.58	0.08	3.08
	8a	2.13	1.55	1.73	1.48	1.33	1.28
G8	11i	0.00	0.15	0.50	0.35	0.15	0.08
	9c	0.00	0.28	0.08	0.43	0.08	0.45
	6b	0.00	0.00	0.00	0.23	0.08	0.33
	7d	0.00	1.40	0.08	1.00	0.00	2.10
	8d	2.73	1.53	2.30	2.00	1.33	1.08

STANDARD ERROR = 0.2543
LSD at P = 0.05 = 0.498
" " P = 0.01 = 0.655
" " P = 0.001 = 0.837

Table 3.9 Effects of plant age and temperature on mean infection score

Temperature	Plant age: Adult	Plant age: Seedling
20°C	0.66	0.42
15°C	0.80	0.81
10°C	0.45	0.63

STANDARD ERROR = 0.057
LSD at P = 0.05 = 0.112
" " P = 0.01 = 0.147
" " P = 0.001 = 0.187

Table 3.10 Effect of temperature on mean infection score of each plant line

Plant line	Temperature 20°C	15°C	10°C
11i	0.34	0.76	0.28
9c	0.27	0.59	0.37
6b	0.00	0.14	0.10
7d	0.26	0.47	0.73
8a	1.81	2.08	1.21

STANDARD ERROR = 0.09
LSD at P = 0.05 = 0.176
" " P = 0.01 = 0.232
" " P = 0.001 = 0.296

Table 3.11 The effect of temperature on isolate performance

Isolate	Temperature 20°C	15°C	10°C
G9	0.56	0.07	0.48
G10	0.60	1.02	0.48
G11	0.38	0.45	0.64
G8	0.61	0.70	0.57

(Mean infection score is for the mean of all plant lines with each isolate)

STANDARD ERROR = 0.080
LSD at 5% level = 0.176
" " 1% level = 0.232
" " 0.1% level = 0.296

Fig 3·8

THE EFFECTS OF TEMPERATURE AND AND AGE OF PLANT ON MEAN INFECTION SCORE.

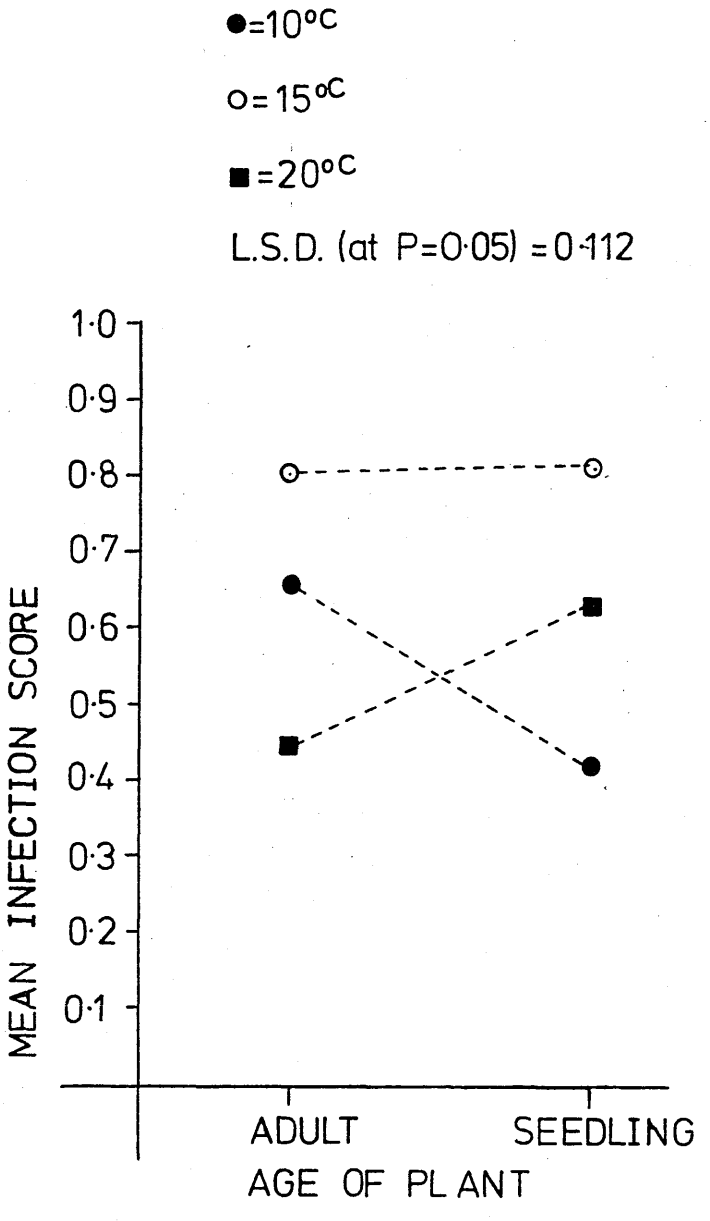


Fig 3.9

THE EFFECT OF TEMPERATURE ON MEAN INFECTION SCORE OF EACH PLANT LINE.

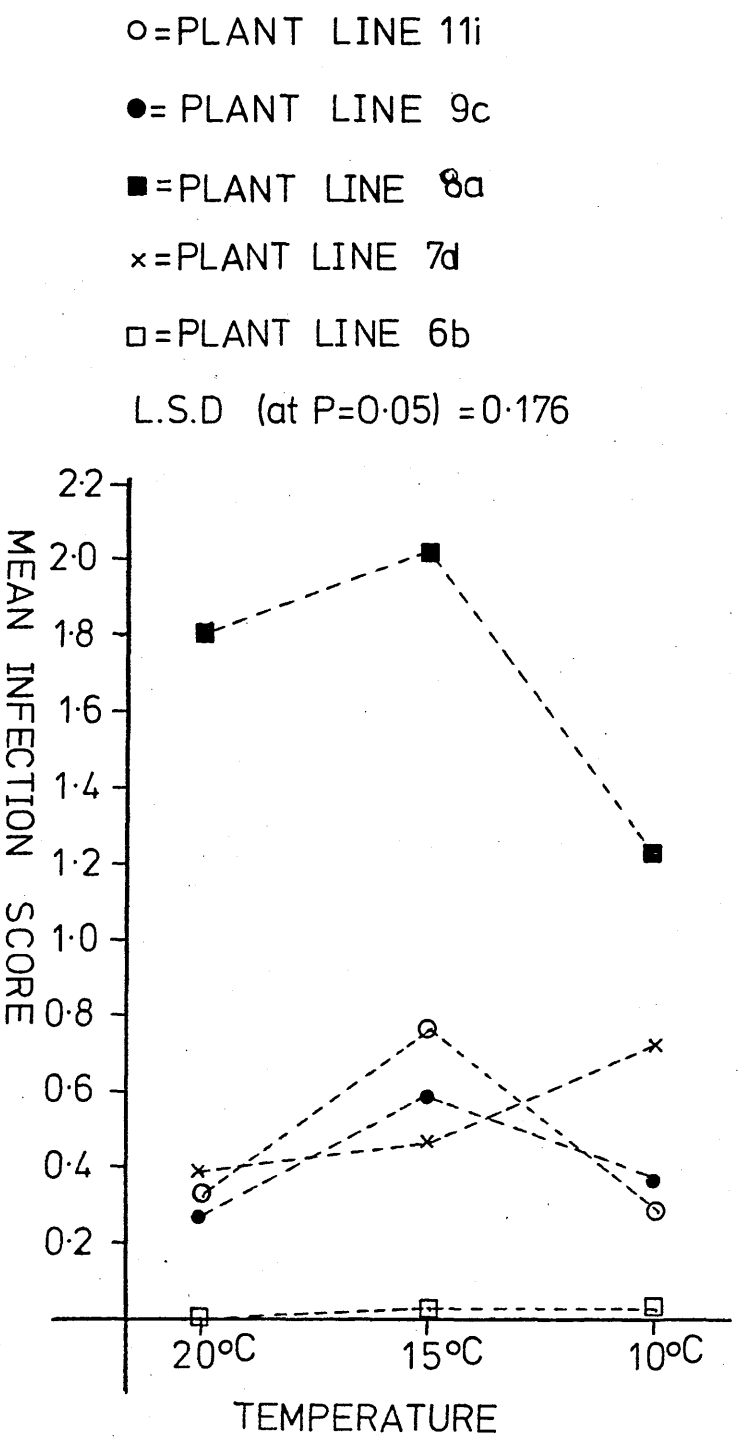
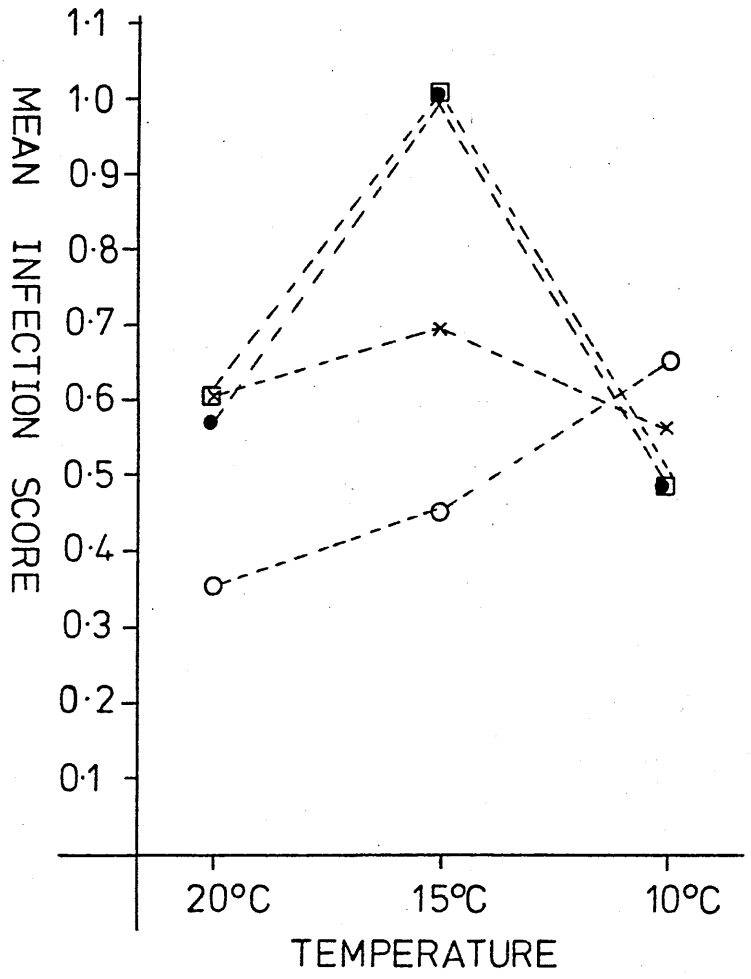


Fig 3.10

THE EFFECTS OF TEMPERATURE ON THE GROWTH OF EACH ISOLATE.

× = Isolate G8 □ = Isolate G10

● = Isolate G9 ○ = Isolate G11



at 15°C than at 20°C. However, one plant line, 7d, became more susceptible as the temperature decreased.

3.3.2.4. The effect of temperature on isolate performance.

The overall response to incubation temperature of each isolate over all plant lines tested is given in Table 3.11 and Fig 3.10. Isolates responded differently to different incubation temperatures. Isolates G9 and G10 behaved in a similar way, producing the highest infection scores at 15°C. Isolate G8 caused more or less constant infection scores regardless of temperature, whilst isolate G11 caused higher infection scores as the incubation temperature was lowered.

3.3.2.5 Effect of leaf age on susceptibility.

Preliminary experiments indicated that the upper leaves of plant line 11i were more susceptible to isolate G12 than the lower leaves. However, this experiment, using other isolates, revealed no such differences. Table 3.12 gives the mean infection scores obtained for the seedling leaves and the four leaf positions for each plant line. The variance ratio for this combination of treatments was significant but most of the difference was due to the differences in susceptibility between the two ages of plant and not to differences between leaf ages of mature plants. The oldest leaves tested from the mature plants tended to be less susceptible than the other leaf categories (old-intermediate, young-intermediate and young). The oldest leaves probably appeared less susceptible as they tended to senesce more quickly during testing and became colonized with contaminants such as Botrytis which discouraged the growth of the mildew.

Table 3.12 Effect of leaf age and plant age on susceptibility

Plant line	Age of leaf Old	Old Int	Young Int	Young	Seedling
11i	0.52	0.77	0.75	0.78	0.22
9c	0.19	0.18	0.25	0.38	0.57
6b	0.00	0.03	0.03	0.03	0.14
7d	0.00	0.10	0.24	0.18	0.85
8a	1.37	2.22	2.56	2.18	1.32
Mean Total	0.415	0.657	0.763	0.708	0.620

LSD for Leaf age x Plant age

LSD = 1.96 x 0.1468 = 0.288 at P = 0.05
" = 2.576 x 0.1468 = 0.378 at P = 0.01
" = 3.291 x 0.1468 = 0.483 at P = 0.001

LSD for Leaf age

LSD = 1.96 x 0.1161 = 0.228 at P = 0.05
" = 2.576 x 0.1161 = 0.299 at P = 0.01
" = 3.291 x 0.1161 = 0.382 at P = 0.001

LSD for Plant age

LSD = 1.96 x 0.0734 = 0.144 at P = 0.05
" = 2.576 x 0.0734 = 0.189 at P = 0.01
" = 3.291 x 0.0734 = 0.242 at P = 0.001

3.3.2.6. Plant and isolate main effects.

Although the variance ratios for isolates and plant line main effects were large when calculated using the residual error, they were no longer significant when they were calculated using the variances for the lower order combinations of treatments. This is explained by specific effects of temperature, plant age or leaf number on particular plant line/ isolate combinations. This emphasises the importance of the effect of the environment on the expression of disease resistance. Kulkarni and Chopra (1982) pointed out that race specific resistance reported in some experimental work, (for example, the work of Scott and Hollins (1977) on resistance of wheat to isolates of Cercosporella herpotrichoides), could be explained by different enviromental effects on the reactions of certain host lines and pathogen isolates. Thus, reaction patterns appearing to be race specific, may, in some cases, be artifacts caused by environmental effects on the reaction between host and pathogen.

3.4 Discussion.

Resistance which is expressed only either at the seedling stage or adult stage has clearly been demonstrated in some groundsel line/isolate combinations. This situation also occurs in crop-pathogen associations. Some cultivars of winter wheat were found to be more susceptible to Erysiphe graminis f.sp. tritici in the adult stage whilst other cultivars were more resistant at the seedling stage (Bennett, 1981a,b). Both forms of resistance are important and are utilized in crops. In some cases the age dependent resistance is only partial, for instance, the slow mildewing of 'Knox' wheat is only expressed in the adult stage (Shaner,1973). In other cases, such as the barley cultivars Athos and Porthos, resistance is complete in seedlings but only partially expressed in adults (Wright and Heale, 1984). Race specific resistance genes to Puccinia recondita in wheat are also known to be expressed differentially at different plant ages (Rajaram, et al 1971).

Experiment 1. indicated that the period of growth over which seedling resistance was expressed was different for different plant lines. Briggle and Scharen (1961) found that this also occurred with resistance to Erysiphe graminis tritici in some wheat cultivars and depended on the culture of powdery mildew used.

There were indications that groundsel lines which tended to be highly susceptible to most isolates at maturity, eg lines 8a and 8g, tended to be less susceptible during the seedling stage. Observations in the field reported in Chapter 8 also indicated that in general, seedlings are less susceptible than adults. It is possible that low seedling susceptibility together with low levels of mildew inoculum in spring and early summer interact to minimize damage to young seedlings

and prevent a severe epidemic before the groundsel seeds.

Experiment 2. indicated that incubation temperature could affect host susceptibility and that this was often age dependent. This is known to occur in some wheat cultivars with race specific resistance to Puccinia recondita (Rajaram et al. 1971., Dyck and Johnson, 1983). The performance of mildew isolates was also temperature dependent but specific to certain plant lines. Different isolates were affected differently by temperature. Generally, the optimum temperature for the growth of mildew was 15°C but isolate G11 grew better at 10°C than at the higher temperatures.

It seems likely that environmental conditions play an important part in the expression of resistance to powdery mildew in natural populations of groundsel. Heather and Chandrashekar (1982a,b) found that temperature and light intensities affected the resistance of Populus spp to Melamspora leaf rust. They postulated that environmental factors played an important role in wild host-parasite interactions. If resistance genes only operate under certain environmental conditions, then the period of time the genes impose selection on the pathogen for virulence to those particular genes is also limited and the build up of races virulent for those resistance genes would be reduced. Kulkarni and Chopra (1982) also found that environment played an important part in the outcome of different host and pathogen combinations. As mentioned previously, differential effects of environment on pathogen and host genotypes can cause reaction patterns to appear race specific. They also supported the hypothesis that environmentally sensitive genes in race specific pathosystems may lead to greater durability of resistance which could help to explain the stability of resistance in natural plant pathosystems.

3.5 Conclusions from experiments 1 and 2.

The main conclusions of these experiments in relation to the testing of groundsel with mildew isolates in further experiments were:

1. Old leaves should not be used.
2. The tests should be incubated at 15°C or above when testing adult plants.
3. The plant lines should be tested at the same stage of growth, normally at about 46 days after sowing when the plants have a plentiful supply of leaves in a suitable state for testing.

CHAPTER 4. CHARACTERIZATION OF MILDEW ISOLATES.

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4.0 CHARACTERIZATION OF MILDEW ISOLATES.

4.1 Introduction.

Harry (1980), and Harry and Clarke(1986) found the differential reactions between the groundsel lines and mildew isolates they investigated indicative of the operation of a 'gene-for-gene' relationship. Investigations on the inheritance of resistance in some groundsel lines by Harry (1980) showed that in most cases response to mildew was regulated by genes at a single locus with resistance dominant to susceptibility. Inheritance studies were not carried out with the fungus as a sexual stage is not known in Britain.

The gene-for-gene hypothesis was proposed and first described in detail by Flor (1956). The hypothesis was based on work reported in several papers (including Flor 1935, 1942, 1946, 1947) on the flax (*Linum usitatissimum*) and flax rust (*Melampsora lini*). The inheritance of resistance in several host varieties and avirulence in several pathogen races was studied simultaneously. For example, Flor (1947) crossed the variety Ottawa, which was susceptible to race 22 but resistant to race 24 of the flax rust, with Bombay, which was resistant to race 22 but susceptible to race 24. At the same time he crossed races 22 and 24. All the F1 progeny from the Ottawa x Bombay cross were resistant to both race 22 and 24. On selfing, the F2 segregated with an approximate ratio of 9:3:3:1. That is 9/16 were resistant to both races, 3/16 were resistant to race 22 but susceptible to race 24, 3/16 were resistant to race 24 but susceptible to race 22, and 1/16 were susceptible to both races.

The cross between the rust races 22 and 24 produced an F1 that was avirulent on both Ottawa and Bombay. The F2 segregated giving

ratios of 9/16 avirulent on both varieties, 3/16 virulent on Ottawa but not on Bombay, 3/16 virulent on Bombay but not on Ottawa, and 1/16 were virulent on both varieties.

Flor (1956) concluded that resistance in each variety to the respective rust races was dominant and that resistance to each race was inherited at a single but separate locus. Virulence was recessive and also controlled by a single locus. The results showed that resistance in each variety and avirulence in each race was conditioned by pairs of complementary genes. From these results and those of many other crosses, Flor proposed the gene-for-gene hypothesis (1956) which states:

'For each gene conditioning rust reaction in the host there is a specific gene conditioning pathogenicity in the parasite'.

The work of Flor stimulated the study of inheritance of resistance and susceptibility in many pathosystems. These include the Lactuca sativa/Bremia lactucae (Crute and Johnson, 1976, and Norwood and Crute, 1984) and the Hordeum vulgare/Erysiphe graminis hordei (Moseman, 1957) pathosystems.

It is not always possible to carry out inheritance studies with both the host and the pathogen. Person (1959) proposed a model, later reviewed by Robinson (1976), whereby a gene-for-gene relationship can be inferred in the absence of inheritance studies in host or pathogen. Person studied the mathematical properties of a theoretical model parasitic system involving a gene-for-gene relationship. He concluded that if the reaction patterns produced by the reactions between several host and pathogen phenotypes in another pathosystem had the same mathematical properties as his model, then it followed that the pathosystem under study must also involve a 'gene-for-gene' relationship. On the basis of this model many other pathosystems

demonstrate 'gene-for-gene' relationships. For example, Bettencourt and Noronha-Wagner (1967) demonstrated that a gene-for-gene relationship occurred in the Coffea arabica/Hemileia vastatrix pathosystem, where a sexual stage in the pathogen is unknown.

The aims of the work in the present chapter were to determine the virulence phenotypes of several mildew isolates collected at two locations. Isolates known to be of different virulence phenotype would be more likely to distinguish different resistance phenotypes in the groundsel populations to be examined in work reported in Chapter 7. However, it was also desirable to obtain information on the structure of the mildew poulation, that is, which virulence phenotypes were common in the mildew population, and whether there were any differences between the mildew populations from Glasgow and the N.V.R.S. Harry (1980) found that all the mildew isolates she investigated were complex, having virulence for all but one of the resistance factors she identified. The majority, i.e. 8 out of 9 isolates Harry studied, had unique virulence phenotypes. This suggested that there might be a large number of phenotypes in the pathogen population. However, Harry's method of isolate collection was to obtain most isolates from plant lines already known to possess resistance to one or more of the previously tested isolates. This would probably ensure that the isolates were of different phenotype and also to be complex for virulence. Isolates collected in this way would not necessarily be a representative or random sample of the total population. All the mildew isolates collected by Harry were from Glasgow, so they could be considered to be components of the same population.

The isolates obtained in the present study were taken randomly from groundsel plants of completely unknown resistance phenotype. It was hoped that these isolates would be representative of the two

mildew populations at Glasgow and N.V.R.S. so that a comparison between the two mildew populations could be made.

4.2. Materials and methods.

Twelve single conidial chain isolates from the N.V.R.S (N1 to N12) collected in 1983 and twelve from Glasgow (G1 to G12) collected in 1984, were obtained and maintained as described in Chapter 2. Each isolate was tested on leaf segments from the 50 inbred groundsel lines. Plants of the same line were assumed to have similar resistance phenotypes so that two or three plants of each line could be grown up for each batch of testing.

The two populations of isolates were tested on separate occasions, the N.V.R.S. isolates were tested on the plant lines using N.V.R.S facilities, whilst the Glasgow isolates were tested at Glasgow. Wherever possible, different isolates were tested on all the 50 lines, on different, but consecutive days to avoid cross contamination.

Where a plant line/isolate test gave a completely resistant or inconsistent low infection type, the test was repeated using leaves from the same plant. For this second test the plants were 8 to 9 weeks old.

4.3. Results.

The infection types 0, 1, 2-, 2+, 3 and 4 were transformed for the purposes of analysis to 0, 1, 1.5, 2.5, 3 and 4. The mean infection score for each isolate/ plant line pair was calculated for all 24 isolates and 50 plant lines tested, and the results are given in Appendix Tables 4.1 and 4.2.

4.3.1. The determination of resistance and susceptibility.

The mean scores were divided into classes within limits of 0.2 units and the frequencies of each mean score class are plotted in Fig 4.1. The least frequent mean infection score fell between the limits of 0.4 and 0.6. The trough in infection scores between these limits was quite prominent, indicating that the infection scores fall into two groups. A mean infection score of 0.6 or less was considered to reflect a resistant reaction, whilst mean scores above 0.6 were taken to reflect increasing degrees of susceptibility.

4.3.2. Characterization of different mildew races and plant line phenotypes.

Table 4.1 gives the results of all 24 isolate/ plant line compatibility tests categorised into resistant and susceptible reactions. Eighteen of the mildew isolates (nine of the Glasgow isolates: G1, G2, G6, G7, G8, G9, G10, G11 and G12, and nine of the N.V.R.S. isolates: N1, N2, N4, N5, N6, N7, N8, N9 and N11) and 21 of the plant lines were different from each other and their reactions are given in Table 4.2. Only one of the Glasgow isolates (G8) had a phenotype similar to one of the N.V.R.S. isolates (N3). None of the 18 isolates had a phenotype similar to any found by Harry (1980). This indicates that the number of different mildew isolates in each area is likely to be high.

The reactions summarized in Table 4.2 confirm the presence of race specific resistance in the pathosystem. Specific resistance factors may be postulated and assigned to the plant lines. Likewise the corresponding avirulence factors may be assigned to the mildew isolates. Plant lines with resistance to isolate G1 are allocated resistance factor 1, plant lines resistant to isolate G2 are allocated

Fig 4.1

Histogram of the % of reactions of each infection type lying between each 0·2 limit produced by the 24 isolates on 50 inbred groundsel lines.

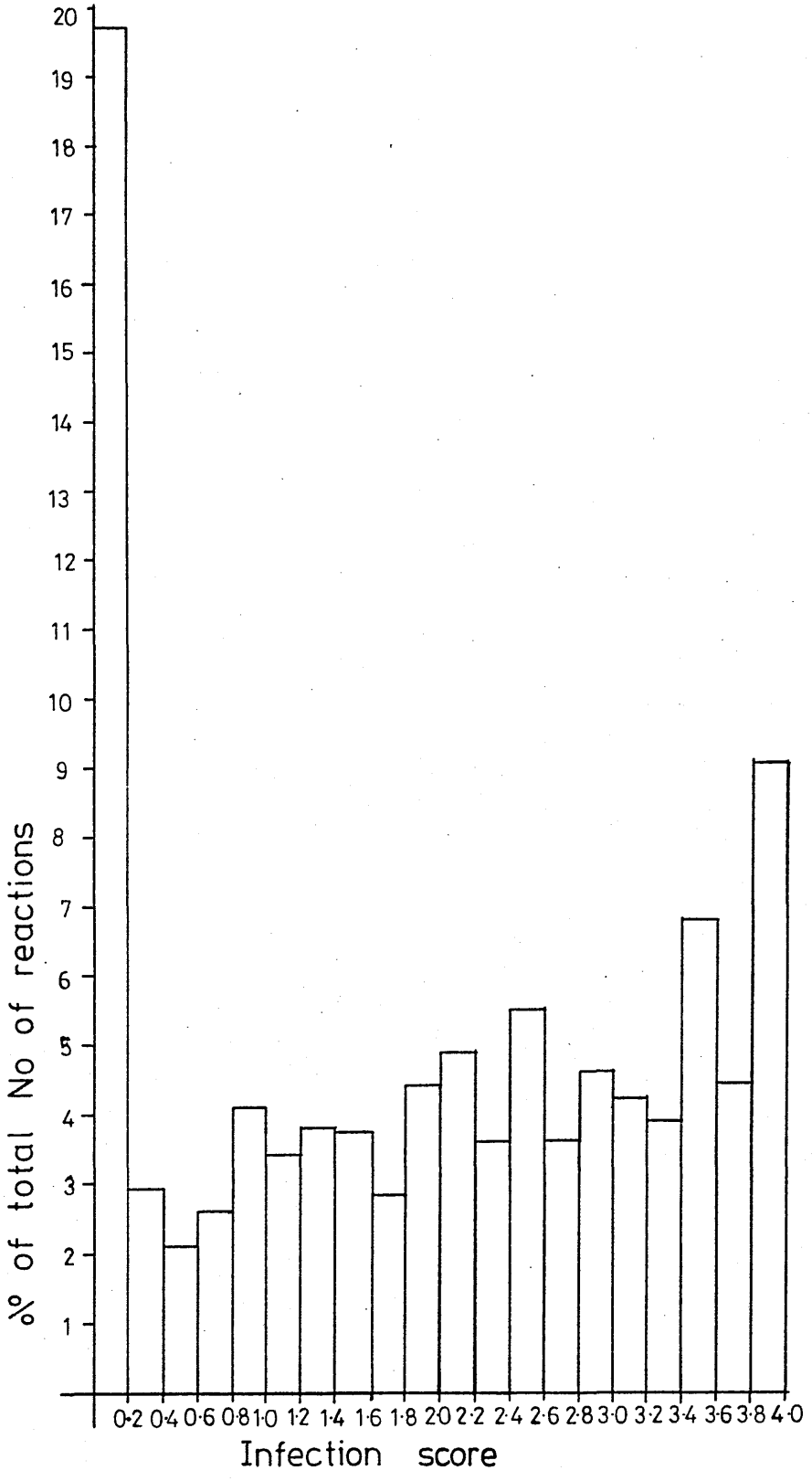

Table 4.1 Compatibility of each groundsel line with each isolate

Plant Line	Isolate																						
	G 1	G 2	G 3	G 4	G 5	G 6	G 7	G 8	G 9	G 10	G 11	G 12	N 1	N 2	N 3	N 4	N 5	N 6	N 7	N 8	N 9	N 10	N 11	N 12
1c	+	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1e	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1f	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+
1g	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1h	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+
1i	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1m	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+
1n	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1p	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+
1s	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2a	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2d	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
2e	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
2i	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
3f	+	R	+	+	+	+	+	+	+	+	+	R	+	+	+	R	+	+	+	+	+	+	R	+
3g	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
4a	R	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	R	+
4h	R	R	R	+	+	R	R	+	R	+	+	R	R	R	+	+	+	+	+	R	+	R	R	R
5a	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
6b	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
6d	R	+	+	+	+	+	+	+	R	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
6f	+	R	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	R	+	+	+	R	+
7a	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
7b	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
7c	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+
7d	R	R	R	R	R	+	R	R	+	R	R	+	R	R	R	R	R	R	R	R	R	R	R	R
7f	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
8a	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
8g	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
9a	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
9c	R	R	R	R	R	+	R	R	+	R	R	+	R	R	R	R	R	R	R	R	R	R	R	R
9d	R	R	R	R	R	+	R	R	+	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
9g	+	R	R	R	+	R	R	+	R	R	+	R	R	R	+	R	R	+	R	R	R	R	+	R
10j	+	+	+	+	+	+	+	+	+	+	+	+	R	R	+	R	R	R	R	R	R	R	R	R
11a	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
11e	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
11i	R	R	R	R	R	+	R	+	+	R	R	+	R	+	+	+	+	R	R	R	R	R	+	R
14h	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
15c	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
15j	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
16d	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
16f	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
17h	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
18i	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
19b	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+
23f	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
23g	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
23i	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
24f	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
24j	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

R = Resistant
+ = Susceptible

TABLE 4.2. The phenotypes of the unique isolates and plant lines.

R = Resistant (Mean infection score 0.6 or less)

+ = Susceptible.

	ISOLATE																
PLANT LINE	G 1	G 2	G 6	G 7	G 8	G 9	G 10	G 11	G 12	N 1	N 2	N 4	N 5	N 6	N 7	N 8	N 9	N 11
1c	+	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1e	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1f	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+
1m	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+
1p	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R
3f	+	R	+	+	+	+	+	+	R	+	+	R	+	+	+	+	+	R
3g	+	+	+	+	+	+	+	+	R	+	+	+	+	+	+	+	+	+
4a	R	R	+	+	+	+	+	+	+	+	+	+	+	+	R	+	+	+
4h	R	R	R	R	+	R	+	+	R	R	R	+	+	+	+	R	+	R
5a	R	+	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
6b	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R
6d	R	+	+	+	+	R	+	+	R	+	+	+	+	+	+	+	+	+
6f	+	R	+	+	+	+	+	+	+	+	+	R	+	+	R	+	+	R
7d	R	R	+	R	R	+	R	R	+	R	R	R	R	R	R	R	R	R
9d	R	R	+	R	R	+	R	R	R	R	R	R	R	R	R	R	R	R
9g	+	R	R	R	+	R	R	+	R	R	R	R	R	+	R	R	R	+
10j	+	+	+	+	+	+	+	+	+	R	R	R	R	R	R	R	R	R
11i	R	R	+	R	+	+	R	R	+	R	+	+	+	R	R	R	R	+
19b	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	R	+
23f	+	R	+	+	+	R	+	+	+	+	+	+	+	+	+	+	+	+
24f	R	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

resistance factor 2, and so on. In this way the resistance phenotypes of the lines to the various isolates may easily be described as in Table 4.3. The reaction patterns are indicative of a 'gene-for-gene' relationship (Person, 1959). However, a genetical analysis was not carried out to determine if each factor was controlled by a single gene.

4.3.3. A possible 14 gene model to explain the data

It is possible to develop gene models to fit the data on the assumption that a 'gene-for-gene' system is operating. The following 14 gene model was derived with the aid of a computer program developed by Sutherland (1986) at the N.V.R.S.

Plant lines which are resistant solely to a single isolate identify unique single factors that may be controlled by a minimum of one gene for resistance, and that this is matched by a minimum of one avirulence gene in the mildew. In Table 4.2 it can be seen that plant lines 1c, 1f, 1m, 1p, 3g, 19b and 24f, each show resistance to only one isolate, indicating that 7 different resistance genes (labelled R1 to R7) and 7 matching avirulence genes (A1 to A7) are required to explain the reactions. Removing the reactions that can be explained by resistance genes R1 to R7 leaves Table 4.4 consisting of the, as yet, unexplained reactions. From Table 4.4 it can be seen that plant lines 5a and 4a possess resistance reactions that can be explained by the action of two more resistance genes: R8 and R9, with their matching avirulence genes: A8 and A9. The reactions explained by these two genes can then be eliminated to leave the reactions given in Table 4.5.

Plant line 6b is resistant to every isolate and so its reaction may either be determined by the action of several genes or, one gene for which all the isolates have matching avirulence. It is possible

TABLE 4.3. Resistance factors assigned to each plant line.

1 = Resistance to isolate G1	10= Resistance to isolate N1
2 = " " " G2	11= Resistance to isolate N2
3 = " " " G6	12= Resistance to isolate N4
4 = " " " G7	13= Resistance to isolate N5
5 = " " " G8	14= Resistance to isolate N6
6 = " " " G9	15= Resistance to isolate N7
7 = " " " G10	16= Resistance to isolate N8
8 = " " " G11	17= Resistance to isolate N9
9 = " " " G12	18= Resistance to isolate N11

Plant line	Resistance factors.
1c	2
1e	None
1f	10
1g	None
1h	18
1i	None
1m	12
1n	18
1s	None
2a	None
2d	None
2e	9
2i	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
3f	2,9,12,18
3g	9
4a	1,2,15,18
4h	1,2,3,4,6,9,11,16,18
5a	1,6
6b	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
6d	1,6,9
6f	None
7b	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
7c	18
7d	1,2,4,5,7,8,10,11,12,13,14,15,16,17,18
7f	None
8a	None
8g	None
9a	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
9c	1,2,4,5,7,8,10,11,12,13,14,15,16,17,18
9d	1,2,3,4,6,7,9,10,11,12,13,14,15,16,17
9g	2,3,4,6,7,9,10,11,12,13,15,16,17
10j	10,11,12,13,14,15,16,17,18
11a	None
11e	None
11i	1,2,4,7,8,10,14,15,16,17
14b	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
15c	None
15j	None
16d	None
16f	1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18
17h	9
18i	None
19b	17
23f	6
23g	None
23i	None
24f	1
24j	None

TABLE 4.4. Reactions remaining when resistance to genes 1 to 7 accounted for.

Plant line	Isolate G6	G7	G8	G9	G10	G11	N2	N5	N6	N7	N8
4a										R	
4h	R	R		R			R				R
5a				R							
6b	R	R	R	R	R	R	R	R	R	R	R
6d				R							
6f										R	
7d		R	R		R	R	R	R	R	R	R
9d		R	R		R	R	R	R	R	R	R
9g	R	R		R	R		R	R		R	R
10j							R	R	R	R	R
11i		R			R	R			R	R	R
23f				R							

TABLE 4.5. Reactions remaining after resistance to genes 1 to 9 accounted for.

Plant line	Isolate. G6	G7	G8	G10	G11	N2	N5	N6	N8
4h	R	R				R			R
6b	R	R	R	R	R	R	R	R	R
7d		R	R	R	R	R	R	R	R
9g	R	R		R		R	R		R
10j						R	R	R	R
11i		R		R	R			R	R

TABLE 4.6. Simplified version of Table 4.5 to show that 5 genes are required to explain the remaining resistant reactions.

Plant line	Isolate G6	G8	G10	G11	N6
4h	R				
7d		R	R	R	R
9g			R		
10j					R
11i			R	R	R

Table 4.7 14 gene model

		G1	G2	G6	G7	G8	G9	G10	G11	G12	N1	N2	N4	N5	N6	N7	N8	N9	N11	
	1c		R																	R2
	1e																			0
	1f										R									R4
	1m												R							R5
	1p																		R	R7
P	3f		R							R			R						R	R2 R3 R5 R7
L	3g									R										R3
A	4a	R	R													R				R1 R2 R9
N	4h	R	R	R	R		R			R	R	R					R		R	R1 R2 R3 R4 R7 R8 R10
T	5a	R					R													R1 R8
	6b	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R	R1 R2 R3 R4 R5 R6 R7 R8 R9 R10 R11
L	6d	R					R			R										R1 R3 R8
I	6f		R										R			R			R	R2 R5 R7 R9
N	7d	R	R		R	R		R	R		R	R	R	R	R	R	R	R	R	R1 R2 R4 R5 R6 R7 R9 R11
E	9d	R	R		R	R		R	R	R	R	R	R	R	R	R	R	R	R	R1 R2 R3 R4 R5 R6 R7 R9
	9g		R	R	R		R	R		R	R	R	R	R		R	R	R		R2 R3 R4 R5 R6 R8 R9 R12
	10j										R	R	R	R	R	R	R	R	R	R4 R5 R6 R7 R9 R13
	11j	R	R		R			R	R		R				R	R	R	R		R1 R2 R6 R9 R14
	19b																	R		R6
	23f		R				R													R2 R8
	24f	R																		R1
		A1	A2	A10	A10 A11 A12 A14	A11	A8	A11 A12 A14	A11 A14	A3	A4	A10 A11 A12 A13	A5	A11 A12 A13	A11 A13 A14	A9	A10 A11 A12 A13 A14	A6	A7	

R = Resistant reaction
A_1 to A_{12} = Avirulence genes
R_1 to R_{12} = Resistance genes

that plant line 6b has the genes of plant lines 4h and 7d as it gave a resistant reaction where one or both of these plant lines also gave a resistant reaction.

It is difficult to see immediately from Table 4.5 how many more genes are needed to explain the remaining reactions, but the computer analysis (Sutherland, 1986) revealed that a minimum of five more genes is probably required. Table 4.6 gives a simpified version of the reactions produced by the 5 additional genes and it can be seen that 5 genes do adequately explain the reactions.

Table 4.7 represents the complete 14 gene model, with the resistance and avirulence genes assigned to the appropriate plant lines and isolates.

4.4. Discussion

The proposed gene model estimated that a minimum of 14 genes were required to explain the phenotypes of the 21 different plant lines and 18 isolates. Fourteen genes can give rise to 2^{14} or 16,384 different resistance or virulence phenotypes. The potential variability in the pathosystem is enormous. Harry (1980) concluded that eight genes determined the reaction patterns of the host lines and isolates she worked with. Genes identified by Harry did not appear to be the same as any of the genes proposed in the isolates used in the present study. We may then assume that at least 22 genes have been identified in the pathosystem, which may give rise to 2^{22} or 4,194,304 possible phenotypes.

The number of genes identified only represents the minimum number of genes involved. The number of genes detected depends on the particular isolates and plant lines used for testing. More than one gene may be involved in conferring resistance or avirulence reactions that appear to be produced by only one gene. Modifier genes such as those found by Johnson and Law (1967) which increase or decrease the effects of resistance genes in wheat varieties Hope and Chinese spring to *Puccinia graminis tritici* may be operating in the system. The modifier genes may mask resistance or virulence genes or make it appear that more genes are operating than is really the case.

The 14 gene model presented in Table 4.7 shows that all the isolates are complex as far as virulence is concerned, having several virulence genes. Harry (1980) also found this to be so, however, all eight of her isolates had only one avirulence gene. This is not necessarily the case in the present study. Twelve of the 18 different isolates did appear to have only one avirulence gene but the remaining

isolates were postulated to possess 2, 3, 4 and up to 5 avirulence genes. Since each isolate appears to have relatively few avirulence genes in comparison to virulence genes, it is tempting to postulate that avirulence genes in the pathogen recognise resistance genes in the host and thereby elicit a resistant reaction. Ellingboe (1982), on the basis of his studies on the interactions between Erysiphe graminis hordei and barley genotypes, together with evidence from other pathosystems, concluded that the simplest and most probable explanation of the gene-for-gene relationship is that recognition occurs between avirulence and resistance genes to elicit a resistant reaction.

In cereal crop/ powdery mildew pathosystems the frequency of recognised virulence genes tend to reflect the amount of usage of the appropriate resistant cultivars (Wolfe and Schwarzbach, 1978a). As the acreage of cultivars with combined resistance increases then so does the frequency of the corresponding complex virulence in the mildew. However, it has been noted, for example, that in the U.K. combined virulence for Mlg and Mlas genes in Erysiphe graminis hordei populations did not increase as quickly as expected (Wolfe and Schwarzbach, 1978b). Likewise combined virulence for resistance genes Mlas and Mla4/7 in European barley powdery mildew populations occurred with low frequency. These races with combined virulence tended not to do well on Mlas or Mla4/7 cultivars.

In E. graminis avenae populations, the evolution of complex resistance has been rapid in the U.K. This may be a reflection of the relatively low acreage of oats grown in the U.K. and likewise the oat powdery mildew population would also tend to be small. Consequently, there is more likely to be selection for complex virulence, providing the pathogen with the necessary genes for colonization of several cultivars, thereby ensuring its survival (Wolfe and Schwarzbach,

1978b). In barley the acreage grown is much greater, therefore there may be a greater selective advantage for adaptation in the pathogen to particular cultivars that do not necessarily have combined resistance.

The acreage of particular cereal varieties grown will effect the frequency of various virulence phenotypes in the pathogen. In some pathosystems, one particular race of the pathogen becomes far more common than any other. For example race 56 of Puccinia graminis tritici was found to constitute 97% of the stem rust population, in Kansas, U.S.A., in some years (Browder, 1966). This is not always the case in crop pathosystems. Wolfe and Schwarzbach (1978b) cited an example where 18 m^3 of air were trapped in Germany. Eight isolates were obtained from the air sample, all of which were unique. In general, the cereal powdery mildew population in the atmosphere is thought to be highly heterogeneous. Only a small proportion of spores (0.1% was estimated by Gregory, 1973) actually get dispersed into the air. Once in the atmosphere, spores from different sources are likely to be thoroughly mixed. In cumulus clouds the spores are held under suitable conditions to remain viable for long periods of time and may be transported large distances (Hirst et al, 1967). Hermansen and Wiberg, (1972) have found evidence that powdery mildew conidia from Europe and the U.K were responsible for outbreaks of powdery mildew in Denmark. Wolfe and Schwarzbach (1978) found that the the virulence composition of barley powdery mildew in Germany did not significantly differ in virulence composition between sampling sites over 100 km apart. However, this was referring to the total virulence in the population since bulk populations of isolates were compared not individual isolates. In the U.K (Bennett and van Kinis, 1983) found the frequency of various virulence types of wheat powdery mildew tended to vary from region to region. This was probably a reflection of the particular wheat varieties grown in those regions. The

frequency of pathogen races with complex virulence has a tendency to increase as the acreage of varieties possessing the corresponding resistance genes in combination increases. This has led to fears that 'super races', possessing virulence to every resistance gene in the host, may develop.

The mode of reproduction of the pathogen may have a profound effect on the variability and race structure of the pathogen population. Roelfs and Groth (1980) studied intensively the virulence of two populations of Puccinia graminis tritici with respect to 16 virulence loci. One stem rust population was predominantly the result of asexual reproduction, whereas the second had the opportunity of reproducing sexually as the alternate host 'barberry' (Berberis vulgaris) had not been eradicated from the area. In the asexual population, 2,377 isolates were studied and only 17 different phenotypes with respect to the 16 virulence loci were detected. In the sexual population, there appeared to be greater variability, 100 different phenotypes were detected in the 426 isolates tested. The asexually produced groundsel powdery mildew population was far more variable than that of the asexually produced stem rust population. All the isolates from the asexually produced stem rust population were complex, the majority having about 10 of the virulence genes. None of the races had less than 5 virulence genes. This is thought to be a reflection of the matching resistance genes that were common in the wheat varieties grown in the sampling area. This also demonstrates that asexual populations can rapidly accumulate virulence genes. Unnecessary virulence genes, matching resistance gene combinations not grown in the areas sampled, occurred in both asexual and sexual populations of stem rust. This was a little suprising in the sexual population since one would expect selection against unnecessary virulence genes when they can become singly exposed after

recombination. In the asexual population unnecessary virulence genes may remain longer because there is selection for the genotype as a whole which may also carry necessary virulence.

The frequencies of various virulence genes were approximately random in the sexually produced stem rust population. Only phenotypes carrying many or very few virulence genes were rarer than expected. In the asexual population certain gene combinations were more frequent than expected, suggesting that these genotypes had a selective advantage.

The composition of pathogen populations that colonize wild hosts has been little studied. Dinoor (1977) found that the population of oat crown rust colonizing wild oats was highly heterogeneous. Over 3 years, 91 different races were collected. These races were also capable of colonizing oat crop cultivars so the results may have been biased by a large build up of particular races on certain crop cultivars.

In a more natural situation, as in the groundsel/mildew pathosystem, the host is far more heterogeneous than in the crop situation and this is certainly reflected in the pathogen population. Eighteen out of 24 isolates were found to be different. Some isolates were similar ie G3 = G7, G4 = G10, G5 = G11, N8 = N10 and N12, and G8 = N3. This suggested that although the population was highly heterogeneous some mildew isolates may be more common in the population. However, the large number of possible phenotypes makes it difficult to say whether this is really so, since suitable groundsel lines that distinguish these isolates may not have been tested.

The large number of phenotypes detected also made it difficult to compare the Glasgow and N.V.R.S mildew populations. Only one isolate phenotype ie N3 and G8 was common to both populations. One could say that as all the other isolates were different, then the two

populations were different. However, with the large number of phenotypes possible it is just as likely that as many different isolates would be collected from one mildew population.

The large number of different phenotypes in the mildew population and the fact that the isolates tend to be complex suggests that selection in the pathogen has been for each isolate to be able to colonize the majority of the groundsel plants. Only 25% of the possible 900 reactions between the 18 unique isolates and 50 groundsel lines were resistant. It must be borne in mind that the 50 inbred groundsel lines were deliberately chosen for testing as they possessed a high proportion of individuals that were resistant to Harry's isolates and they were collected from all around the U.K, so that pathogen and host would not necessarily be adapted to each other.

Although the isolates are complex, there appears to be no evidence of the presence of a super race capable of overcoming every gene for resistance in the host. If a super race does exist then it does not seem to have any great selective advantage or it would have been detected in the mildew isolates collected or at least have become apparent on the 'trap' groundsel lines discussed in Chapter 5.

CHAPTER 5 MONITORING THE MILDEW POPULATION.

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5.0. MONITORING THE MILDEW POPULATION AT N.V.R.S.

5.1. Introduction.

The most extensive studies of virulence phenotypes in pathogen populations have been carried out on pathogens of cereal crops. Most of these studies have taken the form of race surveys to detect virulence phenotypes which may be a potential threat to resistant varieties of the host. The U.K. cereal pathogen virulence survey is carried out annually on a range of cereal pathogens including mildews and rusts. Quantifying the results of such surveys is difficult as the races detected and their frequencies are dependent upon the chosen sampling method.

Collections from scattered localities and cultivars approach the most representative samples of a pathogen population (Jeger and Groth, 1985). However, even these collections may be biased. There may be a tendency for certain pathogen virulence phenotypes to be detected more often than they really occur in the pathogen population if collections are only made from certain cultivars and not all the cultivars grown in the region are sampled proportionately.

Other sampling methods involve collections of the pathogen of interest from cultivars containing specific resistance phenotypes, often referred to as 'trap cultivars'. Obviously, only those races that are capable of colonizing the trap cultivars will be detected. Browder (1966) found using trap cultivars that the frequencies of some races of Puccinia graminis tritici were significantly different from those determined using more random methods of isolate collection. Generally 'trap cultivars' tend to detect matching virulence using smaller sample sizes than are found in random samples, so, they are

useful for detecting rarer virulence phenotypes. The static nurseries of small plots of trap cultivars used by Priestley (1978) had the advantage that they sampled the pathogen population for a long period of time and at all stages of development of the host. Mobile nurseries of seedlings of relevant differential lines used by Eyal et al (1973) were useful as they could be used for short periods of time but repeatedly over a season to see how the pathogen population changed. Schwarzbach (1979) developed a method whereby a spore trap was used for collecting large volumes of air directly on to host leaves which could then be incubated under standard environmental conditions, making it easier to quantify results. Race collections may be quantified by, for example, counting the numbers of lesions on the trap cultivar leaves in comparison to lesions on cultivars thought to be susceptible to every race. However, differences in leaf tissue surface area, plant architecture and age may introduce inaccuracies (Jeger and Groth, 1985).

For this work it was decided that it would be desirable to determine whether the mildew isolates collected randomly (i.e. the isolates assessed in Chapter 4) were in fact representative of the most common isolates in the population. Small 'trapping nurseries', using one plant of each of ten lines known to have a range of resistance phenotypes were exposed to the natural population of mildew at N.V.R.S. The nurseries were replaced every month to observe any changes in the population over a period of time. It was also hoped that the nurseries would detect any virulence in the population capable of colonizing the plant lines previously thought to have resistance to all isolates. By collecting the mildew from the resistant lines and testing it on other lines more could be learnt about the rarer virulence phenotypes.

5.2 Experiment 1. Mildew 'trapping'.

5.2.1. Materials and methods.

Groundsel lines 4h, 6b, 7b, 7d, 8a, 8g, 9a, 9c, 9g and 11i were grown as required in mildew free cabinets. When the plants were about five weeks old, one plant from each plant line was placed in a polythene tunnel. It had been noted that powdery mildew developed more frequently on plants kept in glasshouses or polythene tunnels than those that were kept in the open. The plants were replaced at monthly intervals from January 11th 1984 until December 8th 1984. The old plants were discarded before the fresh set was placed in the tunnel. The flower heads were cut from the plants before they seeded to avoid contaminating the N.V.R.S. groundsel population. The side flaps of the polythene tunnel were kept open throughout, to allow free circulation of air and access for incoming mildew spores.

Eight leaf segments were cut from each plant line 2, 3 and 4 weeks after the plants had been placed in the tunnel. The leaf segments were placed in Petri dishes on benzimidazole supplemented agar and incubated for 14 days at 15°C. The approximate percentage of leaf segment area colonized with mildew was recorded.

5.2.2. Results.

Mildew was not found on any groundsel at N.V.R.S. until August in 1984. This late appearence was reflected by the fact that the groundsel in the polythene tunnel showed no signs of mildew infection until September. The mean percentage leaf area colonized by mildew on each plant line at each sampling period is given in Table 5.1.

Table 5.1 Mean % leaf area colonized on incubated detached leaves from 'trap' groundsel lines

Plants first exposed	Plant line	Number of weeks plants exposed 2wk Mean	2wk Se	3wk Mean	3wk Se	4wk Mean	4wk Se
10. 9.84	4h	0.1	0.13	0.3	0.16	1.4	1.24
	6b	0.0	0.00	0.0	0.00	0.0	0.00
	7b	0.0	0.00	0.1	0.13	0.0	0.00
	7d	0.0	0.00	0.3	0.16	0.0	0.00
	8a	5.6	3.05	12.0	7.23	19.0	7.09
	8g	0.1	0.13	15.8	5.58	29.4	12.41
	9a	0.0	0.00	0.3	0.16	0.1	0.13
	9c	0.0	0.00	0.0	0.00	0.1	0.13
	9g	0.0	0.00	0.0	0.00	0.0	0.00
	11i	0.0	0.00	0.1	0.13	3.4	3.10
8.10.84	4h	1.9	1.87	6.3	6.25	8.1	4.11
	6b	0.0	0.00	0.0	0.00	0.0	0.00
	7b	0.0	0.00	0.0	0.00	0.0	0.00
	7d	0.0	0.00	0.0	0.00	0.0	0.00
	8a	8.9	2.39	23.1	6.61	23.9	6.26
	8g	0.0	0.00	0.0	0.00	17.6	7.34
	9a	0.0	0.00	0.0	0.00	0.0	0.00
	9c	0.0	0.00	0.0	0.00	0.0	0.00
	9g	0.0	0.00	0.0	0.00	0.1	0.13
	11i	0.0	0.00	0.0	0.00	1.3	1.25
8.11.84	4h	0.0	0.00	0.0	0.00	0.0	0.00
	6b	0.0	0.00	0.0	0.00	0.0	0.00
	7b	0.0	0.00	0.0	0.00	0.0	0.00
	7d	0.0	0.00	0.0	0.00	0.0	0.00
	8a	0.0	0.00	2.5	1.64	0.0	0.00
	8g	0.0	0.00	0.0	0.00	0.0	0.00
	9a	0.0	0.00	0.0	0.00	0.0	0.00
	9c	0.0	0.00	0.0	0.00	0.0	0.00
	9g	0.0	0.00	0.0	0.00	0.0	0.00
	11i	0.0	0.00	0.0	0.00	0.0	0.00

Se = Standard error
(only sets of data from sampling dates where mildew was detected are included)

Plant lines 8a and 8g, previously found to be susceptible to all 24 isolates, were the first to show relatively heavy levels of infection. Plant line 4h, previously found to be resistant to many but not all isolates, showed low levels of mildew infection at the same time. One week later plant lines 7b, 7d, 9a and 11i started to show low levels of infection and a week later plant line 9c also succumbed to low levels of infection. These data suggest, as expected, that virulence to plant lines such as 8a and 8g is common in the mildew population, while virulence to plant lines previously found to be resistant to the 24 isolates is present but probably at a lower frequency.

In October there were indications that mildew was beginning to decline and components of the population with virulence for the more resistant plant lines were detected less often. Mildew capable of colonizing line 4h remained relatively abundant but by November only plant line 8a became infected. In December mildew was not detected on any of the groundsel lines and it was assumed that weather conditions were no longer conducive to conidial dispersal or germination and the study was terminated.

5.3. Experiment 2 Virulence in the mildew population.

5.3.1. Introduction.

Experiment 1 demonstrated that there were mildew races in the mildew population at N.V.R.S. capable of colonizing groundsel lines which were resistant to some or all of the 24 isolates G1 to G12 and N1 to N12 tested earlier. An experiment was designed to determine the virulence characteristics of the mildew population colonizing a

particular plant line. Also, it was hoped to determine whether plants were colonized by one or several different races of mildew at any one period in time.

5.3.2. Materials and methods.

Tests were carried out whenever enough mildew inoculum could be obtained from any of the the plant lines of the previous investigation on the sampling dates, 1.10.84, 8.10.84, and 5.11.84. The plant lines were 8a and 8g (susceptible to all 24 isolates), 4h, 7d, and 11i (resistant to some of the isolates) and 6b, 7b and 9a (resistant to all 24 isolates). The inoculum collected from the 'trap lines' was tested on ten plant lines (4h, 6b, 7b, 7d, 8a, 8g, 9a, 9c, 9g and 11i). The population of conidia produced on the leaf segments from a particular plant line from a particular sampling date were gently dusted into a small clean Petri dish with a paint brush and mixed. The conidia were then transferred, using the brush, on to six leaf segments from each of the 10 plant lines and incubated at 15^{o}C for 9 days. The infection scores 0, 1, 1.5, 2.5, 3 and 4 were recorded.

Some of the above tests produced abundant conidia and it was decided that some mildew populations would be examined in greater detail on the 50 inbred groundsel lines. The mildew populations from all the sampling dates originating from each of the plant lines 8a, 8g, 7d and 9a were bulked together separately and used to inoculate the 50 inbred groundsel lines. Six leaf segments of each plant line were tested with each of the mildew populations and incubated as before.

5.3.3. Results.

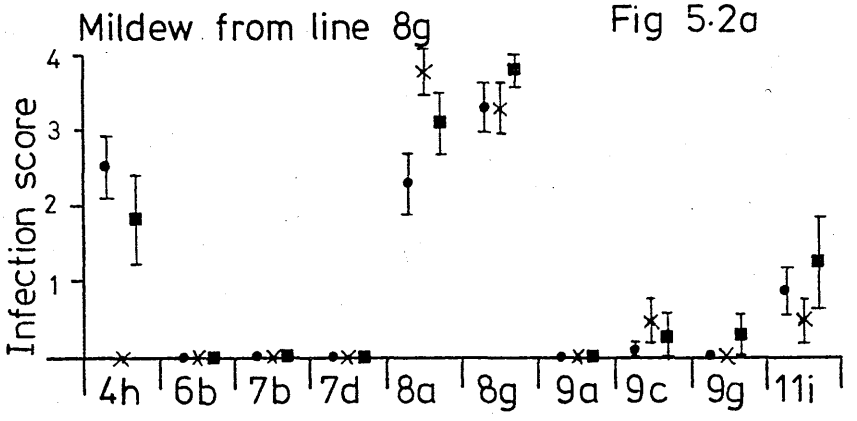
Only a few plant lines in Experiment 1. produced sufficient conidia to test ten plant lines. The mean infection scores produced on each of the 10 plant lines by each mildew population are given in Appendix Table 5.1. and Figs 5.1a-d and 5.2a-d.

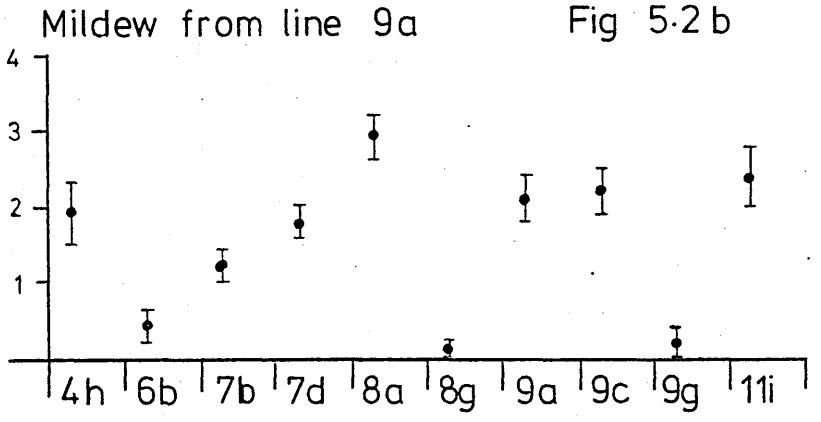
The mildew populations from plant lines 8a and 8g were both similar in that they were capable of colonizing plant lines 4h, 8a, 8g, 9g and 11i. However, mildew from plant line 8a was also found to colonize plant lines 7d and 9c at a low level indicating a slight difference between the two populations. Both populations were incapable of colonizing the plant lines 6b, 7b and 9a, which were resistant to all isolates studied previously. This suggests that mildew with virulence to lines 6b, 7b and 9a does not occur commonly on plant lines lacking the resistances present in these lines, probably due to a lack of adaptation.

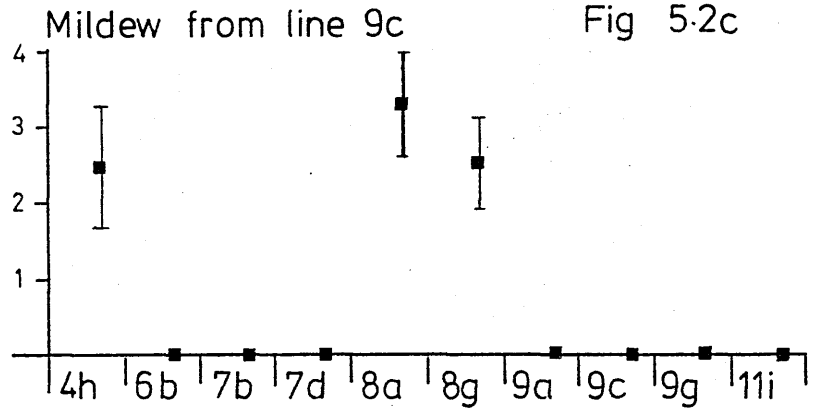
The mildew populations from plant lines 7b and 7d were found to be capable of colonizing all the other plant lines except 8g. However, their mean infection scores on each plant line tended to be lower than the mean infection scores obtained with mildew populations from plant lines 8a and 8g suggesting that although they were capable of attacking most plant lines they were less aggressive. This hypothesis is also supported by the fact that although mildew found on resistant plants was capable of colonizing plant line 8a it was rarely detected on plant line 8a during the course of this experiment. The results also indicate that plant line 8g shown earlier to be susceptible to all 24 mildew isolates, posssessed specific resistance to some mildew races and was therefore different from line 8a.

Mildew from plant line 9a was also capable of colonizing all 10 plant lines except for 8g, but tended to be just as aggressive as

Fig 5·2a-d. Mean infection scores produced by each mildew population on each plant line, continued.







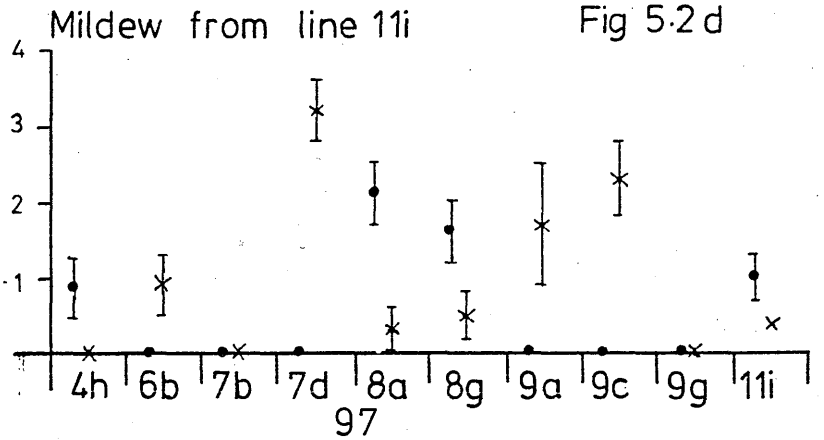
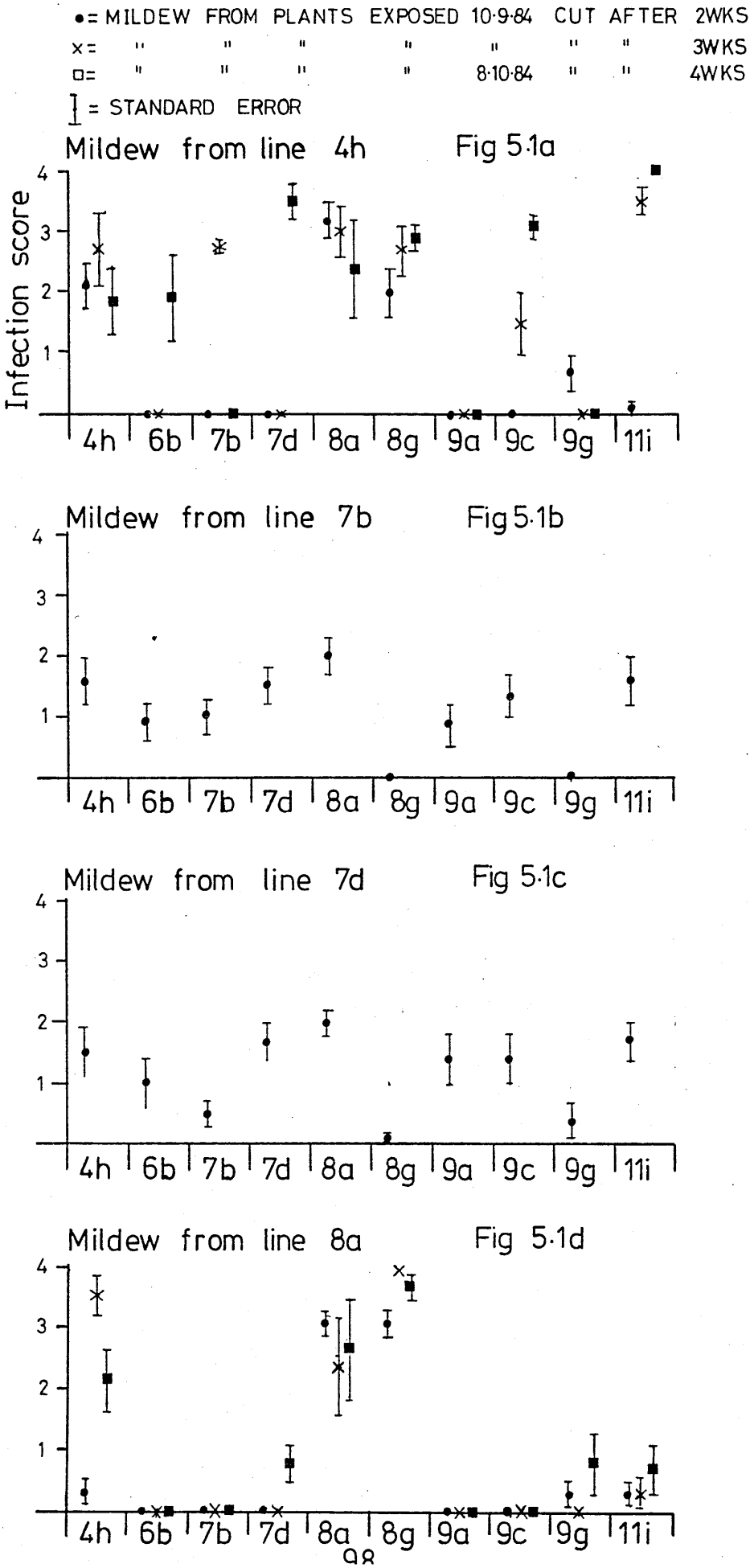


Fig 5.1a-d. Mean infection scores produced by each mildew population on each plant line.



mildew from the susceptible lines indicating that not all mildew races capable of colonizing the resistant plants lack aggressiveness.

Mildew from plant line 4h was capable of colonizing all the plant lines except for line 9a. It is most probable that 4h supported a mixture of mildew races, including races with virulence to lines 8a and 8g, as well as races with virulence to lines with a phenotype similar to 6b and 7b. Since virulence to lines 6b, 7b, 7d, 9c and 11i was not detected at every sampling date this indicates that the mildew population on line 4h was a mixture of races the composition of which changed with time.

The mildew found on plant line 11i was capable of infecting all 10 plant lines except 7b and 9g, but the results varied between two sampling dates, indicating that this was a mixed population particularly with respect to virulence on plant line 4h.

The mean infection score obtained for each bulked up mildew population originating from plant lines 8a, 8g, 7d and 9a on each of the 50 inbred groundsel lines is given in Table 5.1. The mildew populations from plant lines 8a and 8g were generally capable of colonizing all the plant lines except for some of those already found to possess resistance to all 24 isolates previously tested, eg plant lines 2i, 7b, 9a, 14b and 16f. The race composition of the two mildew populations was probably different since mildew from line 8a infected plant lines 3f, 6b, 7d and 9c whilst mildew from line 8g did not.

The mildew populations originating from plant lines 7d and 9a were found to be avirulent on several plant lines (eg 1e, 1n, 1s and 7a), that were susceptible to all 24 of the isolates tested earlier. These plant lines, therefore, possess resistance to some of the races thought to be rare in the mildew population.

TABLE 5.2. The mean infection scores produced by the mildew populations from groundsel lines 7d, 8a, 8g and 9a on the 50 inbred groundsel lines.

	Origin of mildew population.			
Plant line	7d	8a	8g	9a
1c	0.58	2.75	1.38	0.63
1e	0.00	4.00	3.25	0.00
1f	3.38	3.25	4.00	2.95
1g	1.33	4.00	3.33	3.17
1h	2.00	3.25	1.50	1.92
1i	3.00	3.08	3.00	2.67
1m	0.00	0.58	3.58	0.00
1n	0.00	2.25	0.54	0.00
1p	4.00	2.67	4.00	3.83
1s	0.00	1.75	3.08	0.00
2a	3.58	4.00	2.00	3.50
2d	3.83	3.33	3.17	3.33
2e	0.67	2.00	1.50	1.92
2i	2.75	0.00	0.00	1.38
3f	0.67	1.17	0.50	1.67
3g	3.17	2.50	2.83	2.50
4a	1.63	2.92	1.83	1.75
4h	2.67	4.00	1.08	3.50
5a	2.17	2.42	3.25	1.50
6b	2.67	1.21	0.00	2.33
6d	0.00	4.00	2.42	0.00
6f	0.00	1.13	2.08	0.00
7a	0.00	4.00	2.25	0.00
7b	2.08	2.50	0.00	0.00
7c	3.00	3.75	2.75	2.83
7d	2.17	2.21	0.00	1.92
7f	0.00	3.83	2.21	0.00
8a	3.50	3.83	4.00	3.42
8g	0.00	3.83	2.83	0.25
9a	1.75	0.00	0.00	1.58
9c	3.33	3.75	0.08	3.58
9d	3.42	1.08	0.25	3.25
9g	0.38	0.25	0.33	0.00
10j	1.67	3.42	2.50	2.67
11a	2.42	2.50	3.17	2.83
11e	0.00	3.33	3.75	0.00
11i	4.00	2.75	0.33	4.00
14b	2.17	0.00	0.00	3.17
15c	3.83	4.00	2.42	3.83
15j	2.25	3.50	2.25	4.00
16d	0.00	0.13	3.67	0.13
16f	2.50	0.00	0.00	2.83
17h	1.79	2.25	3.58	2.08
18i	2.50	3.08	3.00	2.33
19b	2.08	3.83	3.25	2.92
23f	0.75	2.75	2.50	1.21
23g	1.83	4.00	3.33	2.50
23i	1.33	3.83	1.58	2.13
24f	0.00	2.08	4.00	0.00
24j	3.83	2.25	2.08	2.58

5.4. Discussion.

A 'super race' capable of colonizing all the groundsel lines was not detected using the 'trap groundsel lines'. Likewise a groundsel line with resistance to all the mildew isolates was not detected. Groundsel lines thought to be resistant to all mildew isolates eventually succumbed to infection. Line 6b, showed no infection during the mildew 'trapping', but previous observations of natural mildew infections in glasshouses showed that there was mildew in the population capable of growing on 6b. The mildew traps detected mildew populations (or isolate mixtures) capable of colonizing plants thought to be resistant to all previously tested mildew isolates. However, these mildew populations were not capable of colonizing some lines that were previously thought to be susceptible to all isolates. These mildew populations were not as common on the lines thought to be highly susceptible to every isolate even though further tests showed that they were capable of colonizing and reproducing on those lines. This suggests the mildew populations from the resistant plants did not compete well with those populations normally occurring on the susceptible lines, indicating that some mildew isolates may become better adapted to some groundsel plants than to others.

The two populations of mildew from susceptible and mainly resistant plants appeared to be different with respect to virulence. Roelfs and Groth (1980) observed a similar situation in an asexually reproducing stem rust (*Puccinia graminis tritici*) population. Virulence phenotypes tended to fall into certain groups of genetic similarity, separated from each other by larger genetic differences. Effectively, two different stem rust genotypes, either differed slightly when they were members of the same group, or differed greatly

when they were members of different groups. This is to be expected where little or no recombination takes place between components of a population.

The evidence also suggests that the populations of mildew on the plants were composed of a mixture of races with different phenotypes and that the composition may change with time.

Harry (1980) was able to detect more resistance phenotypes in the groundsel lines than those detected by the isolate collections in Chapter 4. This was probably because her isolates were obtained from plants already known to have resistance and were more likely to be rare isolates, which seem to be capable of distinguishing additional resistance phenotypes. The evidence from Experiment 2, supports this hypothesis, since mildew from the resistant lines 9a and 7d was able to detect resistance in lines previously thought to be susceptible to all isolates.

The mildew 'trapping' technique could be useful for rapidly detecting the presence of particular races of the pathogen, if suitable differential groundsel lines were available. This would provide a feasible way of carrying out further studies on the pathogen population, although it would have the disadvantage that the only races that could be detected would be those capable of colonizing the differential groundsel lines.

CHAPTER 6. EVIDENCE FOR PARTIAL RESISTANCE TO ERYSIPHE FISCHERI IN GROUNDSEL.

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6.0. EVIDENCE FOR PARTIAL RESISTANCE TO ERYSIPHE FISCHERI IN GROUNDSEL.

6.1 Introduction.

During the early stages of this work it was observed that some groundsel line/isolate combinations produced relatively low infection types. The reactions were not classed as resistant because they produced a mean infection score above 0.6. However, they indicated some degree of resistance since high scores of 3 or 4 in any particular test were rare. Relatively low levels of susceptibility have also been noted in some crop cultivars and the terms partial resistance or incomplete resistance have been applied. The characteristics of partial or incomplete resistance in groundsel to Erysiphe fischeri appeared to include the relatively slow production of fewer and smaller colonies in comparison with those produced in highly susceptible reactions. These characteristics are often observed where partial resistance occurs in crop cultivars. For example, the expression of partial resistance in the barley variety Vada to Puccinia hordei was reported to be characterized by the production of fewer and smaller rust pustules than those produced on highly susceptible varieties (Clifford and Clothier, 1974). The relatively high resistance of some wild relatives of oat to Erysiphe graminis avenae may also be attributed to the production of relatively small colonies (Carver and Carr, 1978). Partial resistance in some oat cultivars was found to be characterized by a longer latent period, a reduction in spore production and a reduced area of leaf colonised by powdery mildew (Jones, 1978).

Incomplete or partial resistance may play an important role in

slowing down the development of an epidemic, and hence, contribute to the defence strategy of the host. Reduced sporulation rates, longer latent periods and fewer, smaller colonies contribute to a reduction in the rate of spread of an epidemic between and within plants (Parlevliet, 1979). Partial resistance has been utilized in some crop cultivars, and in some cases has been proved to be a highly useful and durable form of resistance. For example, the incomplete resistance of Cappelle-Desprez wheat to Puccinia striiformis has remained commercially useful for over 25yrs despite its widespread use (Johnson, 1983). The slow mildewing of Knox wheat to Erysiphe graminis tritici was still stable after 20yrs in 1973, (Shanner 1973a and b).

Partial resistance does not neccessarily confer stability. For instance, Joss Cambier possessed incomplete resistance to Puccinia striiformis but succumbed to an epidemic of yellow rust in 1971 and 1972 (Johnson, 1983). Incomplete resistance is often assumed to be under polygenic control or to be race non specific; this does not necessarily have to be the case, the unstable partial resistance of Joss Cambier proved to be race specific according to Johnson and Taylor, (1972).

Incomplete resistance is comparatively harder to assess than the clear cut reactions associated with complete resistance, so that methods of assessment need to be quantitative. Tests need to be repeated several times to produce a reliable mean score and to check that reduced levels of susceptibility are not just the product of environmental conditions. Often partial resistance is only expressed in a host at a certain stage of development or under a particular set of environmental conditions.

Once the actual existence of partial resistance has been established it is even more difficult to determine whether it is race

specific or race non-specific in nature. Where complete resistance is concerned, a race specific reaction is said to occur where one cultivar is resistant to a particular isolate while a second cultivar is susceptible to that isolate. The first cultivar is, however, capable of showing a susceptible reaction to another isolate. This has become known as a quadratic check, eg.

	Isolate 1	Isolate 2
Cultivar 1	-	+
Cultivar 2	+	+

The arrangement known as a reciprocal check provides stronger evidence for the presence of race specific resistance, eg.

	Isolate 1	Isolate 2
Cultivar 1	-	+
Cultivar 2	+	-

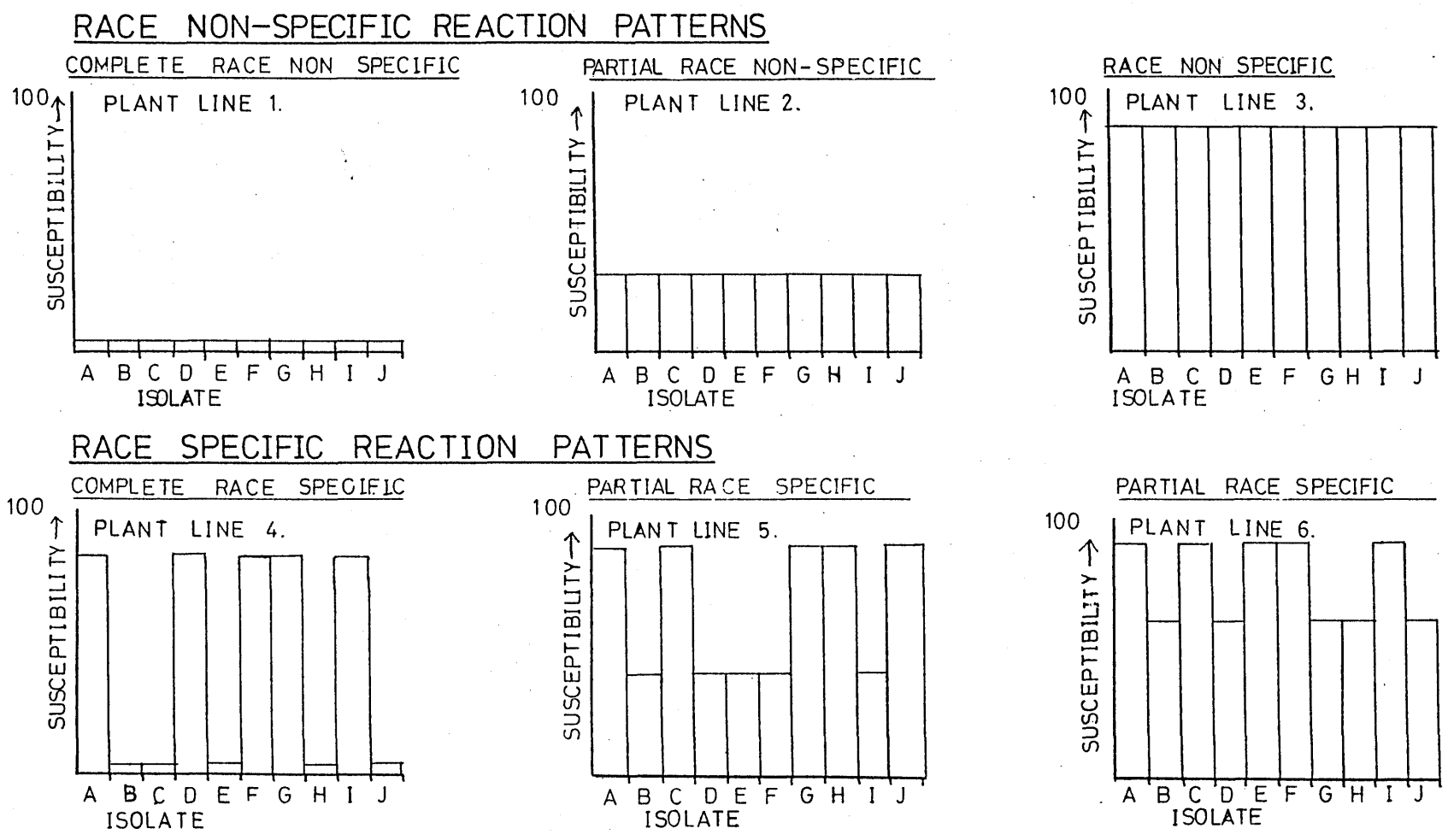
Where - = incompatible or resistant reaction, and

+ = compatible or susceptible reaction

The same principle may be applied to demonstrate the existence of a race specific partial interaction but an intermediate level of susceptibility replaces the completely resistant reaction.

Histograms of the infection scores produced with various isolates on particular cultivars may give a general picture of the type of resistance present in the host. Fig 6.1 represents the patterns produced where cultivars possess various levels of either race specific or race non-specific resistance. A complication arises where a plant line shows a combination of race specific complete, race specific partial, race non-specific complete and race non-specific

Fig 6.1. HISTOGRAMS SHOWING TYPICAL RACE NON-SPECIFIC AND RACE SPECIFIC PATTERNS.



partial resistance. The histograms that may result from different cominations of these resistance types can become difficult to interpret.

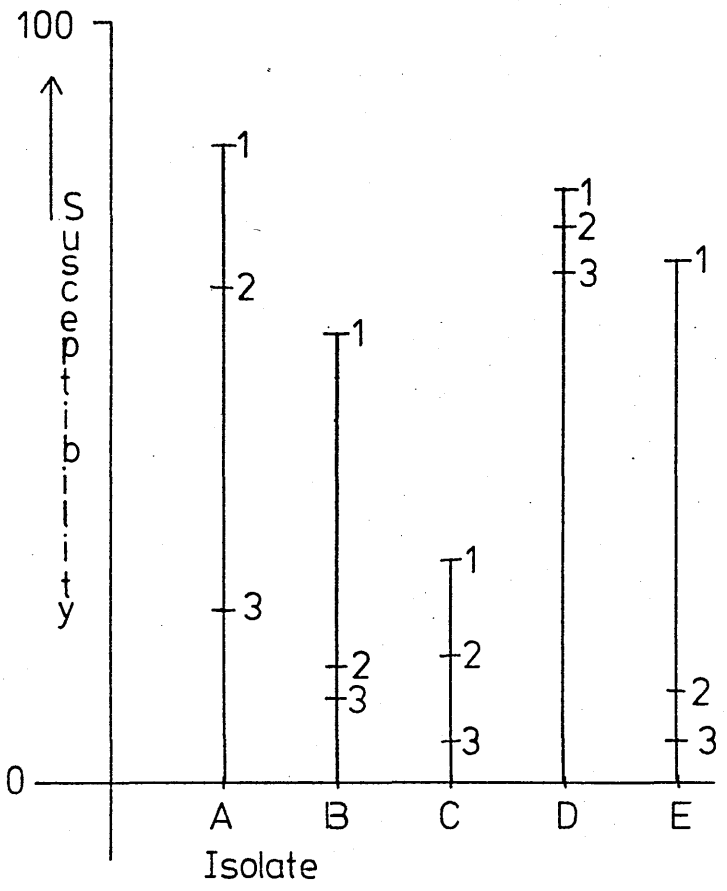
Where several isolates and host varieties are assessed an analysis of variance may be applied to the data. A race specific relationship is said to occur where a significant cultivar x isolate variance ratio is obtained (Van der Plank, 1968). However, this may not necessarily be due to race specific effects. The data may need to be examined more closely. A significant cultivar x isolate variance ratio may be produced where cultivars show different levels of susceptibility to various isolates, but in fact retain the same ranking order, which is indicative of a race non-specific relationship. Fig 6.1 shows a hypothetical example where the susceptibility of three cultivars 1, 2 and 3 show different levels of susceptibility to 5 isolates A, B, C, D and E. The reactions could be interpreted as being indicative of a race specific relationship. Cultivar 1 may produce a highly susceptible reaction with isolate A or a highly resistant one with isolate C. However, cultivar 1 is always more susceptible than cultivars 2 or 3, there is constant ranking and, therefore, evidence of a race non-specific relationship.

Scott and Hollins (1977) studied the resistance of wheat to several isolates of Cercosporella herpotrichoides and came across this problem. Analysis of variance produced a significant cultivar x isolate interaction. However, on a close examination of the results many of the cultivar reactions were constantly ranked and some of the variation could be explained by environmental effects. Ranking the reactions of cultivars to various isolates may strengthen or weaken the evidence for race non-specific resistance. Spearmans ranking correlation coefficients (Parker, 1973) may be calculated to determine whether cultivars are constantly ranked. However, where infection

Fig 6·2

Plant lines showing constant ranking but may have a significant plant line × isolate interaction.

1, 2 and 3 = Plant lines



scores are very similar whatever the isolate used, the test becomes inaccurate as scores may have very different rankings even though they are not significantly different statistically.. Ordinarily one would not regard the reactions as being different.

A second way of examining the reactions of a set of cultivars to a set of isolates, is to make paired comparisons of the relative susceptibilities to the set of isolates of each plant line in all possible pairs. The reactions of each plant line are plotted against each of the others. For example, Figs 6.3a and 6.3b represent the patterns obtained where the reactions are constantly ranked ie race non-specific and where there is no constant ranking ie race specific. Where all the points lie below or above the diagonal line, constant ranking is observed. Where the points are scattered above and below the line, the ranking is not constant.

To determine the existence and degree of partial resistance in the groundsel/ powdery mildew pathosystem, the results of several repeated tests were examined to try to obtain a reliable mean infection score. Several of the groundsel lines originally used to differentiate the mildew isolates were used as controls in the testing of the N.V.R.S. and Glasgow groundsel populations (See Chapter 7). Thus, they were tested on numerous occasions with five N.V.R.S. and five Glasgow isolates. It was hoped that the control tests could be used to detect the presence and characteristics of partial resistance. The following experiment constitutes a more detailed analysis of a proportion of the control test results.

Fig 6.3 a+b

Paired comparisons of plant line susceptibilities.

Fig 6.3a Constant Ranking
Race non-specific

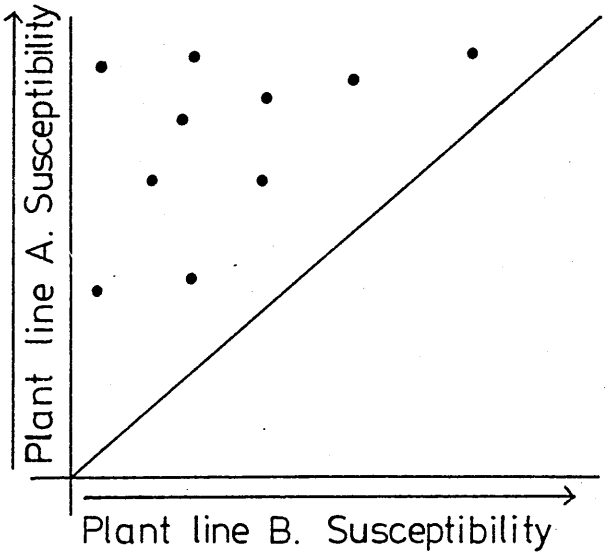
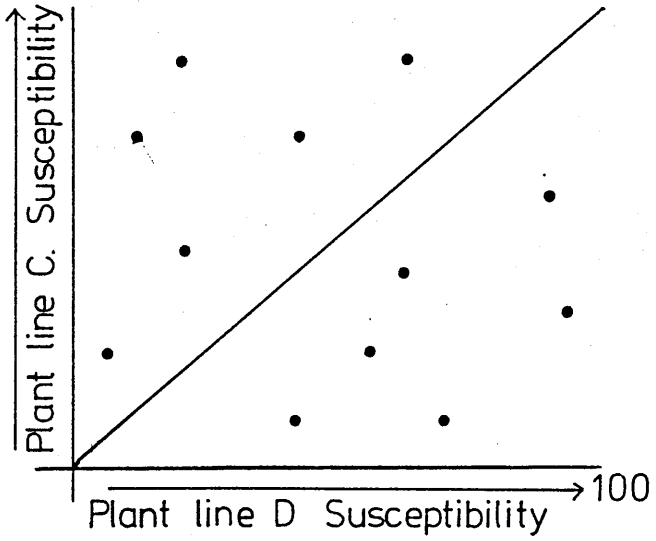


Fig 6.3b Non constant ranking
Race specific resistance



6.2. Materials and methods.

The reactions of ten plant lines (4h, 6b, 7b, 7d, 8a, 8g, 9a, 9c, 9g and 11i) to five isolates from Glasgow (G8, G9, G10, G11 and G12) were determined. Leaves from the plant lines were tested when the plants were about 6 weeks old and again when the plants were about 8 weeks old. The experiment was repeated on five separate occasions.

The reactions of an additional 10 lines (1m, 1n, 2a, 3f, 4a, 6d, 6f, 9d, 10j and 19b) to five isolates from the N.V.R.S. (N1, N4, N5, N7 and N11) were determined. Tests were carried out on leaves from plants at 6 and 8 weeks of age on two occasions. A third test used the leaves from 8 week old plants only.

6.3. Results.

The mean infection scores obtained with the five Glasgow isolates (G8, G9, G10, G11 and G12) and the five N.V.R.S. isolates (N1, N4, N5, N7 and N11) are given in Appendix Table 6.1 and Table 6.2 respectively. For the purposes of analysis of variance the infection types 0, 1, 2-, 2+, 3 and 4 were transformed to 0, 1, 1.5, 2.5, 3 and 4 respectively. Initially analysis of variance was carried out separately on the data obtained from the two sets of isolates.

Table 6.1 gives the analysis of variance for the five Glasgow isolates. The variation between the replicate experiments was not significant, therefore, combining the results of the five experiments for analysis was justified. There was a significant difference between inoculation times indicating that 56 day old plants were significantly more susceptible than 42 day old plants, the total mean infection scores were 1.69 and 1.46 respectively.

There were significant differences between the mean infection scores of the different plant lines. This was to be expected since some plant lines were completely resistant to all five Glasgow isolates while others were very susceptible. The data were then analysed excluding the results from the completely resistant plant lines 6b, 7b and 9a, and this analysis of variance is given in Table 6.2. The differences in susceptibility between the remaining plant lines were still found to be significant. The mean infection scores for each plant line are given in Table 6.3.

A similar analysis was carried out on the data obtained with the five N.V.R.S. isolates (N1, N4, N5, N7 and N11) omitting those data from the plant lines 7b and 9a which were completely resistant to all the N.V.R.S. isolates. The analysis of variance is given in Table

Table 6.1 Analysis of variance for the 5 Glasgow isolates on all 10 plant lines

Source of variation	DF	SS	MS	VR	P
Inoculation	1	19.04	19.04	18.46	NS
Inoc. Replicate	8	8.06	1.01	0.98	NS
Isolate	4	134.98	33.75	32.71	NS
Plant line	9	4383.23	487.03	472.10	0.001
Isolate, plant line	36	1402.96	38.97	37.78	0.001
Residual	441	454.95	1.03		
Total	499	6403.21	12.83		
Error between dishes	500	284.08	0.57		
Error within dishes	2000	527.00	0.26		
Grand Total	2999	7214.29			

NS = Not significant DF = Degrees of freedom SS = Sums of squares
MS = Mean square VR = Variance ratio P = Probability

Table 6.2 Analysis of variance using the 5 Glasgow isolates but omitting lines 6b, 7b and 9a

Source of variation	DF	SS	MS	VR	P
Inoculation	1	27.89	27.89	19.31	NS
Inoc. Replicate	8	10.90	1.36	0.94	NS
Isolate	4	189.51	47.38	32.81	NS
Plant line	6	289.95	469.99	325.45	0.001
Isolate, plant line	24	1348.31	56.18	38.91	0.001
Residual	306	441.91	1.44		
Total	349	4838.45	13.86		
Error between dishes	350	282.58	0.81		
Error within dishes	1400	525.50	0.38		
Grand Total	2099	5646.54			

Coefficient of variation = 31.1%

Table 6.3 Mean score for plant lines 4h, 7d, 8a, 8g, 9c, 9g, and 11i with isolates G8, G9, G10, G11, G12

Isolate	Plant line						
	4h	7d	8a	8g	9c	9g	11i
G8	0.92	0.13	3.12	3.12	0.13	0.17	1.00
G9	0.00	2.09	3.40	3.22	2.30	0.36	3.53
G10	1.33	0.13	3.48	3.47	0.08	0.68	0.74
G11	3.54	0.00	3.10	3.30	0.03	0.90	0.33
G12	3.60	0.05	3.15	3.13	0.00	0.42	0.26

Table 6.4 Analysis of variance of the 20 plant lines with the 5 NVRS isolates

Source of variation	DF	SS	MS	VR	P
Inoculation	1	0.85	0.85	0.27	NS
Inoc. Replicate	3	35.01	11.67	3.73	NS
Isolate	4	27.94	6.99	2.23	NS
Plant line	17	2337.01	137.47	43.91	0.001
Isolate, plant line	68	589.88	8.67	2.77	NS
Residual	356	1114.56	3.13		
Total	449	4105.26	9.14		
Error between dishes	450	606.63	1.35		
Error within dishes	1800	996.00	0.55		
Total	2699	5707.88			

Coefficient = 51.4%

6.4. These results also revealed that there was no significant difference between the repeated experiments. However, unlike the results for the five Glasgow isolates, there was no significant difference in susceptibility between plants at 42 and 56 days of age. The mean infection scores for the plant lines with scores greater than 0 are given in Table 6.5.

A separate analysis of variance was also carried out on the data of the lines tested with both the Glasgow and N.V.R.S. isolates which gave mean scores greater than 0 to at least one isolate (Table 6.6). Again there was a significant variance ratio for the plant lines. The mean infection scores for the 7 plant lines (4h, 7d, 8a, 8g, 9c, 9g and 11i) are given in Table 6.7.

6.3.1. Evidence for race-specific partial resistance.

The variance ratio for the plant line x isolate combination was significant for the data for the 5 Glasgow isolates (Table 6.2.) and for all ten isolates on plant lines 4h, 7d, 8a, 9c, 9g, and 11i (Table 6.6.). This indicates that partial race specific resistance may be operating. For example, line 4h is significantly less susceptible to isolate G10 and G8, than to G11. Likewise, line 11i is less susceptible to isolates G10 and G8 than to G9 (See Table 6.3.).

The mean scores of each plant line produced with each isolate it was tested with are presented as histograms in Figs 6.4a-d to 6.8a+b. The histograms give a clear presentation of the different patterns of infection types for each plant line. They provide similar information to that found with analysis of variance. There is evidence for race-specific partial resistance since some plant lines show a low level of susceptibility to some isolates but are highly susceptible to others. For example, line 4h shows a significantly higher level of susceptibility to isolate G11 than to isolate N7, G10 or G8 (Fig

Table 6.5 Mean infection scores of 20 plant lines with NVRS isolates omitting completely resistant lines 7b and 7d

Isolate	Plant line								
	1m	1n	2a	3f	4a	4h	6b	6d	6f
N1	2.67	2.40	2.72	0.73	1.88	0.58	0.08	2.20	2.08
N4	0.62	1.08	2.80	0.78	0.92	3.03	0.00	2.73	0.55
N5	2.18	1.68	2.57	1.48	1.38	8.53	0.00	2.58	1.52
N7	2.23	2.47	2.72	1.13	2.05	1.00	0.00	2.15	1.68
N11	1.55	1.98	2.55	1.48	1.38	0.62	0.20	2.37	0.98
Isolate	Plant line								
	7d	8a	8g	9c	9d	9g	10j	11i	19b
N1	0.35	3.17	3.05	0.05	0.00	0.00	1.05	0.38	2.07
N4	0.62	2.03	2.72	0.25	0.20	0.00	0.95	1.17	1.53
N5	0.00	2.82	2.90	0.00	0.00	0.20	0.68	1.12	2.62
N7	0.05	3.15	2.45	0.00	0.10	0.23	1.07	0.48	2.12
N11	0.92	3.28	2.97	0.47	0.95	0.10	1.07	2.17	1.62

LSD at P = 0.05 = 0.896
P = 0.01 = 1.179
P = 0.001 = 1.503

Table 6.6 Analysis of variance for plant lines 4h, 7d, 8a, 8g, 9c, 9g, and 11i with all 10 isolates

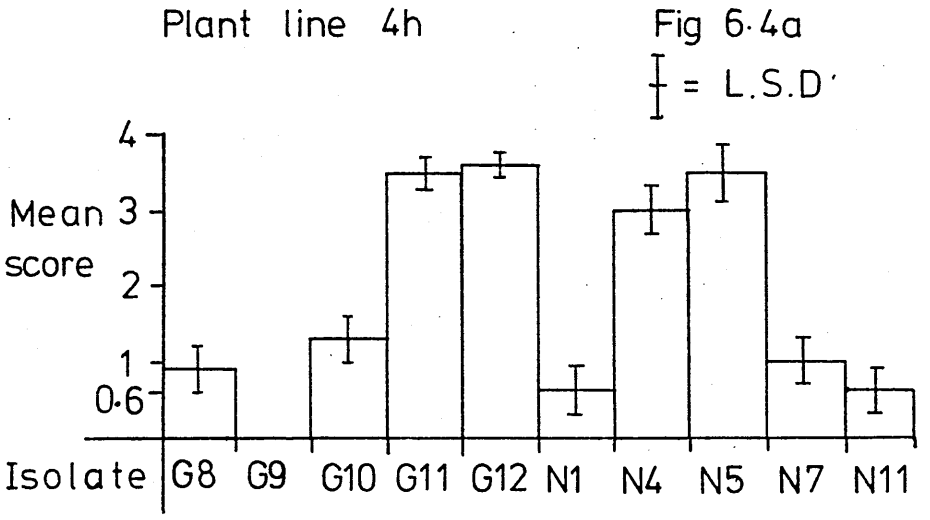
Source of variation	DF	SS	MS	VR	P
Isolate	9	287.46	31.94	18.67	NS
Plant line	6	4118.89	686.48	401.36	0.001
Isolate, plant line	54	1679.94	31.11	18.19	0.001
Residual	455	778.23	1.71		
Total	524	6864.52	13.10		
Error between dishes	525	464.67	0.89		
Error within dishes	2100	818.33	0.39		
Grand Total	3149	8147.52			

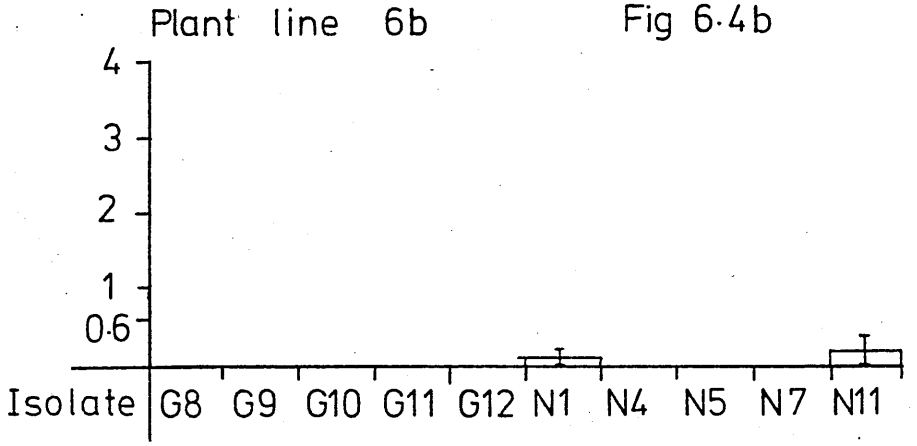
Coefficient of variation = 35.9%

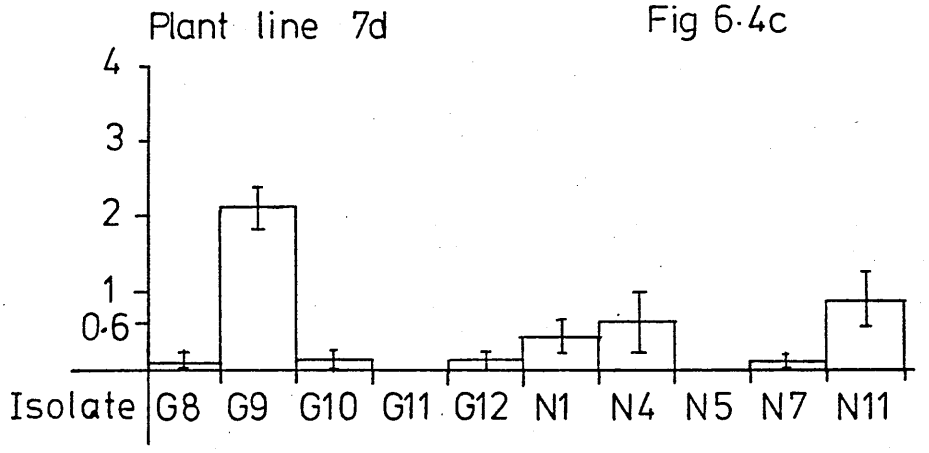
Table 6.7 Mean infection score for lines 4h, 7d, 8a, 8g, 9c, 9g, and 11i with each of the 10 isolates

Isolate	Plant line						
	4h	7d	8a	8g	9c	9g	11i
G8	0.92	0.13	3.12	3.21	0.13	0.17	1.00
G9	0.00	2.09	3.40	3.22	2.30	0.36	3.53
G10	1.36	0.13	3.48	3.47	0.08	0.68	0.74
G11	3.54	0.00	3.10	3.30	0.03	0.90	0.33
G12	3.60	0.05	3.15	3.13	0.00	0.42	0.26
N1	0.58	0.15	3.17	3.05	0.05	0.00	0.38
N4	3.03	0.62	2.03	2.72	0.25	0.00	1.17
N5	3.53	0.00	2.82	2.90	0.00	0.20	1.12
N7	1.00	0.05	3.15	2.45	0.00	0.23	0.48
N11	0.62	0.92	3.28	2.97	0.47	0.10	2.17

Fig 6·4a-d. Histograms of mean infection scores for each plant line tested with each isolate.







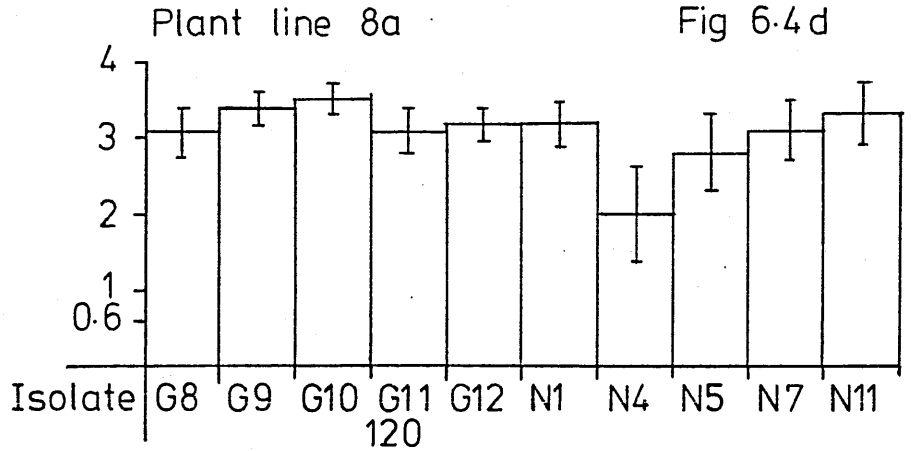
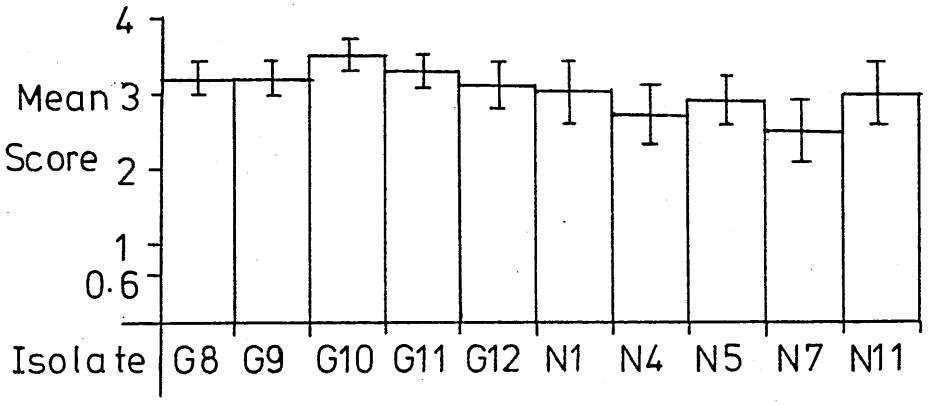
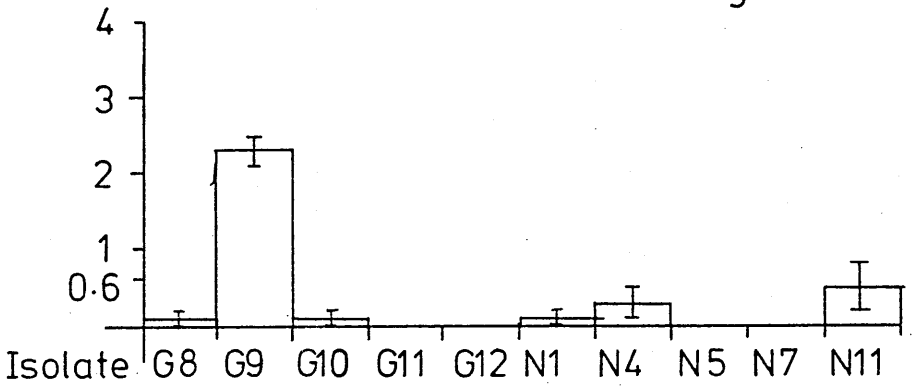


Fig 6·5a-d. Histograms continued.

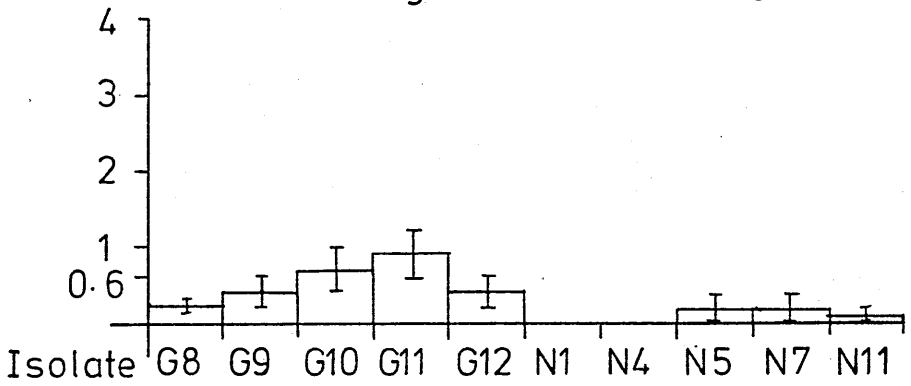
Plant line 8g Fig 6·5a



Plant line 9c Fig 6·5b



Plant line 9g Fig 6·5c



Plant line 11i Fig 6·5d

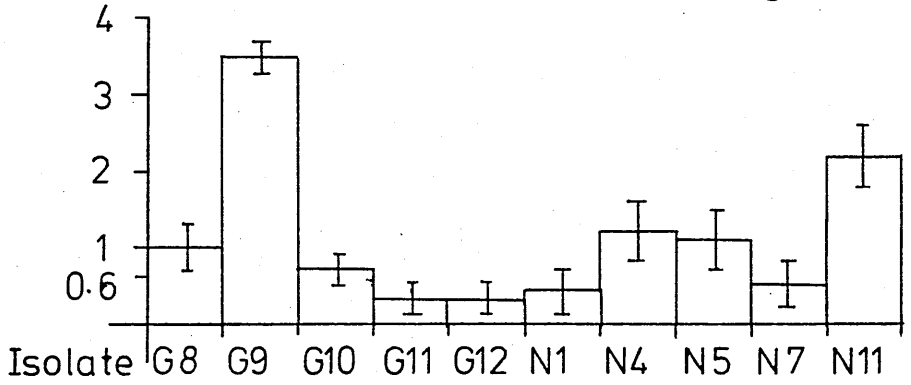
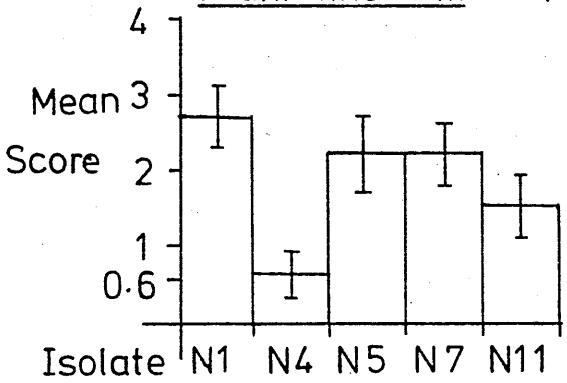
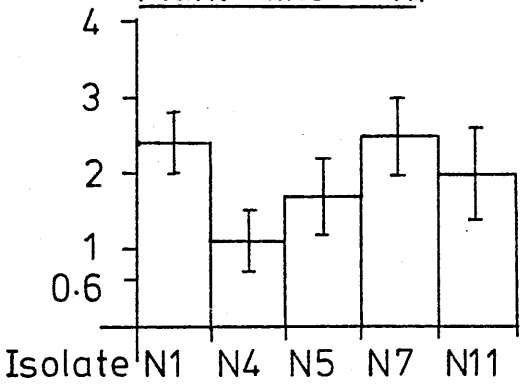


Fig 6·6a-d. Histograms continued.

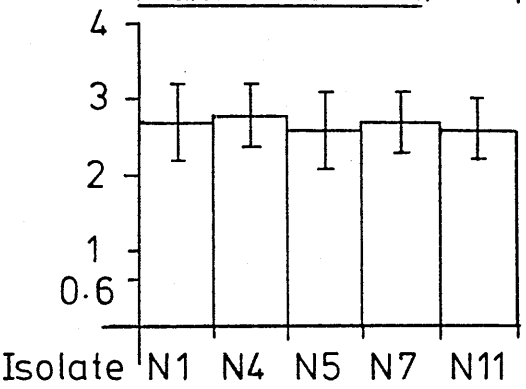
Plant line 1m. Fig 6·6a



Plant line 1n. Fig 6·6b



Plant line 2a. Fig 6·6c



Plant line 3f. Fig 6·6d.

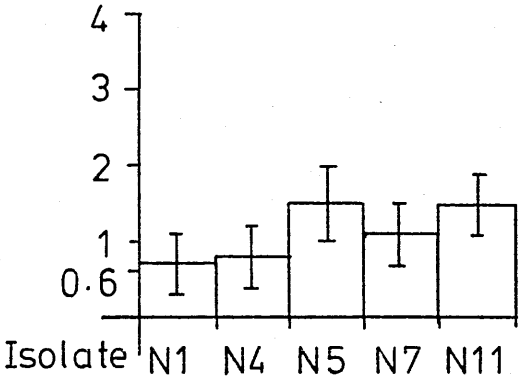
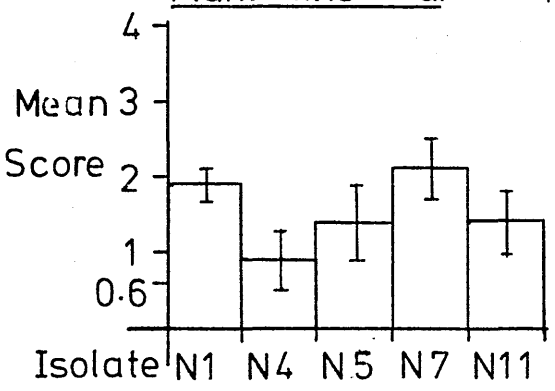
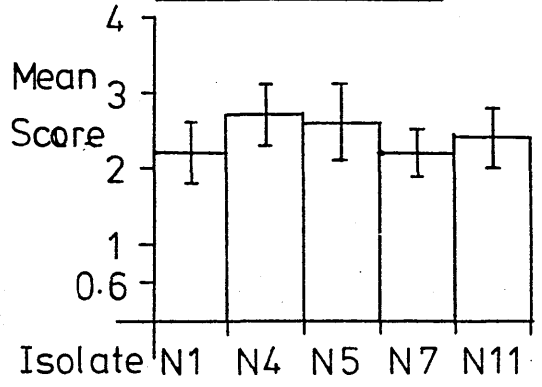


Fig 6·7a-d. Histograms continued.

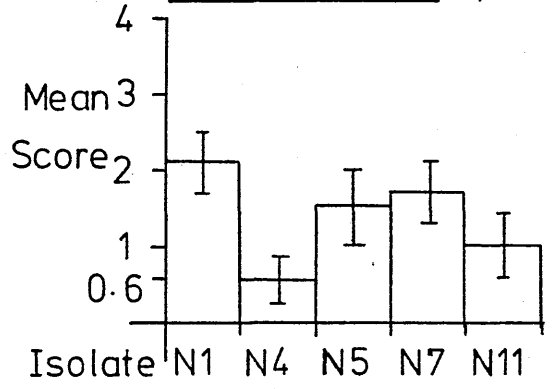
Plant line 4a. Fig 6·7a



Plant line 6d Fig 6·7b



Plant line 6f Fig 6·7c



Plant line 9d Fig 6·7d

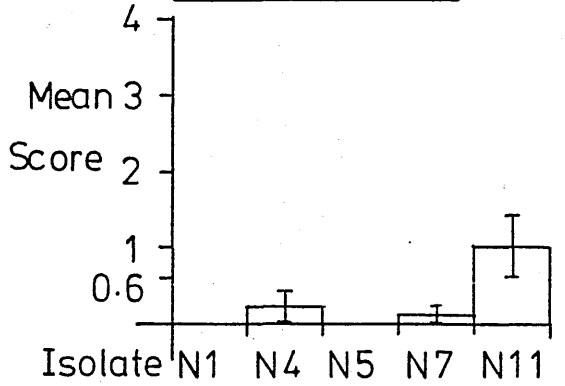
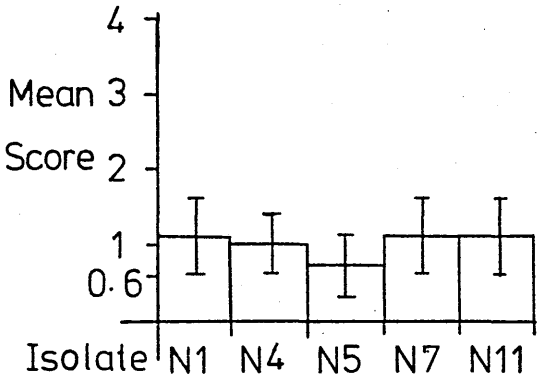
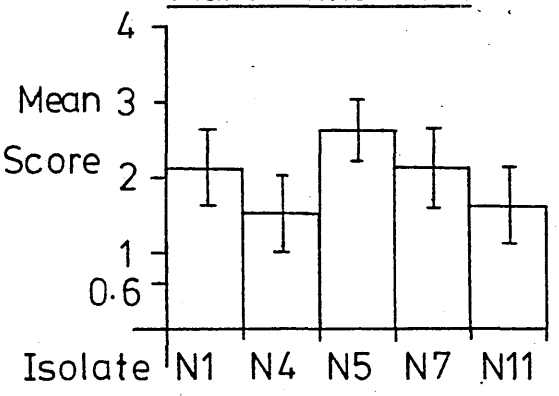


Fig 6·8a+b Histograms continued.

Plant line 10j Fig 6·8a



Plant line 19b. Fig 6·8b



6.4a). Line 7d shows an intermediate level of susceptibility to isolate G9, but an even lower level of susceptibility to isolate N11 (Fig 6.4c). Unfortunately there were no cases fitting a reciprocal check pattern, where differences in reactions were statistically significant, to establish definitely the presence of race-specific partial resistance.

6.3.2. Evidence for race non-specific partial resistance.

The histograms presented in Figs 6.4a-d to 6.8a+b give indications that some plant lines, such as, 3f, 2a, 4a, 6d and 10j gave equally low infection scores whichever the isolate they were tested with. This indicates that these lines may have race non-specific partial resistance. All the analyses of variance showed there was a significant variance ratio for plant lines, indicating that some plant lines are generally less susceptible than others to a range of isolates.

The presence of race non-specific resistance was investigated further using two methods. Both methods used the results obtained from the 7 plant lines tested with all 10 isolates; the mean infection scores are given in Table 6.7.

6.3.2.1 Method 1.

The mean infection score of each plant line to each isolate was ranked (Appendix Table 6.3). Spearmans ranking correlation coefficient (Parker, 1973) was calculated for each plant line pair in turn (Appendix Table 6.4). A significant correlation coefficient indicates that a particular plant line pair are constantly ranked in relation to each other, whichever the isolate used for testing, thereby indicating the occurrence of race non-specific resistance. A non-significant coefficient indicates race specificity. A mixture of significant and

non significant ranking correlation coefficients were obtained depending on the plant lines compared. Therefore, some plant lines showed both race specific resistance and race non-specific resistance depending on which plant line they were compared with. However, the method was not very reliable. The plant line pair, 8a and 8g, showed a non-significant coefficient. Normally these lines are of equally high susceptibility no matter which isolate is used for testing. A small difference in susceptibility was affecting the ranking substantially, giving a non-significant ranking coefficient.

6.3.2.2 Method 2.

Plant line/isolate relationships were investigated using a similar method to that of Crute et al (1983). The relative susceptibilities of plant lines 4h, 7d, 8a, 8g, 9c, 9g and 11i to the 10 isolates were compared with each other. The infection scores obtained with each isolate for each plant line were plotted against each of the other plant lines in turn. Figs 6.9a-f to 6.12a-f show the patterns which emerged.

The graphs involving plant line 4h are presented separately as it appears to produce race specific patterns of reactions when tested with most of the other lines. Points lie either side of the diagonal when the susceptibility of 4h is compared with that of lines 7d, 8a, 9c and 11i. The pattern of points produced with lines 8g and 9g lie either side of the diagonal, but very close to it so that the differences between the plant lines may not be significant.

The reaction patterns of line 9g fall very close to the diagonal but also either side of it. This may be an indication of race specificity but it is more likely the line behaves in a race non-specific way as it only shows a very low level of susceptibility to all isolates. It would, therefore, only appear to be race specific

Fig 6·9a-f

Susceptibility of each plant line to each isolate plotted against the susceptibility of each of the other plant lines

FIG 6·9a

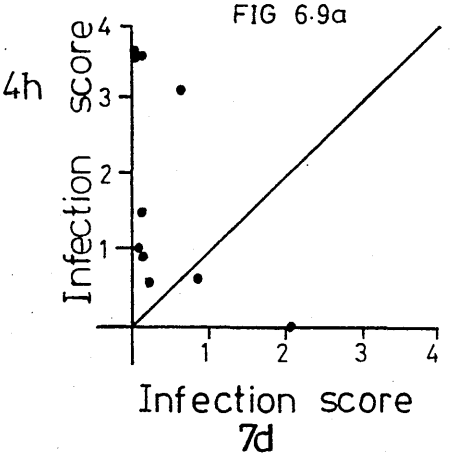


FIG 6·9b

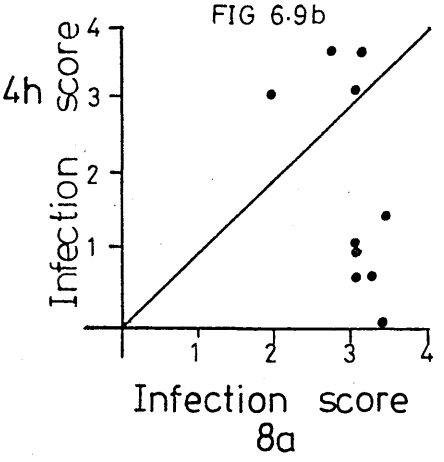


FIG 6·9c

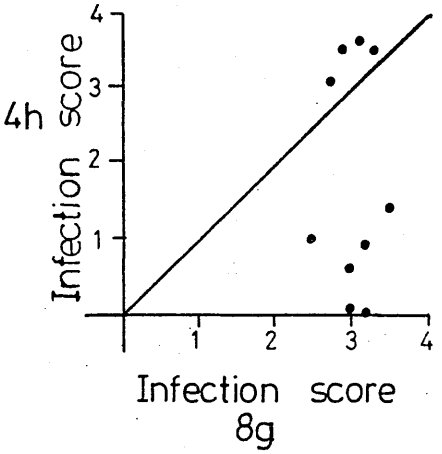


FIG 6·9d

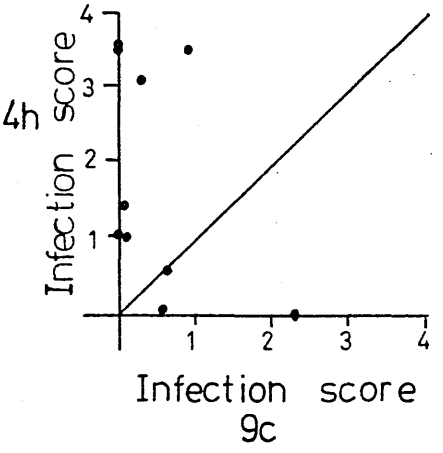
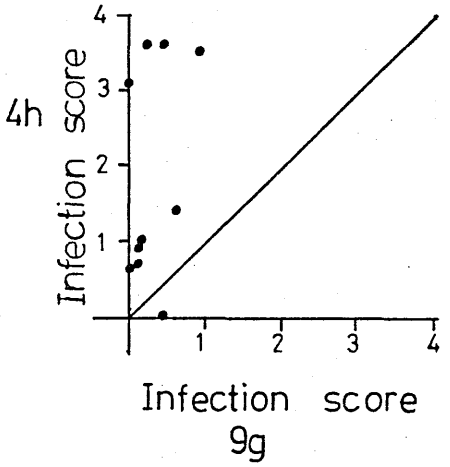


FIG 6·9e



6·9f

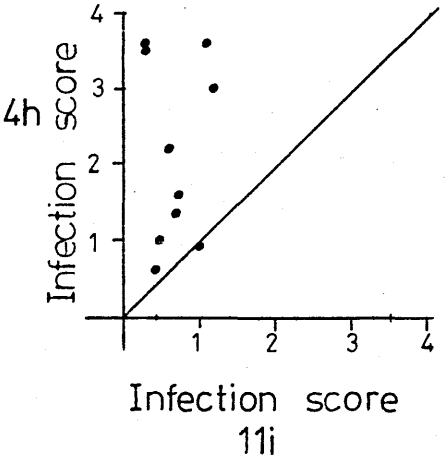


Fig 6·10a-e

Paired plant line comparisons continued.

Fig 6·10a

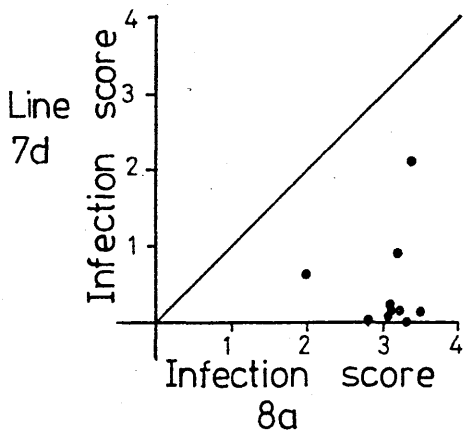


Fig 6·10b

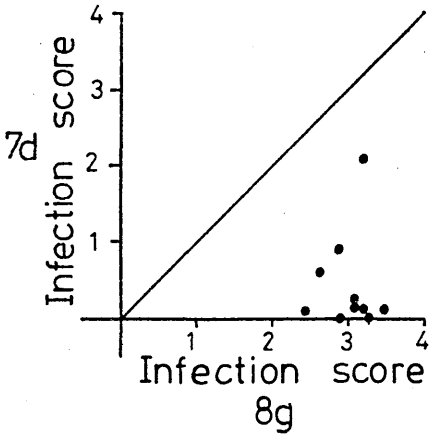


Fig 6·10c

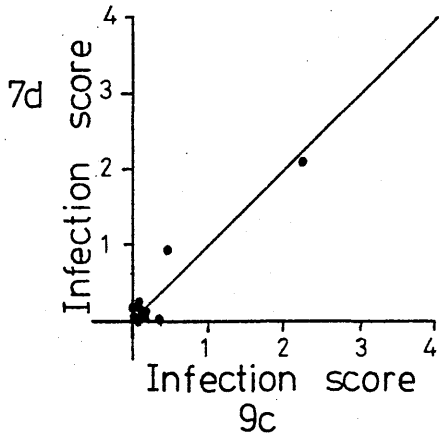


Fig 6·10d

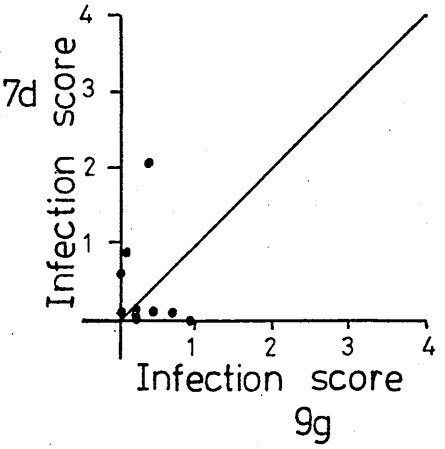


Fig 6·10e

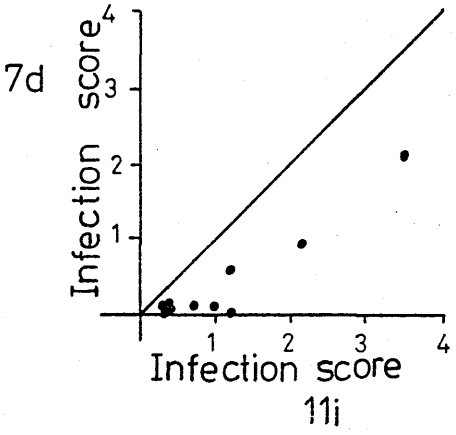


Fig 6·11a-d

Paired plant line comparisons continued

Fig 6·11a

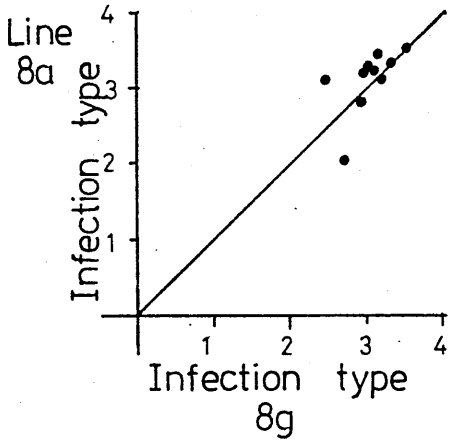


Fig 6·11b

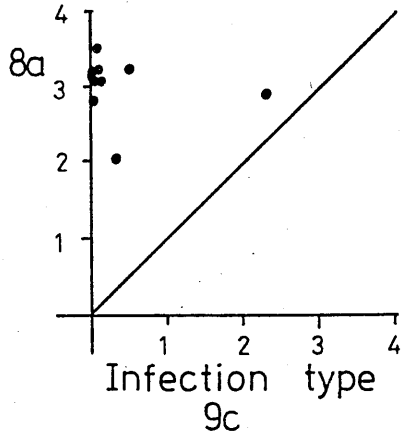


Fig 6·11c

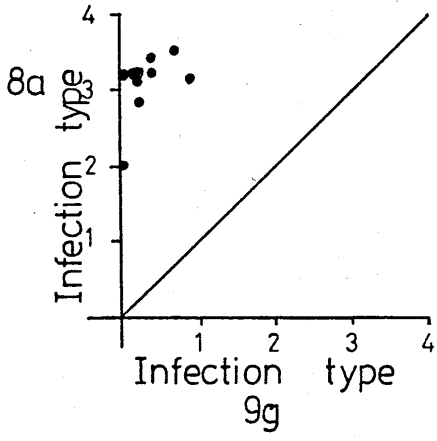


Fig 6·11d

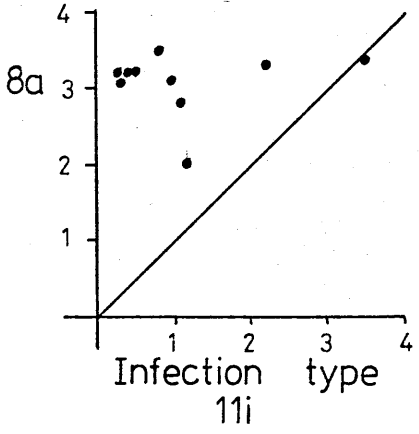


Fig 6·12a-f

Paired plant line comparisons. continued

Fig 6·12a

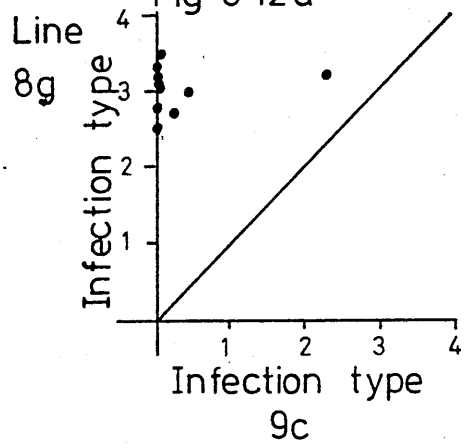


Fig 6·12b

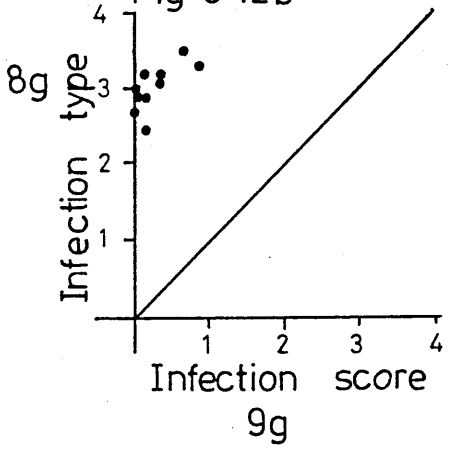


Fig 6·12c

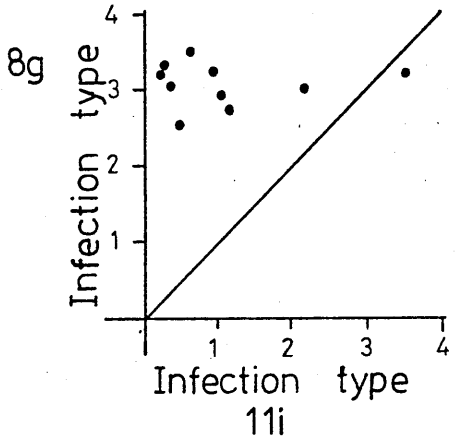


Fig 6·12d

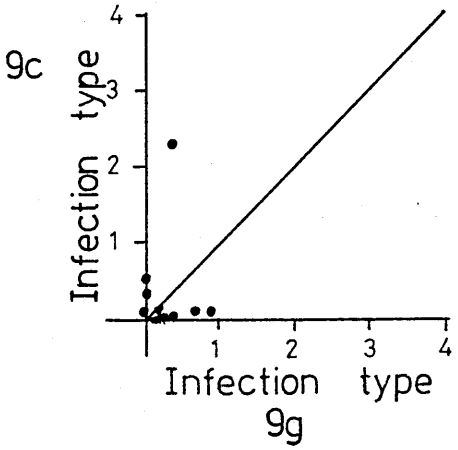


Fig 6·12e

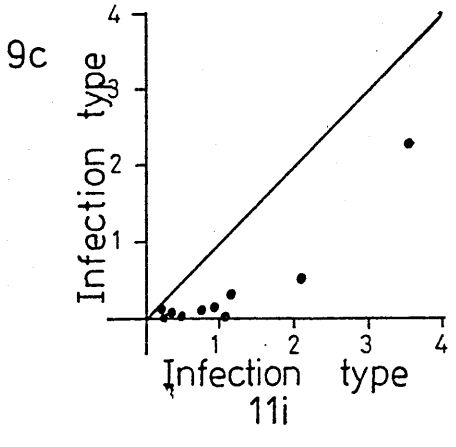
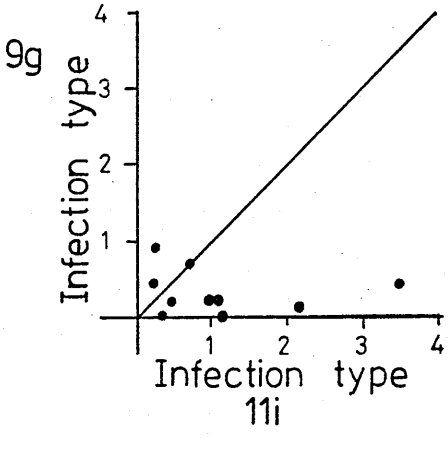


Fig 6·12f



when compared with highly susceptible lines such as 8a and 8g.

The reaction patterns of the other plant lines (7d, 8a, 8g, 9c and 11i) when compared with each other show that the majority of the points lie on one side of the diagonal or the other, thus indicating race non-specificity. Only 7d in comparison with 8a appears to show some race specificity since points fall either side of the diagonal. Using the diagrams, one can also rank the lines in order of susceptibility. For example, all the points for 8a lie towards the 8a half of the diagram, except for when it is plotted against 8g. Thus 8a is more susceptible than all the other lines except for 8g and to a few isolates when compared with line 4h. The ranking in order of susceptibility is:

8a = 8g.
11i.
7d = 9c.
9g.

The response of plant line 4h is predominantly race specific. Depending upon isolate, it can be more susceptible than 8a or 8g, and less susceptible than 11i, 9c or 7d.

6.3.3 The presence of several types of resistance in the same plant. The histograms presented in Figs 6.4 to 6.8 suggest that some plant lines carry both race specific and race non-specific resistance. For example, plant line 7d could be said to have a mixture of race specific complete, race specific partial and race non-specific partial resistance. Line 7d shows only moderate susceptibility to isolate G9, therefore, it probably has a reasonable level of non-specific resistance.

6.3.4. A comparison of the performance of the isolates from N.V.R.S. and Glasgow.

The infection scores produced by the two sets of isolates were compared to see if either the N.V.R.S. isolates or the Glasgow

isolates had a tendency to elicit a greater proportion of susceptible reactions. Analysis of variance (Table 6.6) showed that there was no significant difference between isolates for the general level of infection they produced.

6.4 Discussion.

Partial resistance has clearly been demonstrated to be operating within the groundsel/powdery mildew pathosystem. Its occurrence is relatively common indicating that it is likely to be an important component of the groundsel defence strategy. Incomplete or partial resistance in addition to complete resistance has also been reported to occur in wild *Glycine* spp. to the leaf rust fungus *Phakopsora pachyrhizi* (Burdon and Marshall, 1981). In this pathosystem partial resistance was expressed as a reduction in the number and size of rust pustules, and an increase in the time taken for rust pustules to develop in comparison with those produced on more susceptible plants. However, only one isolate originating from a single urediospore was used for screening so that the specificity of partial resistance could not be determined. Quantitative or partial resistance was found in *Avena sterilis* and *A.barbata* to *Puccinia coronata avenae* (Wahl,1970), but the evidence suggested that the resistance was 'horizontal' or race non-specific in nature.

In the groundsel/powdery mildew pathosystem, there is evidence that partial resistance may be both specific and non-specific, and that both forms of resistance can occur in the same plant. As Johnson (1979) has pointed out, this can be observed where forms of resistance are under the control of different genes although Nelson (1978) has argued that it is not possible for one plant to possess both types of

resistance if the term race specific resistance is strictly applied.

Parlevliet and Zadoks (1977) proposed a model showing how resistance that was apparently race non-specific could in fact be regulated by specific gene-for-gene interactions in the same way as race specific resistance. It can be argued that there is really no such thing as race non-specificity, and that such evidence for it that has been presented here may simply be due to the restricted number of isolates that were examined.

On the other hand, Vanderplank (1984) argued that host reactions can appear to be race specific, when they are in reality non-specific. This is because responses occur at the two extremes of susceptibility and resistance with no intermediate responses, even though there are no inversions in ranking. As Clifford (1974) notes, 'man catalogues resistance into two types for convenience but nature never intended such a division'.

The situation encountered in the groundsel/powdery mildew pathosystem is confusing. Barrett (1985) pointed out that our simplistic view of the gene-for-gene hypothesis, developed from the study of crop systems in which disease resistance has been manipulated by man, is not likely to be able to explain the complexities of a natural pathosystem.

Clearly the interactions in the groundsel/powdery mildew pathosystem are highly complex. To complicate things further environmental factors such as temperature, and factors such as plant age, also affect the expression of resistance. Reports that the expression of partial resistance in crop systems is affected by environment are common. The partial resistance of coffee (C.arabica) to coffee leaf rust (Hemileia vastatrix) is affected by temperature, light intensity and leaf age. The partial resistance of some individuals is suspected of being under the control of incompletely

dominant genes, whilst the resistance of other individuals is thought to involve polygenes (Eskes, 1983). Heather and Chandreshekar (1982a,b) found that environment played an important role in the expression of resistance in wild Populus spp. to leaf rust. Here, partial resistance is also an important component of the defence strategy. Lewellen (1967) discovered that some wheat varieties resistant to Puccinia striiformis carried incompletely dominant major genes for resistance but the expression of resistance was also affected by minor genes that were in turn affected by the environment.

The defence strategy of groundsel to Erysiphe fischeri is clearly complex. In the wild pathosystem it appears that complete race specific resistance would rarely act in isolation having evolved alongside and in conjunction with other forms of resistance. One form of resistance is unlikely to succeed in isolation from any other over a period of time. Evolutionary forces have probably ensured that several forms of resistance work interactively to provide the most successful insurance for both host and pathogen survival.

The importance of the interaction between the different types of resistance, the effect of the environment and host factors such as stage of development have probably been underestimated in the past. Complete race specific resistance has usually been bred into crops in isolation of the other forms of resistance. The repeated backcrossing of resistance genes into an agronomically desired cultivar has led to the loss of partial resistance or other genes associated with the expression of the major gene for resistance itself. In the absence of genes for partial resistance, selection in the pathogen for virulence to match a gene regulating complete resistance may occur rapidly because there are no other additional forms of resistance to modulate the rate of reproduction of the virulent variant.

CONTENTS. Page.

7.0. RESISTANCE IN THE N.V.R.S. AND GLASGOW GROUNDSEL POPULATIONS.

7.1. Introduction.

Essential prerequisites of the work described in this chapter were to develop suitable sampling techniques and strategies, and to compare their efficiencies in detecting different phenotypes in groundsel for resistance to mildew. The main aims of the work were to:

1. Determine the number and frequency of resistance phenotypes in the groundsel population as a whole, and two separate populations at the N.V.R.S. and Glasgow.
2. Determine the distribution and heterogeneity of resistance phenotypes in sub-plots within the main areas.
3. Determine if the resistance phenotypes and their distribution changed from year to year.

7.1.1. The distribution of sampling points.

The breeding system and the distribution of individuals are important factors to consider when choosing sampling stategies. Where genotypes are distributed randomly, as in a freely out-crossing species, samples taken at random may adequately represent the original population for both the degree of variation and the population mean (Lewis and Taylor, 1967). They suggested randomly sampling an area by dividing it into equally sized quadrats and taking at least five individuals at random from within each quadrat.

Groundsel is considered to be inbreeding (Haskell, 1953). Consequently, one would expect progenies to tend to be very similar to their parents. Despite the fact that the achene of groundsel has a pappus attached to it, the majority of the achenes tend to fall and

develop into plants close to the parent plant. This tends to result in a patchy distribution of individuals with different genotypes. Where the distribution of genotypes is patchy, Jain (1975) suggested a sampling strategy where individuals were taken from widely scattered sampling points. It is possible that an inbreeding population may approach the structure of an outbreeding population if there is an efficient seed dispersal mechanism (Jain, 1975). The pappus may provide this in some cases; also groundsel tends to occupy disturbed areas and there may have been efficient localised mixing of the seed bank by activities such as ploughing. However, it is unlikely that achenes are dispersed over wide areas by such means.

The majority of the samples taken in the present study were from widely dispersed points appropriate for a patchy distribution of genotypes.

7.1.2. The size of area considered to constitute a population.

In a crop situation, the area over which a population is said to be distributed is often conveniently determined by field or farm boundaries. However, as Marshall and Brown (1975) pointed out, there are usually no such boundaries in natural plant populations. The limits of an area said to hold a population have to be decided by the observer. In the groundsel populations studied, several of the areas had obvious boundaries, or else plot sizes were limited by the area of land available for experimentation.

7.1.3. Size of sample represenative of a population.

The numbers of individuals that need to be sampled to accurately represent the original population depend on the size of the population, the variability in the population and the degree of variability one wishes to detect (Yates, 1981). In general, the

larger the population and the greater the variability within it, the larger the sample needs to be. A general formula may be applied to determine the size of sample depending on the standard error it is desirable to detect.

Where p = the proportion of units of a given type in the whole population, q = the proportion not of the given type in the population and n = the size of the sample required, then;

$$\text{standard error of } p = \sqrt{\frac{pq}{n}}$$

$$\text{or} \qquad n = \frac{pq}{(\text{required standard error of } p)^2}$$

Marshall (personal communication) suggested another formula for determining the sample size based on the known frequency 'f' of a particular allele 'A' and the probability 'P' of finding it within a sample size 'n'. The formula is designed to apply to homozygous individuals as would be expected with an inbreeder.

To detect an allele 'A' at a frequency of 'f' in a population then the probability that 'A' does <u>not</u> appear in a sample of 'n' individuals is:

$$'P' = \text{approx } e^{-nf}$$

Since e^{-3} is approximately 0.05, then to have a 5% chance of finding an allele at a particular frequency, the sample size would be given by:

$$n = \frac{3}{f}$$

Thus to have a 5% probability of finding an allele present in the population at a frequency of 0.01, a sample of at least 300 individuals is required to detect it.

Unfortunately, both these methods of determining sample size require previous knowledge of the frequency of the various individuals sought in the population and the degree of variability in the

population. In the case of groundsel these factors are unknown as far as resistance phenotypes are concerned and so they can only be used as a general guide.

7.1.4. Sampling strategies used by other workers.

Several sampling strategies have been reported in the literature for sampling natural populations. Hull (1974) in a study of the distribution of the radiate form of Senecio vulgaris, which is a relatively rare phenotype, investigated 45 sampling sites and took samples of between 50 and 500 individuals.

Allard (1970) also suggested using very large sample sizes to detect genetic variability in wild oats (Avena fatua), which is an inbreeder, for use in breeding programmes. The strategy suggested for sampling an area 600km x 200km was as follows:

10 seeds collected (from the same panicle) per plant,

200 plants per local population (defined as a site approximately 50m by 50m),

5 local populations per region (defined as an area of 5km by 5km),

20 regions sampled per east west transect,

5 transects distributed at more or less 200km in a north west direction.

This type of strategy was designed to detect as much variation in the wild population as possible. Qualset (1975) suggested using sample sizes of 500 or more to detect suitable breeding material in wild populations, but also pointed out that this was impractical in many situations.

The work of Dinoor (1970) on the collection of wild oats with resistance to oat crown rust (Puccinia coronata avenae), provided some useful information about sampling strategies. He found that the most

efficient way to detect different resistance phenotypes was to take relatively small samples of individuals from many locations. If resistance was found in that location the site should then be intensively sampled. The locations for sampling should be about 5km apart and predetermined by drawing a grid on the map of the whole area to be investigated.

Marshall and Brown (1975) also proposed a more realistic sampling strategy for examining populations where there is very little information on the frequency and variability of particular types of individual.

1) Collect 50 to 100 individuals per site.

2) Sample as many sites as possible within the time available.

3) Sample from as broad a range of environments as possible.

In this way only the more common types of individual would be detected on each site.

Marshall (personal communication) also suggested that a plan of the area to be sampled should be divided into a grid and the position of each individual sampled should be recorded to obtain information on their spatial distribution. Wherever possible, individuals should be sampled from different quadrats of the grid. This sampling strategy was followed for most of the groundsel populations studied. It was aimed to sample 50 individuals from each sampling area but this was not always achieved.

7.1.5. Distance between sampling sites.

The two main sampling sites chosen for examination (Glasgow and N.V.R.S.) were 300 miles apart. If there is geographic variation in the population then this distance should be great enough for differences between populations to become apparent. Since groundsel is considered to be highly inbreeding then, even in the absence of

selection for particular phenotypes, genetic drift should be great enough to cause populations to diverge (Loveless and Hamrick, 1984). Differences in the most common phenotypes should be apparent and detected using relatively small samples. However, the potential ability for groundsel achenes to be dispersed over large distances may limit these effects so that the population structure approaches that of an outbreeding population where there is likely to be less divergence. It seems unlikely that substantial quantities of achenes would be carried 300 miles, causing the populations to become similar.

7.1.6. Sampling techniques.

Two different sampling techniques were used in four small plots at Glasgow.

i) The seed bank was sampled by growing up plants originating from soil samples and collecting their seed.

ii) The established groundsel on each plot was examined by collecting seed at random from 25 mature plants from each plot.

It was hoped that in this way the efficiency of collecting different resistance phenotypes using the two methods could be compared. Also, it was hoped that a determination could be made of any differences between the resistance phenotypes in the seed bank and those observed in established plants.

7.1.7. Inbreeding and homozygosity in groundsel.

Groundsel is generally considered to be an inbreeder. It is capable of outbreeding being hermaphrodite and protandrous with no evidence of self-incompatibility (Fritsch and Salisbury, 1938). The degree of outcrossing in a natural population was reported by Hull (1974) to be as low as 0.1%. Campbell et al (1976), estimated the level of

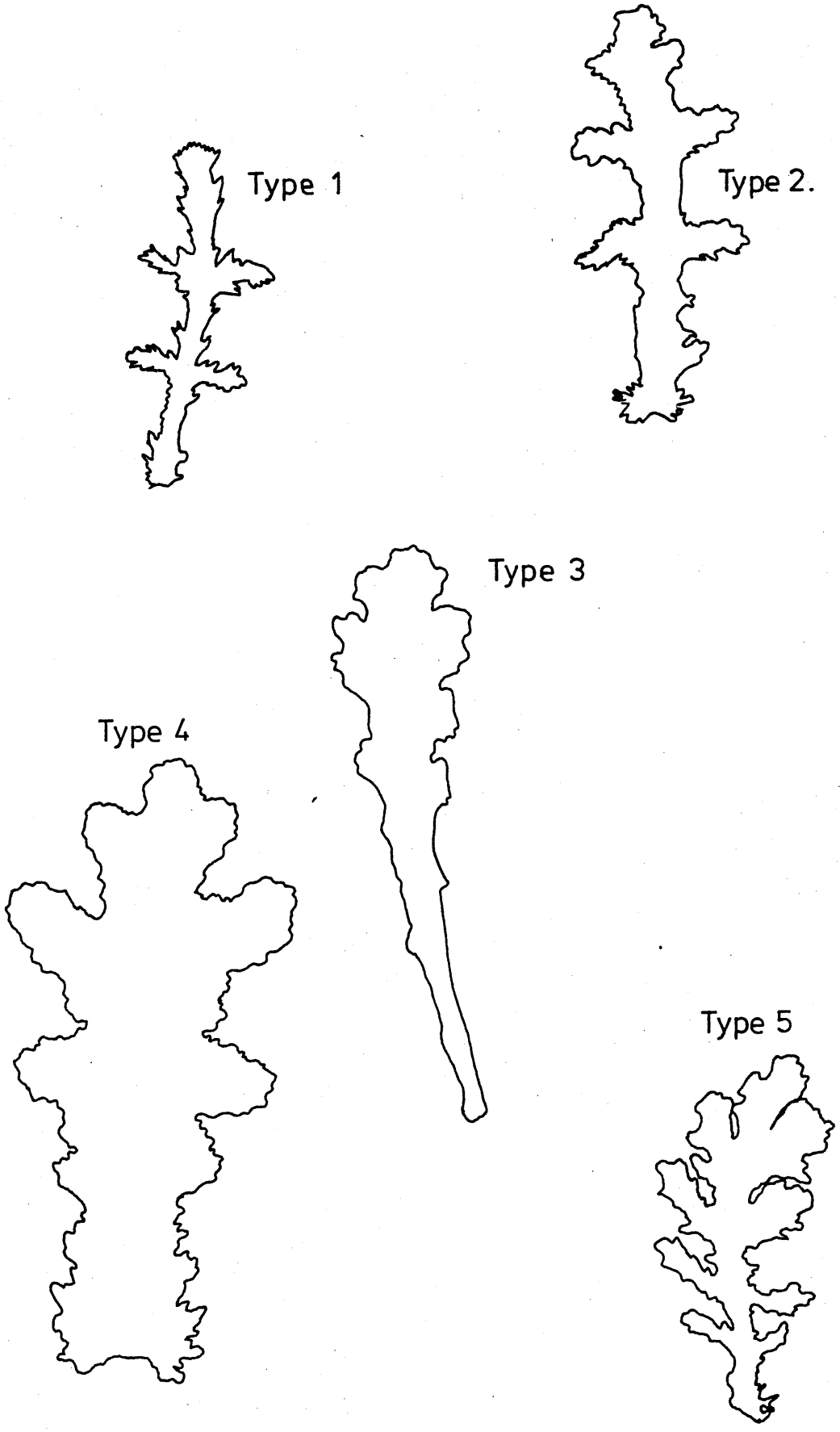
outcrossing to be 22% but their data were based on observations of artificial populations with a high proportion of radiate individuals that attract more insects, the primary cross-pollinating agents of groundsel. Haskell (1953), examined the morphological characters of five generations of groundsel and found little evidence for heterozygosity.

During the present work an attempt was made to determine the homozygosity of groundsel using morphological characters as markers. The morphological characters of several progenies were assessed. Only progenies tested at exactly the same time and in the same conditions could be compared. Stem colour and hairiness, found to be heritable characters by Trow (1913), were recorded together with other qualitative characters such as leaf colour and hairiness. Leaf shape was recorded by making photocopies of the fully developed leaves and categorising them into various shapes as in Fig 7.1. Quantitative characters such as plant height were measured (plant height being from the cotyledonary node to the last node bearing a ferile branch), and the number of nodes in a similar way to Trow (1916). The morphological characters of several sets of assessed progeny are given in Appendix Tables 7.1a for plants grown in the glasshouse at Glasgow and 7.1c for progenies grown in mildew free cabinets at N.V.R.S.

Analysis of variance (Appendix 7.1b and 7.1d) showed that in general there were greater differences for quantitative characters between progeny families than within them. This indicated that individuals within a family were similar and therefore likely to be the result of inbreeding. The infection types on detached leaves of 3 progeny families following inoculation with 10 isolates were also assessed at N.V.R.S. These families were chosen because siblings had previously been shown to be resistant to some if not all isolates. Two of the families gave consistently resistant reactions to all

Fig 7.1

Leaf morphology types.



isolates. However, the third family did not show consistently the expected resistance patterns (Appendix Table 7.2), indicating that segregation was taking place and that outbreeding may have occurred in the recent history of the family. However, only low levels of susceptibility were recorded suggesting that the results could be confounded by the expression of partial resistance. It appears that on the basis of this work and the work of others that plants sampled were, for the most part, homozygous and true-breeding but that some error may have been introduced due to occassional outbreeding when different siblings of a particular plant were tested with the Glasgow and N.V.R.S. isolates.

7.2. Materials and methods.

7.2.1. The areas sampled.

The areas sampled at Glasgow are referred to as follows:

Area GL1 comprised of four 2 m x 4 m plots.

Area GL2, a partially walled garden at Garscube of approximately 100 m x 75 m. Area GL1 lay within area GL2.

Area GL3 of approximately 1.2 Km^2 mainly consisting of park land and housing estates. Areas GL1 and GL2 lay within the area GL3.

The areas sampled at N.V.R.S. are referred to as:

Area N1, a 1 m^2 plot heavily populated with groundsel.

Area N2, a plot 17 m by 17 m. Area N1 lay within area N2.

Area N3 of approximately 6 Km^2 lying within the N.V.R.S. boundaries. Areas N1 and N2 were within area N3.

7.2.2. Collections of groundsel from Glasgow.

7.2.2.1. Sampling from the seed bank.

Four plots each of 2 m by 4 m within the walled garden at Glasgow were sampled. The total area of these four plots will be referred to as area GL1. Four soil samples, each from an area 50 cm by 30 cm and to a depth of 3 cm were taken at random sites from each plot. The majority of viable groundsel seed is thought to reside in the surface 2.4 cm of soil (Chippendale and Milton, 1934). Therefore, these samples should have possessed the majority of the viable groundsel seed bank. A fifth soil sample was taken from each plot by skimming the surface 0.5 cm to 1 cm of top soil from a 1 m by 1 m area. Soil samples were also taken from a private garden near to the main

sampling site.

Each soil sample was placed in a seed tray (37 cm by 24 cm), watered, covered with polythene and placed in gravel trays to be watered from below as required. The seed trays of soil were placed in a glasshouse with no supplementary heating or lighting. After about 1 week, the polythene was removed and after about 2 weeks any groundsel seedlings old enough to be moved were transplanted individually into 12 cm plastic pots filled with S.A.I. compost and left in the glasshouse to develop. Popay and Roberts (1970) found that groundsel seeds did not germinate if they were more than 2 mm below the soil surface. Therefore, once a 'crop' of groundsel was obtained from a soil sample, other plant species were weeded from the tray, and the sample was lightly cultivated to bring previously buried seed to the surface.

When plants reached maturity, seed samples were collected from each plant. This sampling method was not very successful, since only 49 plants developed to set seed from 25 soil samples. The method was also extremely time-consuming. The low recovery rate was partly due to 'damping-off' after transplanting and partly due to heavy aphid (Myzus persicae) infestations at the flowering stage which destroyed many plants before seed could be collected. However, the method could be useful at certain times of the year, especially winter, when seeding groundsel is not abundant in the field, particularly if supplementary lighting could be provided to promote rapid development.

7.2.2.2. Collection of seed from established plants from four 2 m x 2 m plots (Area GL1).

The plots were ploughed in the autumn of 1982 and surface cultivated in March 1983. No herbicide was applied to these areas. In recent times, areas GL1 and GL2 had not recieved any herbicides. During

September and October of 1983 each of the plots was divided into thirty-two quadrats: 50 cm by 50 cm, using cane and twine. Twenty-five quadrats on each plot were randomly selected and one seeding plant from each quadrant was randomly chosen and up-rooted. The harvested plants were placed in beakers of water to set seed in a glasshouse. Seed from each plant was packeted separately.

7.2.2.3. Seed collection from the established plants within the walled garden at Garscube (Area GL2).

The areas under cultivation within the garden were ploughed in the autumn of 1982 and surface cultivated in March 1983. For sampling purposes, a grid was drawn over a plan of the garden so that each square represented an area of approximately 5 m by 5 m. Not every quadrat was populated by groundsel since much of the area was under grass or built on. During September and October groundsel seed was collected in the following way. Wherever possible, one plant was randomly chosen for sampling from within each quadrat that was not occupied by grass or buildings. The position of each plant sampled was recorded on the plan. The plants were placed in beakers of water in a glasshouse and seed collected from each of 52 plants.

7.2.2.4. Seed collection from established plants in the general area around Garscube Gardens at Glasgow (Area GL3).

A grid was drawn over a map of the area so that each square represented an area of approximately 100 m x 100 m. Most of the groundsel populations within GL3 were on private housing estates. From May until October 1983, one plant from within each area, wherever possible, was obtained from the gardens of the houses. The approximate positions of the plants were marked on the map and the seed was collected from the plants as described previously.

7.2.3. Groundsel seed collections from the N.V.R.S.

7.2.3.1. Collections made from established plants in the 17 m by 17 m plot (Area N2) and the 1 m by 1m plot (Area N1).

Two separate collections were made from Area N2, one in August 1983 and the other in August 1984. The area was ploughed in the autumns of 1982 and 1983, then in the following March the area was surface cultivated. There were problems with other weeds in the plot so the plot was treated with selective herbicide. In March 1983 and 1984 the area was treated with Kerb (propyzamide) at a rate of 2.25 Kg of product ha^{-1} to control all species except *Compositae* such as groundsel and mayweed (*Matricaria* spp.). The area was also treated with a trial herbicide CME 127 at a rate of 1.5 litres ha^{-1} in March 1984 to control mayweed. In April 1983 and 1984 the area was raked to allow germination of groundsel. Thistles became a problem on the plot in 1984 so they were spot treated with glyphosate at a rate of 30 ml l^{-1} early in May. The plot was then marked out into 1 m squares. In 1983 and 1984 the frequency of groundsel plants and the number of plants infected with *Erysiphe fischeri* were recorded. After the records were completed in each year, the nearest plant to every other intercept was harvested and its seed collected as before. The intercepts not sampled in 1983 were sampled in 1984. Forty-seven plants in 1983 and 44 plants in 1984 were sampled.

The quadrat with the highest population of groundsel within the 17 m x 17 m plot was intensively sampled in August 1983. This area of 1 m^2 is known as Area N1. Every plant (75 plants) within the square was harvested and its seed collected.

7.2.3.2. Collections of established groundsel from within the N.V.R.S. boundry (Area N3).

A grid was drawn on a map of the N.V.R.S. estate where each square represented an area of approximately 100 m by 100 m. Wherever possible, one seeding plant from each quadrat was harvested, its position marked on the map and the seed collected as before. Two separate collections of seed were made from Area N3, one during June 1983 and the other in June 1984. Forty plants were sampled in 1983 and 60 in 1984.

7.2.4. Control tests.

Wherever possible, each test included control plant lines of a similar age to the groundsel populations under test. The control lines were some of the inbred lines originally used to characterize the mildew isolates which could be used as differentials to distinguish between isolates. From the control tests checks could be made to determine whether the isolates produced expected reactions, providing evidence of any cross-contamination. Plant lines 4h, 6b, 7b, 7d, 8a, 8g, 9a, 9c, 9g and 11i were used as controls when the Glasgow isolates G8, G9, G10, G11 and G12 were tested. An additional 10 lines 1m, 1n, 2a, 3f, 4a, 6d, 6f, 9d, 10j and 19b were used in the control tests when the N.V.R.S. isolates N1, N4, N5, N7 and N11 were used for inoculation.

7.2.5. Testing procedures.

Seeds were sown from the collections in batches of 50 samples and tested with mildew isolates. The Glasgow isolates and the N.V.R.S. isolates were tested on separate occasions. One plant from each seed sample was tested with the Glasgow isolates (G8, G9, G10, G11 and

G12), a second sibling plant from the same parent was tested with the N.V.R.S. isolates (N1, N4, N5, N7 and N11). Since outbreeding in groundsel has been estimated to be as low as 0.1% (Hull, 1974) the sibling plants were probably of identical genotype.

7.3. Results.

7.3.1. Resistance in the groundsel populations.

The infection types 0, 1, 2-, 2+, 3 and 4 were transformed to 0, 1, 1.5, 2.5, 3 and 4 for the purposes of analysis. The mean infection scores were calculated for each plant tested with each isolate. The results of the control tests are given in Appendix Table 7.3. The data for the Glasgow and N.V.R.S. plants are given in Appendix Tables 7.4 and 7.5 respectively.

A table of the different resistance phenotypes and their frequencies in each population are given in Table 7.1.

7.3.2. A comparison between resistance phenotypes detected in plants obtained from the seed bank and those derived from established plants.

Few seed samples were successfully collected by soil sampling in Area GL1. Plot 2 was the only plot from which a reasonable number of samples was obtained. The five mildew isolates collected from Glasgow (G8, G9, G10, G11 and G12) were used to test the reactions of 25 plants collected by soil sampling from Plot 2 and 25 plants collected as seed from established plants. Only two plants out of the 50 tested were resistant to any of the mildew isolates. One plant out of the 25 collected directly from established plants was resistant to isolate G9, whilst one plant out of the 25 from the soil sample collection was resistant to isolates G9 and G12.

The results for the plants obtained from seed from established individuals on all four plots were pooled. Likewise the results for all the plants collected by soil sampling were grouped together. Only 3 plants out of 100 from established plants and two of the 49 plants

Table 7.1. Number of plants in each sampling area with each resistance phenotype.

	ISOLATE										No of
Area GL1	G8	G9	G10	G11	G12	N1	N4	N5	N7	N11	plants
Plot 1	S	S	S	S	R	-	-	-	-	-	2
	S	S	S	S	S	-	-	-	-	-	23
Plot 2	S	R	S	S	S	-	-	-	-	-	1
	S	S	S	S	S	-	-	-	-	-	24
Plot 3	S	S	S	S	S	-	-	-	-	-	25
Plot 4	S	S	S	S	R	-	-	-	-	-	1
	S	S	S	S	S	-	-	-	-	-	24
Soil sample Plot 2 only	S	R	S	S	R	-	-	-	-	-	1
	S	S	S	S	S	-	-	-	-	-	24
All soil samples with Plot 2	S	R	S	S	R	-	-	-	-	-	2
	S	S	S	S	S	-	-	-	-	-	47
Area GL2	S	R	S	S	R	S	S	S	S	S	2
	S	S	S	S	S	S	R	S	S	S	1
	S	S	S	S	S	S	S	S	S	R	1
	S	S	S	S	S	S	S	S	S	S	48
Area GL3	S	R	S	S	S	S	S	S	S	S	1
	S	S	S	S	S	R	S	S	S	S	1
	S	S	S	R	S	S	S	S	S	S	1
	S	S	S	S	R	S	S	S	S	S	1
	S	S	S	S	S	S	S	S	R	S	2
	S	S	S	S	S	S	S	R	S	S	1
	R	S	R	R	S	R	S	R	S	S	1
	R	R	R	R	R	R	R	R	R	R	1
	S	S	S	S	S	S	S	S	S	S	37

R = resistant
S = susceptible
- = Not tested

Table 7.1 continued

	ISOLATE										No of
	G8	G9	G10	G11	G12	N1	N4	N5	N7	N11	plants
Area N1	R	S	S	S	S	-	-	-	-	-	1
	S	R	S	S	S	-	-	-	-	-	43
	S	S	S	R	S	-	-	-	-	-	2
	S	S	S	S	R	-	-	-	-	-	1
	R	S	R	S	S	-	-	-	-	-	1
	S	R	S	R	S	-	-	-	-	-	1
	S	R	S	S	R	-	-	-	-	-	1
	R	R	R	S	S	-	-	-	-	-	1
	R	R	S	R	R	-	-	-	-	-	1
	S	S	S	S	S	-	-	-	-	-	23
Area N2 1983	S	R	S	S	S	S	S	S	S	S	22
	S	S	S	S	R	S	S	S	S	S	1
	R	R	R	R	R	S	S	S	S	S	1
	R	S	R	S	S	R	S	S	R	R	1
	S	S	S	S	S	R	S	S	R	R	1
	R	R	R	R	R	R	R	R	R	R	2
	S	S	S	S	S	S	S	S	S	S	19
Area N2 1984	S	R	S	S	S	S	S	S	S	S	14
	S	S	S	S	S	S	S	S	R	S	3
	R	S	S	S	S	R	S	S	R	R	2
	S	S	S	S	S	S	S	S	S	S	25
Area N3 1983	R	S	S	S	S	-	-	-	-	-	1
	S	R	S	S	S	-	-	-	-	-	2
	S	S	R	S	S	-	-	-	-	-	1
	S	S	S	R	S	-	-	-	-	-	1
	R	R	S	S	S	-	-	-	-	-	1
	R	S	R	S	S	-	-	-	-	-	1
	R	R	R	R	R	-	-	-	-	-	2
	S	S	S	S	S	-	-	-	-	-	31
Area N3 1984	R	R	S	S	S	S	S	S	S	S	1
	S	R	S	S	S	S	S	S	R	R	1
	S	S	S	S	S	S	R	S	S	S	4
	S	S	S	S	S	S	R	R	S	S	1
	S	S	S	S	S	R	S	S	S	R	1
	S	R	R	R	S	S	R	R	S	S	1
	S	S	S	S	S	R	S	S	S	S	1
	S	S	S	S	S	S	S	S	S	R	1
	S	S	S	S	S	S	R	R	R	R	1
	S	S	S	S	S	R	R	S	R	R	1
	S	S	S	S	S	S	S	R	S	S	1
	S	S	S	S	S	S	R	S	R	S	1
	S	S	S	S	S	S	S	S	R	S	1
	S	S	R	S	S	S	R	S	S	R	1
	S	S	S	R	S	R	R	R	R	R	1
	S	S	S	S	S	R	R	R	S	R	1
	S	S	S	S	S	S	S	S	S	S	41

obtained by soil sampling were found to be resistant to one or more isolates. The phenotypes are given in Table 7.1. There appears to be little difference between sampling methods in the efficiency of detecting resistance phenotypes.

7.3.3. A comparison between frequencies of resistance in four 2 m by 4 m plots within Garscube gardens.

Twenty-five plants were obtained from each of Plots 1, 2, 3 and 4, and tested with isolates G8, G9, G10, G11 and G12. The numbers of plants resistant to one or more of the isolates on each of the plots were 2, 1, 0 and 1 respectively. (See Table 7.1 and Fig 7.2)). Both resistant plants from Plot 1 and the single plant from Plot 4 were resistant to isolate G12 only. The resistant plant from Plot 2 was resistant to isolate G9 only. The number of resistant plants detected was too small to make a valid comparison between the plots but it does appear that resistance to G12 may be more common than resistance to other isolates.

7.3.4. A comparison between the frequency of resistance within three different sizes of sampling area at Glasgow.

A study was made to determine the effect of size of the sampling area on the detected frequency of plants with resistance. The three areas sampled were as follows:

Area GL1. The total area of the four plots 4 m by 2 m. (100 plants assessed. Table 7.1. Fig 7.2).

Area GL2. The area within the walled garden at Garscube (approximately 75 m by 100 m). (52 plants assessed. Table 7.1. Fig 7.3.).

Area GL3. An area of just over 1.2 square km, its centre being located within the walled garden at Garscube gardens. (46 plants assessed. Table 7.1. Fig 7.4.).

Key to Figs 7.2 to 7.9. The resistance phenotypes represented by each letter.

Upper case letter denotes all ten isolates considered, lower case letter denotes only the 5 Glasgow isolates were used for testing.

o = All susceptible phenotype.

Resistance phenotype					
G8	G9	G10	G11	G12	Letter
R	+	+	+	+	a
+	R	+	+	+	b
+	+	R	+	+	c
+	+	+	R	+	d
+	+	+	+	R	e
R	R	+	+	+	k
R	+	R	+	+	l
+	R	+	R	+	n
+	R	+	+	R	p
R	R	R	+	+	q
R	R	+	R	R	x
R	R	R	R	R	y

Resistance phenotype										
G8	G9	G10	G11	G12	N1	N4	N5	N7	N11	Letter
+	R	+	+	+	+	+	+	+	+	A
+	+	+	R	+	+	+	+	+	+	D
+	+	+	+	R	+	+	+	+	+	E
+	+	+	+	+	R	+	+	+	+	F
+	+	+	+	+	+	R	+	+	+	G
+	+	+	+	+	+	+	R	+	+	H
+	+	+	+	+	+	+	+	R	+	I
+	+	+	+	+	+	+	+	+	R	J
R	+	R	+	+	+	+	+	+	+	L
+	R	R	+	+	+	+	+	+	+	M
+	+	+	+	+	R	R	R	+	+	T
+	+	R	+	+	+	R	+	+	R	U
+	R	+	+	+	+	+	+	R	R	V
+	+	+	+	+	R	+	+	R	R	W
R	R	R	R	R	+	+	+	+	+	Y
R	+	R	+	+	R	+	+	R	R	Z
R	+	+	+	+	R	+	+	R	R	AA
R	R	R	+	+	+	R	R	+	+	BB
R	+	R	R	+	R	+	R	+	+	CC
+	+	+	+	+	+	R	R	R	R	DD
+	+	+	+	+	R	R	+	R	R	EE
+	+	+	R	+	R	R	R	R	R	FF
+	+	+	+	+	+	R	R	+	+	GG
+	+	+	+	+	R	+	+	+	R	HH
+	+	+	+	+	+	R	+	R	+	II
R	R	R	R	R	R	R	R	R	R	RR

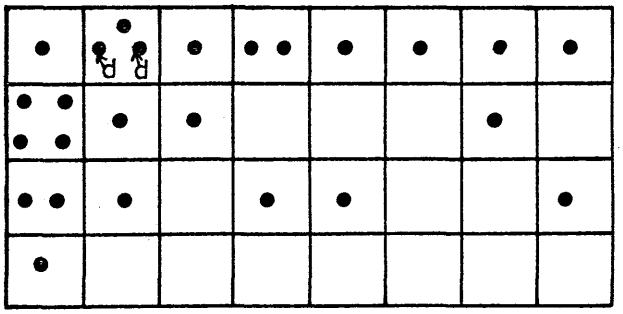
Fig 7.2

GROUNDSEL SAMPLING POINTS AREA GL1 1983

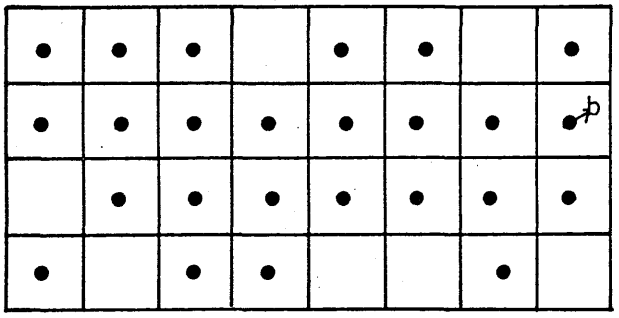
FOUR 4m x 2m PLOTS

= APPROX 1m

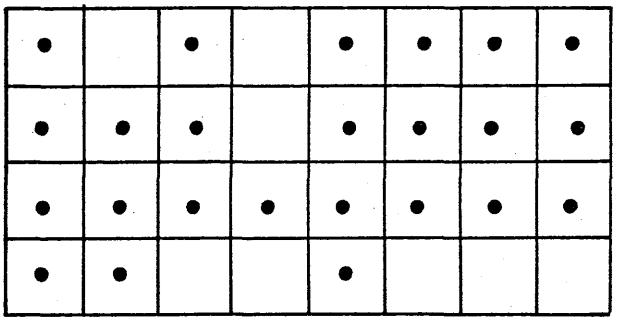
PLOT 1



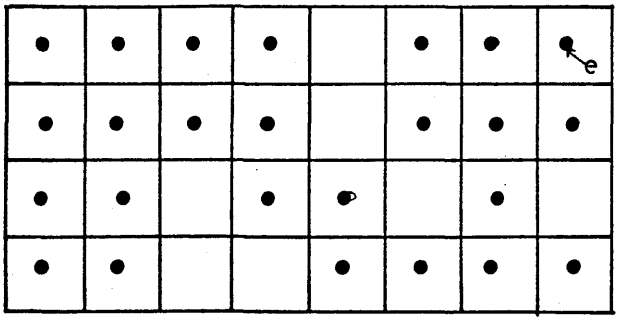
PLOT 2



PLOT 3.



PLOT 4.

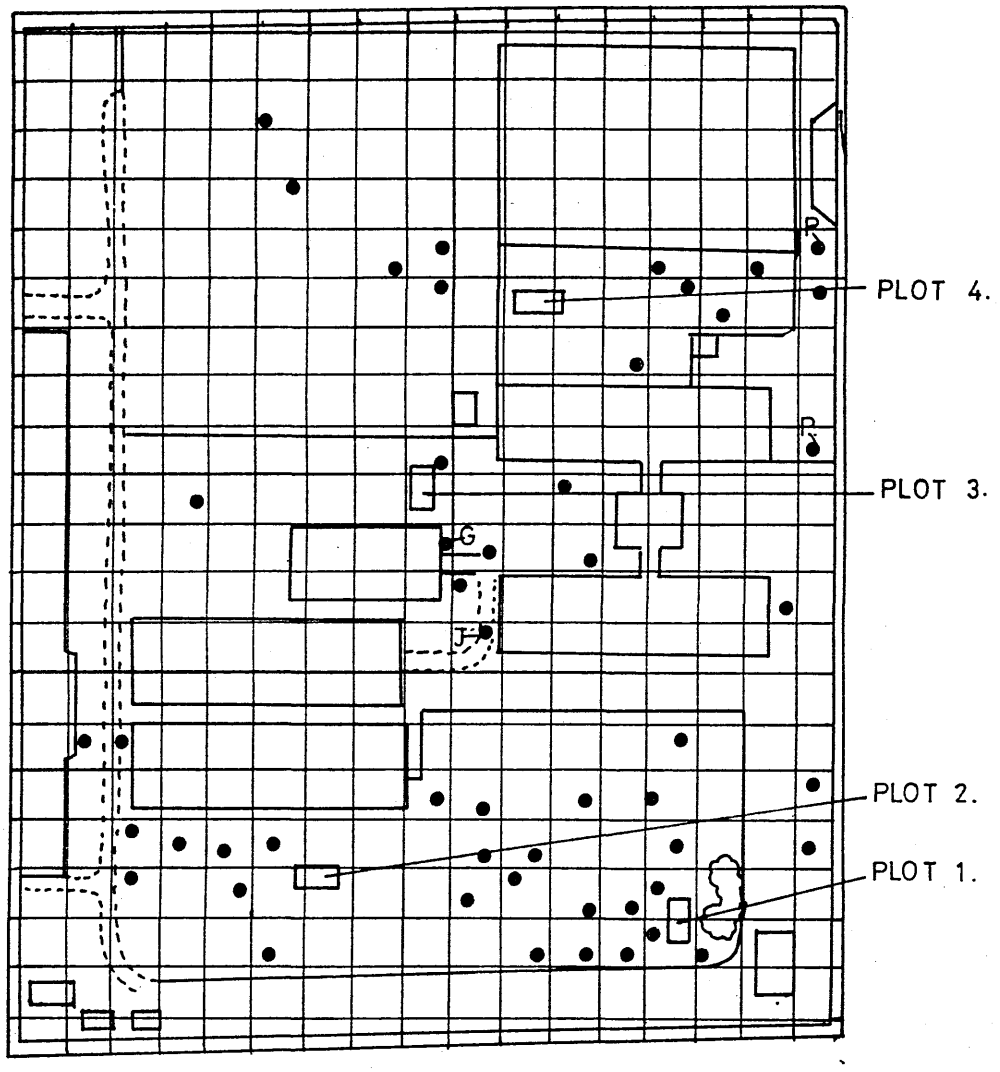


SEE KEY TO FIGS 7·2 TO 7·9 FOR RESISTANCE PHENOTYPE FOR EACH LETTER

Fig 7.3

GROUNDSEL SAMPLING POINTS AREA GL2 1983
GARSCUBE GARDENS

⟷ = APPROX 5m



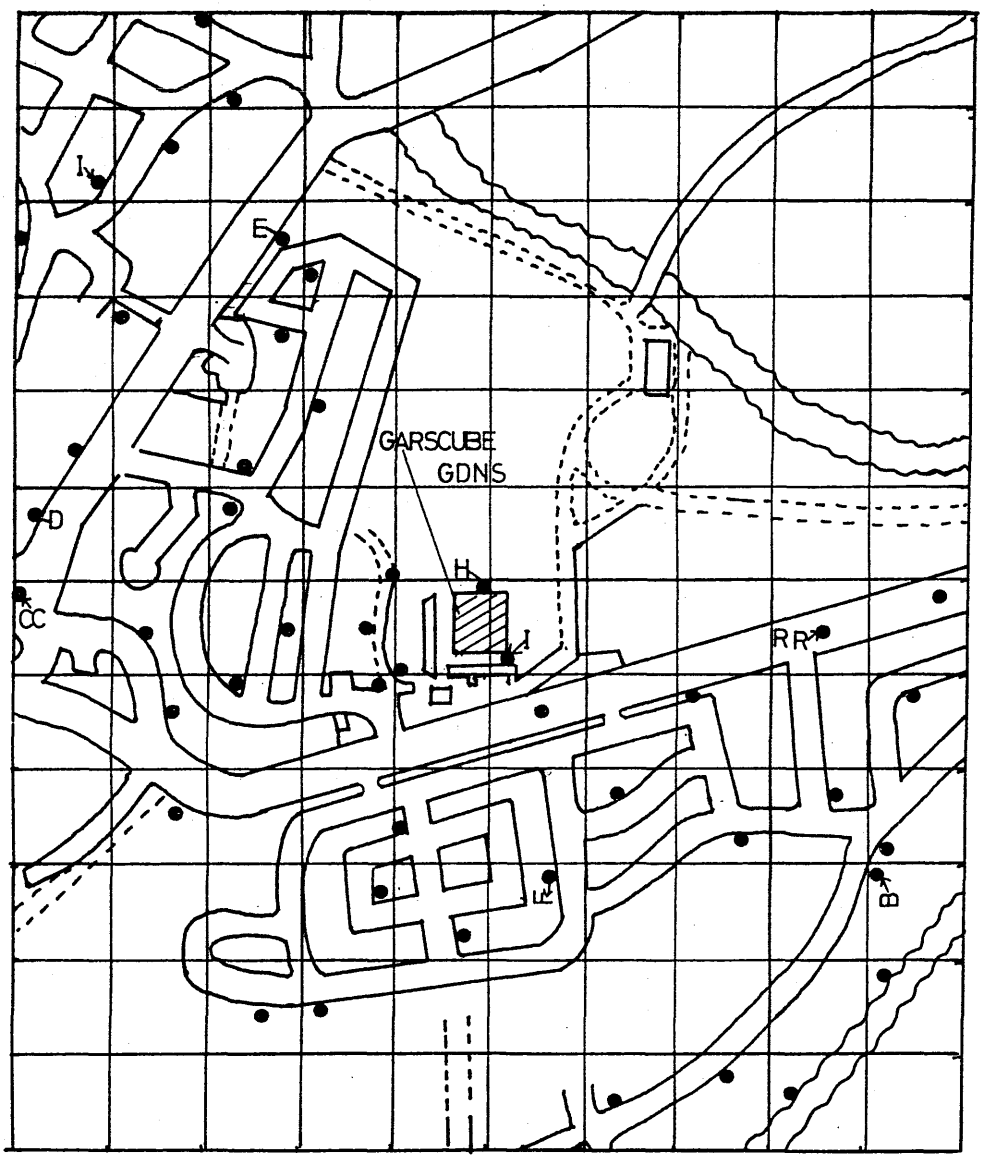
SEE KEY TO FIGS 7·2 TO 7·9 TO FIND WHICH RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

Fig 7·4

GROUNDSEL SAMPLING POINTS AREA GL3 1983

THE GENERAL GLASGOW AREA

= APPROX 100m



SEE KEY TO FIGS 7·2 TO 7·9 TO FIND RESISTANCE PHENOTYPE THE LETTERS REPRESENT

The number of plants possessing resistance to one or more of the isolates G8, G9, G10, G11 and G12 from each of the areas GL1, GL2 and GL3 were 4, 2 and 5 respectively. Statistical analysis (χ^2 = 3.21, P =0.05) showed that there was no significant difference between the sampling areas in the frequency with which resistant plants were detected. However, if one considers the data of a single 2 m by 4 m plot as the smallest sampling area, then as anticipated, the results do indicate that as the size of sampling area is increased then there is a tendency for the number of resistant individuals detected to increase.

The resistance phenotypes detected in the populations are given in Table 7.1. With the five Glasgow isolates on the established plants from area GL1, Plot 1 had two plants with resistance to isolate G12, Plot 2 had one plant with resistance to isolate G9, Plot 3 had no resistant plants, and Plot 4 had one plant with resistance to isolate G12 only. Area GL2 only had one plant with resistance and that was to isolates G9 and G12, while area GL3 yielded 5 resistant plants with 5 different resistance phenotypes.

Figs 7.2, 7.3 and 7.4 represent the spatial distribution of the resistance phenotypes. Resistance did not appear to be concentrated in any particular area but as expected the number of different resistance phenotypes was greatest in the largest sampling area.

7.3.5. A comparison between different sized sampling areas at N.V.R.S..

Three different sizes of sampling area were also studied at the N.V.R.S. Isolates G8, G9, G10, G11 and G12 were again used to test plants obtained from the three areas. The areas which were sampled in 1983 were as follows.

A. Area N1; a plot 1 m^2. (75 plants assessed. See Table 7.1, Fig 7.5).

B. Area N2; a plot 17 m x 17 m sampled 1983. (47 plants assessed. See Table 7.1, Fig 7.6).

C. Area N3; the area within the N.V.R.S station boundaries sampled in 1983. (40 plants assessed. See Table 7.1, Fig 7.8).

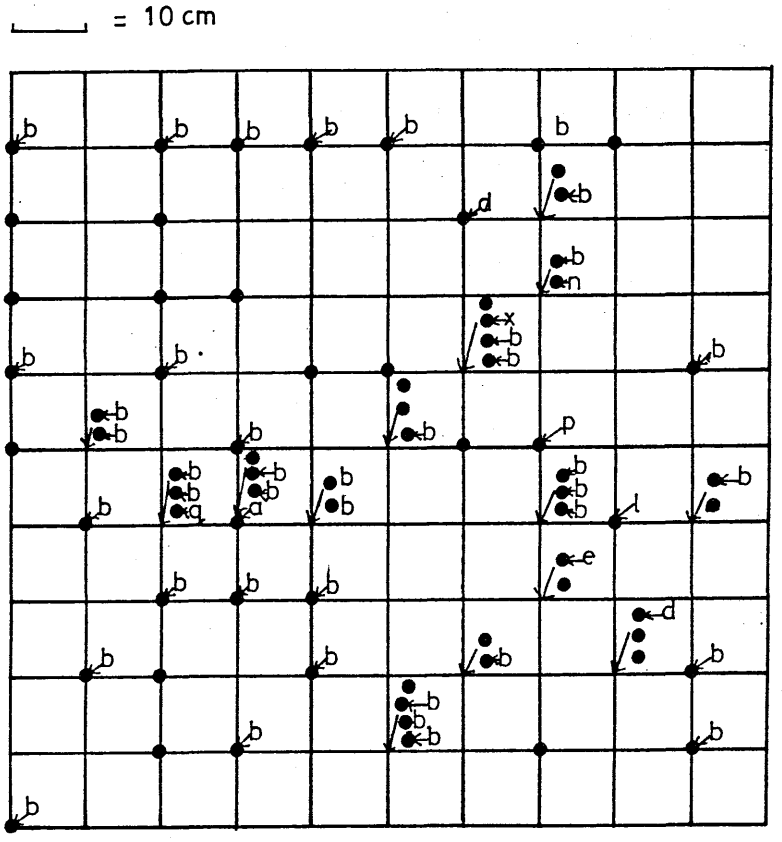
The number of plants from each sampling area N1, N2 and N3 found to possess resistance to one or more isolates were 52(69%), 27(57%) and 9(23%) respectively. Analysis of the data demonstrated a significant difference between the frequencies of resistant plants detected in each area (χ^2 = 23.81, P < 0.001). Unexpectedly, at N.V.R.S. as sample area increased the number of resistant plants detected decreased. However, an examination of the frequencies of the various resistance phenotypes (see Table 7.1) revealed that the high frequency of resistant plants from the two smaller areas was mainly due to the high frequency of plants with resistance to isolate G9 only. When the reactions resistant to G9 only were excluded from the analysis, then the frequency of plants with resistance to the remaining four isolates (G8, G10, G11 and G12) from the Areas N1, N2 and N3 was 9 (12%), 5 (11%) and 7 (18%) respectively. The χ^2 analysis of this data was not significant (χ^2 =1.02, P > 0.05). Diagrams 7.5, 7.6 and 7.8 represent the spatial distribution of the various phenotypes detected within the areas N1, N2 and N3.

7.3.6. Variation in the frequency of resistant plants between years. Seed collections were made in areas N2 and N3 in both 1983 and 1984 (See Figs 7.6 and 7.7, and 7.8 and 7.9). The plants raised from them were tested for resistance to isolates G8, G9, G10, G11 and G12. The

Fig 7.5

GROUNDSEL SAMPLING POINTS
AREA N1 1983

1 m^2 PLOT

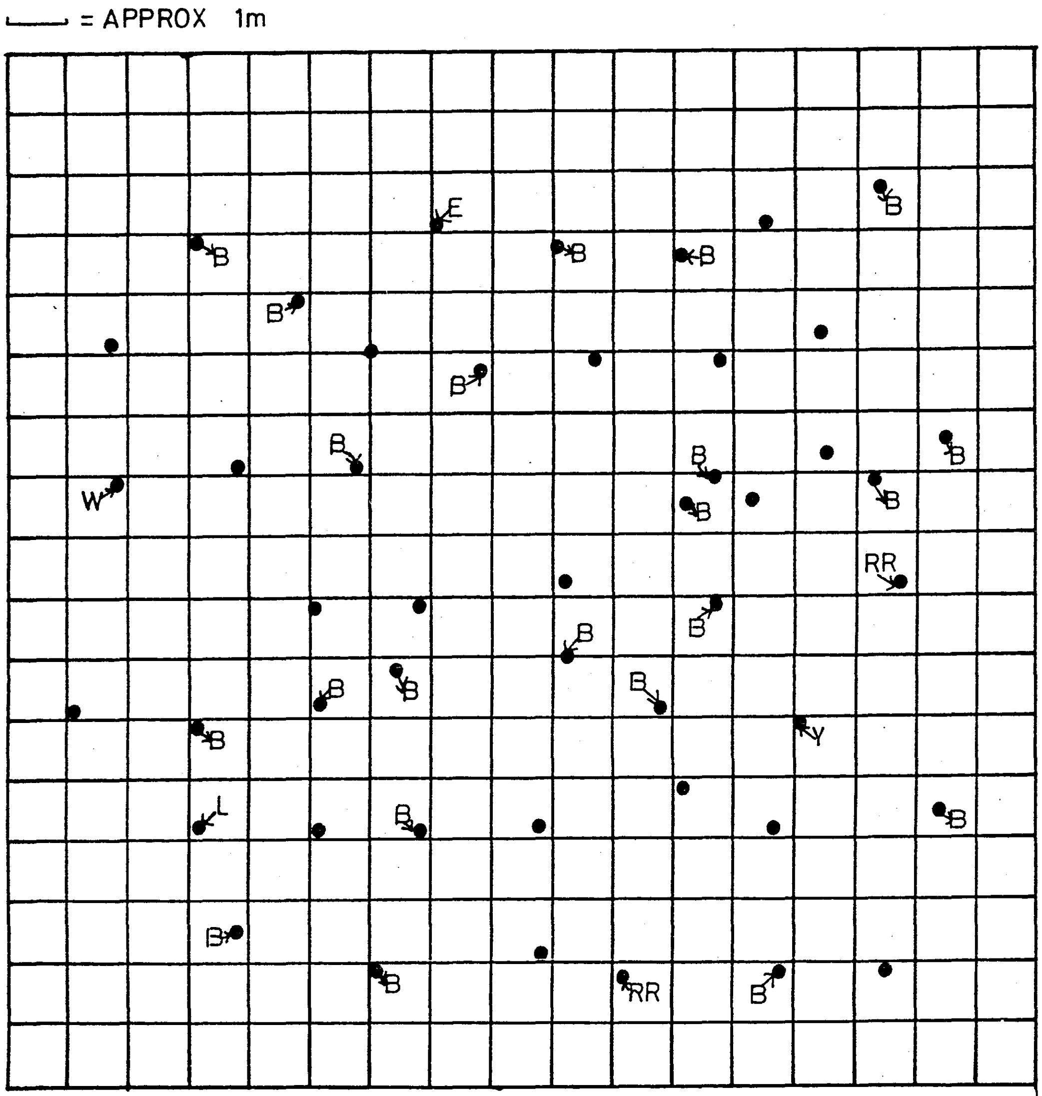


SEE KEY TO FIGS 7.2 TO 7.9 TO FIND WHICH RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

Fig 7.6

GROUNDSEL SAMPLING POINTS <u>AREA N2 1983</u>

17m x 17m PLOT AT N.V.R.S.

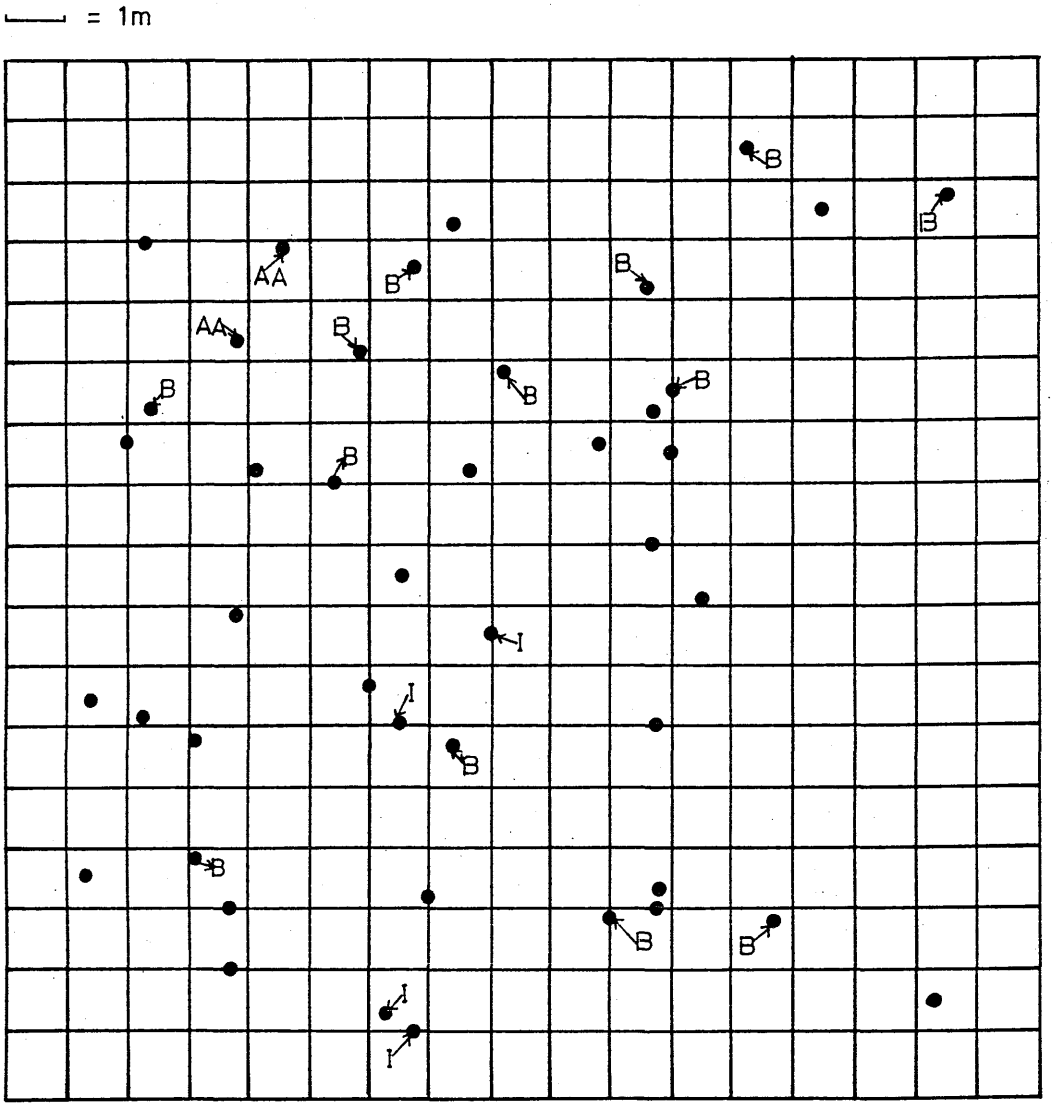


SEE KEY TO FIGS 7·2 TO 7·9 TO FIND WHICH RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

Fig 7.7

GROUNDSEL SAMPLING POINTS AREA N2 1984

17m x 17m PLOT AT N.V.R.S

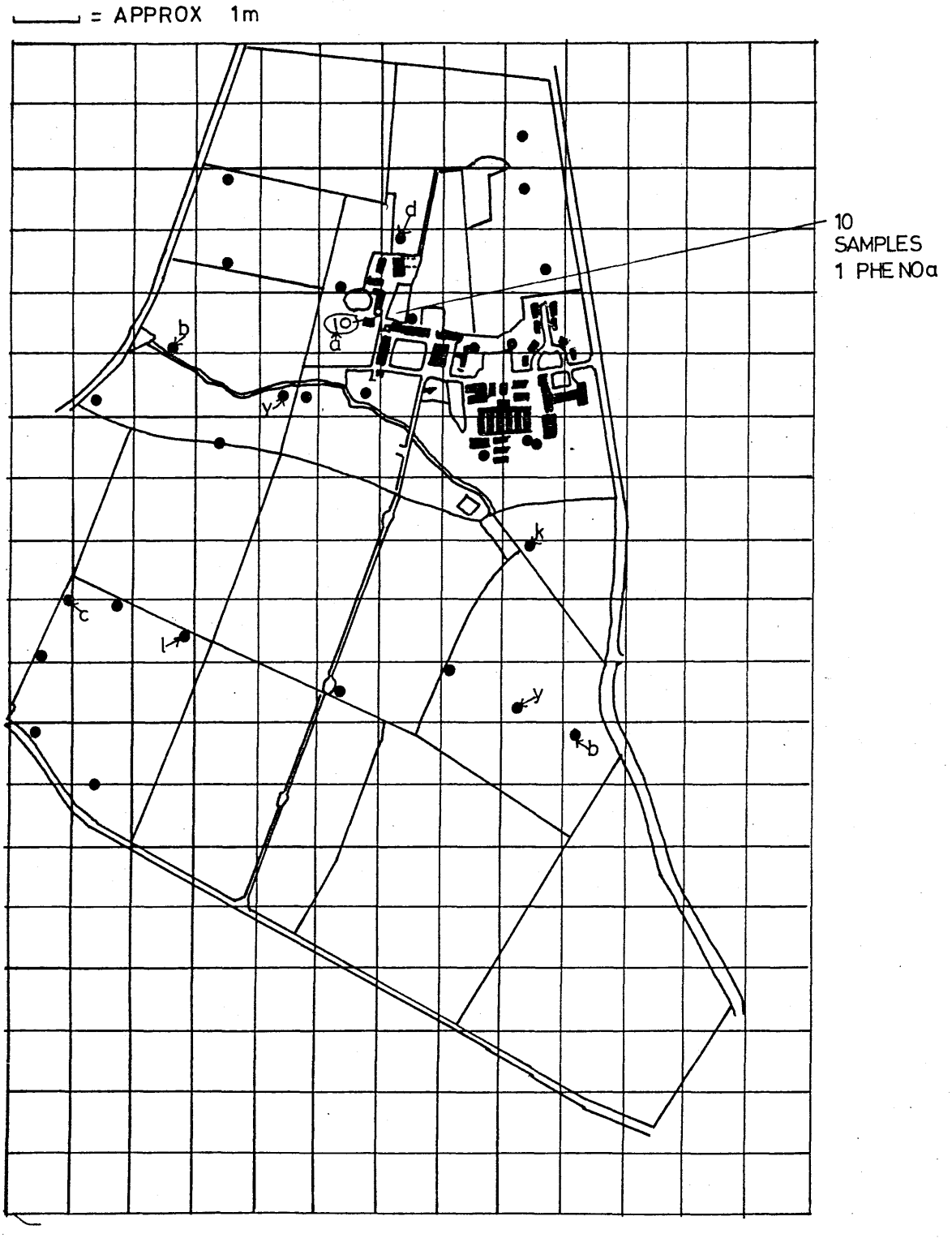


SEE KEY TO FIGS 7.2 TO 7.9 TO FIND WHICH RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

Fig 7·8

GROUNDSEL SAMPLING POINTS AREA N3 1983

GENERAL AREA WITHIN N.V.R.S. BOUNDRIES

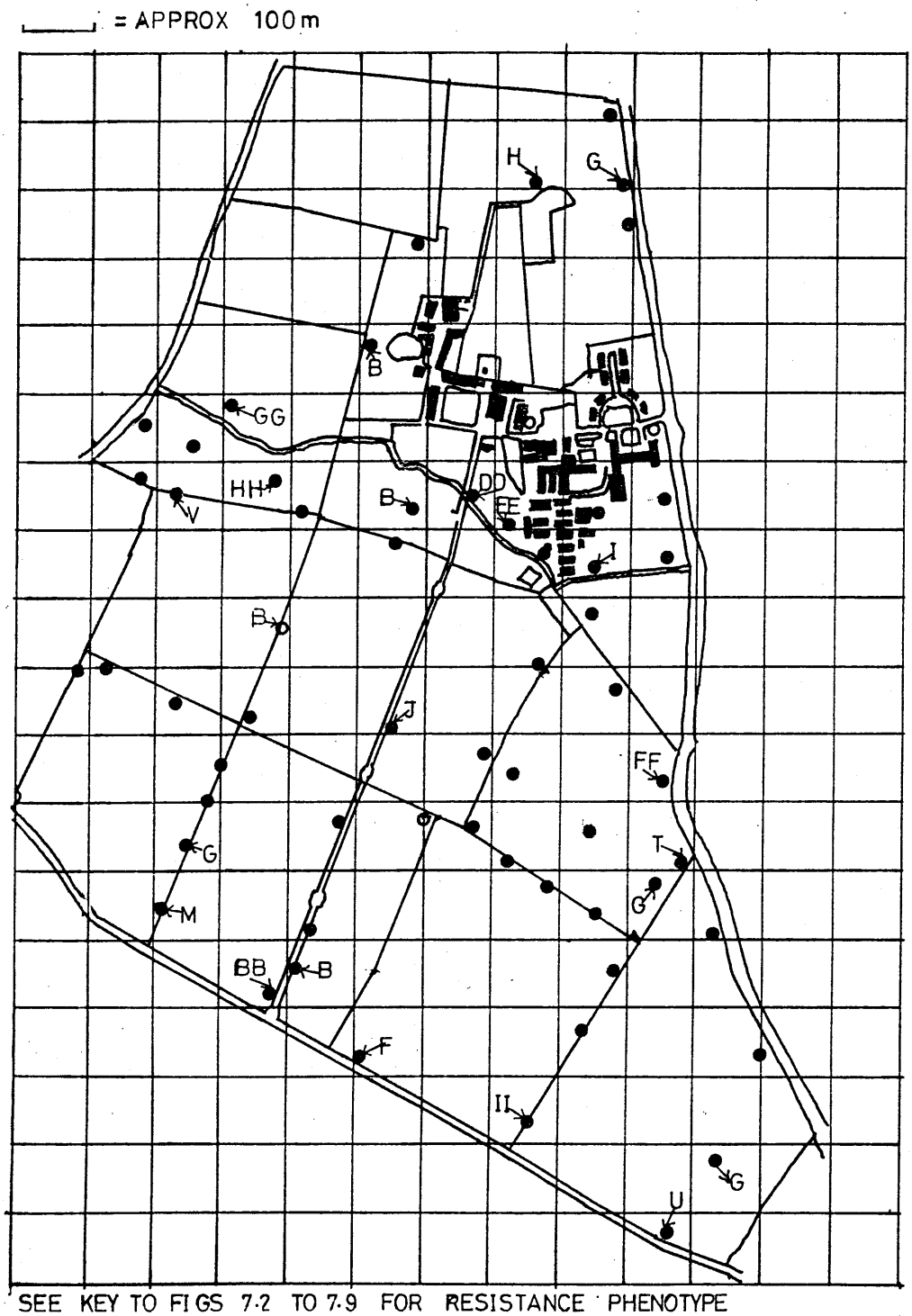


SEE KEY TO FIGS 7·2 TO 7·9 FOR RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

Fig 7.9

GROUNDSEL SAMPLING POINTS AREA N3 1984

GENERAL AREA WITHIN N.V.R.S BOUNDRIES



SEE KEY TO FIGS 7.2 TO 7.9 FOR RESISTANCE PHENOTYPE EACH LETTER REPRESENTS.

number of plants found to be resistant to at least one isolate are given in Table 7.2.

Table 7.2. Variation between years, including resistance to isolate G9.

	1983 SAMPLE		1984 SAMPLE	
	Number of resistant plants	Total number of plants assessed	Number of resistant plants	Total number of plants assessed
Area N2	27	47	16	44
Area N3	9	40	9	60

Analysis of the combined data for both years demonstrated a significant difference ($\chi^2 = 18.76$, $P < 0.001$) in the number of resistant plants between the two areas N2 and N3. The frequency of resistance to the five isolates in area N2 was also found to be significantly greater in 1983 than in 1984 ($\chi^2 = 4.05$, $P < 0.05$). However, no differences were found between years in area N3 ($\chi^2 = 0.92$, $P > 0.05$).

Again many of the plants in Area N2 were resistant to G9 only in both 1983 and 1984. The large number of plants with resistance to isolate G9 only may be explained by the tendency of phenotypes of inbreeding plants to be patchily distributed. Either by chance or by past selection Area N2 had a large proportion of plants with resistance to isolate G9. The data were examined excluding the resistant reactions to isolate G9. The numbers of plants resistant to the other four isolates are shown in Table 7.3.

Variation between years, excluding resistance to isolate G9.

	1983 SAMPLE		1984 SAMPLE	
	Number of resistant plants	Total number of plants assessed	Number of resistant plants	Total number of plants assessed
Area N2	4	47	2	44
Area N3	7	40	4	60

Analysis of the data for area N2 showed that there was no significant difference between years (χ^2 =0.58, P > 0.05), indicating that only the frequency of G9 varied between years. However, analysis of the data for the larger area N3 showed that there was a significant difference between the frequency of resistance to the four isolates between years (χ^2 =5.11, P < 0.05).

7.3.7. A detailed study of the 1 m by 1 m plot.

Area N1, the 1 m^2 plot with the highest density of groundsel on the 17 m x 17 m plot (area N2) was intensively sampled in 1983. Seed was collected from every plant which fruited within this square during July. One plant originating from each of the samples was tested with isolates G8, G9, G10, G11 and G12. A large number of plants, 43 out of the 75 tested (or 57%), were resistant to isolate G9 only. Many other phenotypes, including plants that were susceptible to all five mildew isolates were also found. The resistant phenotypes found are given in Table 7.1. Including the phenotype which was susceptible to all five isolates a total of 10 different phenotypes was detected within this small area. This clearly confirms that the groundsel population is highly heterogeneous. Fig 7.5 represents the distribution of the resistance phenotypes on the plot.

7.3.8. Comparison between the frequency of resistant plants at Glasgow and N.V.R.S. to both sets of isolates.

The frequency of plants with resistance to at least one isolate in the N.V.R.S. and Glasgow groundsel populations was compared. The reactions of ninety-eight plants from Glasgow (52 from GL2 and 46 from GL3 collected in 1983) and 151 plants from N.V.R.S. (47 from N2 collected in 1983, 44 from N2 collected in 1984, and 60 from area N3 collected in 1984) to Glasgow isolates G8, G9, G10, G11, and G12, and to the N.V.R.S. isolates N1, N4, N5, N7 and N11 were assessed. The numbers of plants showing resistance to at least one isolate from each set of isolates is given in Table 7.4.

Table 7.4. N.V.R.S. and Glasgow populations tested with each set of isolates.

	N.V.R.S PLANTS		GLASGOW PLANTS	
	Resistant	Total	Resistant	Total
Glasgow isolates	52(34%)	151	7(7%)	98
N.V.R.S isolates	29(19%)	151	9(9%)	98

Analysis revealed that the Glasgow plants were equally susceptible to both the Glasgow and the N.V.R.S isolates ($\chi^2 = 0.27$, $P > 0.05$). However, the N.V.R.S. plants were more resistant to the Glasgow than to the N.V.R.S. isolates ($\chi^2 = 8.92$, $P < 0.01$). Likewise, the Glasgow isolates ($\chi^2 = 24.49$, $P < 0.001$) and to a lesser extent the N.V.R.S. isolates ($\chi^2 = 4.16$, $P < 0.05$) were significantly less virulent on the N.V.R.S. plants than on the Glasgow plants. These results indicate that most of the differences between the two host samples were due to a higher frequency of resistance in the N.V.R.S. population.

Many of the resistant plants from the N.V.R.S. were resistant to isolate G9 only. The greater frequency of resistance to isolate G9 may have accounted for the main differences between the two groundsel populations from the N.V.R.S. and Glasgow. Table 7.5 summarizes the

data for the number of resistant plants obtained at each location, once resistance to G9 only was omitted.

Table 7.5. N.V.R.S. v Glasgow plants, resistance to G9 omitted.

	N.V.R.S. PLANTS		GLASGOW PLANTS	
	Resistant	Total	Resistant	Total
G8,G10,G11,G12	11 (7%)	151	6 (6%)	98
N.V.R.S. ISOLATES	29 (19%)	151	9 (9%)	98

Analysis revealed, as before, that the Glasgow plants were equally susceptible to both the Glasgow and the N.V.R.S. isolates (χ^2 =0.65, P > 0.05). However, in this case, more of the N.V.R.S. plants were resistant to the N.V.R.S. isolates than to the Glasgow isolates (χ^2 = 9.34, P < 0.01). Equal numbers of plants from N.V.R.S and Glasgow were resistant to the Glasgow isolates (χ^2 =0.13, P > 0.05) but more plants from the N.V.R.S. than from Glasgow were resistant to the N.V.R.S. isolates (χ^2 =4.62, P < 0.05). This analysis indicates some kind of an interaction between N.V.R.S. isolates and N.V.R.S. plants, resulting in a higher number of resistant plants than expected using this combination of isolates and plants.

An analysis was then carried out to try to determine whether the interaction obtained between the N.V.R.S. isolates and N.V.R.S. plants could be explained by differences in resistance between the plant populations alone or whether differences between the mildew populations were also involved. A summary of resistance in each groundsel population to all the isolates (but excluding plants resistant to G9 only) is given in Table 7.6.

Table 7.6. Resistance in the N.V.R.S. and Glasgow plants to all isolates combined but excluding isolate G9.

	SUSCEPTIBLE PLANTS	RESISTANT PLANTS
TOTAL GLASGOW PLANTS =98	86	12 (12%)
TOTAL N.V.R.S. PLANTS =151	121	30 (20%)

There was no significant difference between the two groundsel populations with respect to their overall reactions to the 9 isolates ($\chi^2 = 2.25$, $P > 0.05$). This indicates that the observed interaction between the N.V.R.S. plants and the N.V.R.S. isolates may be due to differences between the mildew populations.

The total numbers of different resistance phenotypes, including resistance to isolate G9, in the Glasgow and N.V.R.S. groundsel populations were 12 and 24 respectively (including the all susceptible phenotype). The phenotypes are given in Tables 7.7 and 7.8 respectively. There appears to be greater heterogeneity in the N.V.R.S. population with greater heterogeneity being detected by the N.V.R.S. isolates. The resistance phenotypes given in the two tables may be explained by a minimum of 9 genes for the Glasgow plants and a minimum of 10 genes for the N.V.R.S. plants if the logic of the 'gene-for-gene' relationship is applied. The maximum number of phenotypes possible in the Glasgow and N.V.R.S. populations are, therefore, 2^9 =515 and 2^{10} =1,024 respectively. The percentage of the possible phenotypes that were detected in each population was the same being 2.3%.

Table 7.7 Resistance phenotypes detected in the Glasgow population (98 plants) using 10 isolates

Phenotype in both populations	Isolate G8	G9	G10	G11	G12	N1	N4	N5	N7	N11
Yes	S	R	S	S	S	S	S	S	S	S
Yes	S	S	S	S	R	S	S	S	S	S
Yes	S	S	S	S	S	R	S	S	S	S
Yes	S	S	S	S	S	S	R	S	S	S
Yes	S	S	S	S	S	S	S	R	S	S
Yes	S	S	S	S	S	S	S	S	R	S
Yes	S	S	S	S	S	S	S	S	S	R
Yes	R	R	R	R	R	R	R	R	R	R
Yes	S	S	S	S	S	S	S	S	S	S
No	S	S	S	R	S	S	S	S	S	S
No	S	R	S	S	R	S	S	S	S	S
No	R	S	R	R	S	R	S	R	S	S

12 Phenotypes, minimum number of genes required =9

Table 7.8. Resistance phenotypes detected in the N.V.R.S. population (151 plants) using 10 isolates.

Phenotype in both populations	ISOLATE G8	G9	G10	G11	G12	N1	N4	N5	N7	N11
Yes	S	R	S	S	S	S	S	S	S	S
Yes	S	S	S	S	R	S	S	S	S	S
Yes	S	S	S	S	S	R	S	S	S	S
Yes	S	S	S	S	S	S	R	S	S	S
Yes	S	S	S	S	S	S	S	R	S	S
Yes	S	S	S	S	S	S	S	S	R	S
Yes	S	S	S	S	S	S	S	S	S	R
Yes	R	R	R	R	R	R	R	R	R	R
Yes	S	S	S	S	S	S	S	S	S	S
No	S	R	R	S	S	S	S	S	S	S
No	S	R	S	S	S	S	S	S	R	R
No	S	S	S	S	S	S	R	R	S	S
No	S	S	S	S	S	R	S	S	S	R
No	R	R	R	S	S	S	R	R	S	S
No	S	S	S	S	S	S	R	R	R	R
No	S	S	S	S	S	R	R	S	R	R
No	S	S	S	S	S	S	R	S	R	S
No	S	S	R	S	S	S	R	S	S	R
No	S	S	S	R	S	R	R	R	R	R
No	S	S	S	S	S	R	R	R	S	S
No	R	S	S	S	S	R	S	S	R	R
No	S	S	S	S	S	R	S	S	R	R
No	R	S	R	S	S	R	S	S	R	R
No	R	R	R	R	R	S	S	S	S	S

24 Phenotypes, minimum number of genes required =10

7.3.8. The resistance phenotypes of the Glasgow and N.V.R.S. groundsel populations with respect to the five Glasgow isolates G8, G9, G10, G11 and G12.

Additional plants were tested using the five Glasgow isolates only. The total number of Glasgow plants tested was 247, and included;

100 plants from the four 2m by 4m plots (Area GL1, Fig 7.2).

52 plants from area GL2 (Fig 7.3).

46 plants from area GL3 (Fig 7.4).

49 plants obtained from soil sampling.

The total number of N.V.R.S plants tested was 266, and included;

75 plants from area N1 (Fig 7.5).

47 plants collected from area N2 in 1983 (Fig 7.6).

44 plants from area N2 in 1984 (Fig 7.7).

40 plants from area N3 in 1983 (Fig 7.8).

60 plants from area N3 in 1984 (Fig 7.9).

It was found that 12 plants (5%) from Glasgow and 113 plants (42.5%) from the N.V.R.S. possessed resistance to one or more of the isolates. This difference was highly significant ($\chi^2 = 99.25$, $P < 0.001$).

When resistance to isolate G9 is disregarded then it can be seen that 11 plants from Glasgow and 17 plants from the N.V.R.S. possess resistance to one or more of the other four isolates. Analysis revealed that there was no significant difference between the groundsel populations as far as resistance to isolates G8,G10,G11 and G12 were concerned ($\chi^2 = 1.8$, $P > 0.05$.

The number of different resistance phenotypes (including the all susceptible) detected in the Glasgow population was 7 and in the N.V.R.S population was 14. The phenotypes are shown in Tables 7.9 and

Table 7.9. Resistance phenotypes detected in Glasgow groundsel population (247 plants) using 5 Glasgow isolates.

Phenotypes in both populations	Isolate G8	G9	G10	G11	G12
Yes	S	R	S	S	S
Yes	S	S	S	R	S
Yes	S	R	S	S	R
Yes	R	R	R	R	R
Yes	S	S	S	S	S
No	S	S	S	S	R
No	R	S	R	R	S

7 Phenotypes, minimum number of genes required = 4

Table 7.10. Resistance phenotypes detected in the N.V.R.S. groundsel population (266 plants) using the 5 Glasgow isolates.

Phenotype in both populations	ISOLATE G8	G9	G10	G11	G12
Yes	S	R	S	S	S
Yes	S	S	S	R	S
Yes	S	S	S	S	R
Yes	S	R	S	S	R
Yes	R	R	R	R	R
Yes	S	S	S	S	S
No	S	R	R	S	S
No	R	R	S	S	S
No	S	R	S	R	S
No	R	S	R	S	S
No	R	S	S	S	S
No	R	R	R	S	S
No	R	R	S	R	R
No	S	S	R	S	S

14 phenotypes, minimum number of genes required = 5

7.10 respectively. The Glasgow phenotypes may be explained by a minimum of 4 genes and the N.V.R.S. by 5 genes. The number of phenotypes possible using these isolates is 2^4 =16 and 2^5 =32 respectively. The percentages of the possible phenotypes that were actually detected were 44% in both cases.

7.4. DISCUSSION.

The evidence clearly indicates that the two populations of groundsel differ with respect to both the number of plants with race specific resistance and the phenotypes detectable with the isolates used. Of all the plants tested 7.6% of the Glasgow plants and 49% of the N.V.R.S. plants were resistant to at least one mildew isolate. The main difference between the populations could be accounted for by the large number of plants resistant to isolate G9 only in the N.V.R.S. population. However, small differences in relation to other resistant phenotypes between the populations were still apparent. Harry (1980) also found indications that resistance in groundsel varied geographically. Her work was based on relatively small samples of about 10 individuals collected from 24 different locations so that comparisons between sampling locations could not be made accurately. There are few reports in the literature of studies of the levels or distribution of resistance in wild populations. Perhaps the most relevant are those by Dinoor (1970) on crown rust resistance in wild oats and Burdon and Marshall (1981) on resistance in wild soya beans to leaf rust.

7.4.1. The proportion of plants with race specific resistance in wild populations.

Dinoor (1970) examined resistance in wild oat (*Avena sterilis*) populations to two races, 264 and 276, of oat crown rust (*Puccinia coronata avenae*) and found 10% of the plants tested were resistant to race 264, whilst 5.5% were resistant to race 276. Work by Wahl (1970) on the same host and pathogen found similar proportions of resistant plants. Out of 8,514 *Avena sterilis* seedlings tested, 492 or 5.7%

were found to be resistant to either or both races 264 and 276 of Puccinia coronata avenae. The proportion of resistant adult plants was higher, 498 out of 3,081 or 16.2% were resistant to one or both of the rust isolates.

Burdon and Marshall (1981) investigated the proportion of resistant plants in six species of Glycine to one isolate of Phakopsora pachyrhizi and found that the number of resistant plants varied depending on the Glycine species examined. Table 7.11 shows the percentage of resistant plants ranged from 13% to 42%.

Table 7.11. Percentage of resistant plants detected of different Glycine spp..

Glycine species	Number of plants tested	% of plants Resistant	Susceptible
G. canescens	23	13	87
G. clandestina	40	15	85
G. tabacina (2n)	29	14	86
G. tabacina (4n)	47	42	68
G. tabacina (2n+4n)	76	32	68
G. tomentela	45	33	67

The numbers of groundsel plants in each population at Glasgow and N.V.R.S. that were resistant to each mildew isolate are presented in Table 7.12. The proportion of plants resistant to each isolate ranges between 1% for isolates G8, G10 and G11 in the Glasgow plants, to 37% for isolate G9 in the N.V.R.S groundsel population. Apart from resistance to isolate G9 the percentage of plants resistant to each isolate ranged from 1% to 10%, a level similar to that found by Dinoor (1970) in the wild oat/crown rust pathosytem. It may also be noted that, except for resistance to isolate G12, there were slightly more plants resistant to each of the other isolates in the N.V.R.S. than in the Glasgow populations of groundsel.

Harry (1980) detected resistance in 149 out of 250 (59%) inbred groundsel lines, collected from various parts of the British Isles, to

Table 7.12. Number of groundsel in each population resistant to each mildew isolate.

Glasgow plant population		No plants resistant	Total no of plants assessed	% plants resistant
Isolate	G8	2	247	1%
	G9	7	247	3%
	G10	2	247	1%
	G11	3	247	1%
	G12	9	247	4%
	N1	3	98	3%
	N4	2	98	2%
	N5	3	98	3%
	N7	3	98	3%
	N11	1	98	1%
N.V.R.S. plant population				
Isolate	G8	15	266	6%
	G9	94	266	35%
	G10	13	266	5%
	G11	12	266	5%
	G12	9	266	3%
	N1	11	151	7%
	N4	14	151	9%
	N5	8	151	5%
	N7	15	151	10%
	N11	13	151	9%

one or more of 9 mildew isolates that she used for testing. However, not all the lines were tested with all nine isolates, but they were all tested with 5. The percentages of plants resistant to each isolate were:

20.4% resistant to isolate 1.
22.8% " " " 2.
17.6% " " " 3.
29.6% " " " 4.
23.6% " " " 5.

A greater proportion of the plants sampled by Harry (1980) were found to have resistance to one or more of the isolates used by Harry than was found in the present study using different isolates. The difference in the numbers of resistant plants detected were probably due to the different strategies used to sample the host and pathogen populations. The groundsel collections made by Harry (1980) were based on small samples of about 10 individuals from 24 widely dispersed locations in the British Isles. Within each location seed samples were taken from plants at least 30 m apart to avoid sampling from the same parentage. This type of method was found by Dinoor (1970) to maximise the numbers of resistant plants detected in the wild oat/crown rust pathosystem. The groundsel sampled in the present study were within two limited areas, one of approximately 1.2 km^2 at Glasgow and 6 km^2 at N.V.R.S. and this may have reduced the chances of detecting markedly different groundsel phenotypes. Each location was, however, more intensively sampled so that the probability of detecting differences between the two populations was increased greatly.

The sampling method used by Harry to obtain her mildew isolates may also have tended to maximise the number of resistance phenotypes that

could be detected. The first isolate obtained by Harry was collected at random since it was obtained from a groundsel plant of completely unknown resistance phenotype. Subsequent isolates were obtained from plants known to be resistant to the isolates tested earlier. One would expect that such a method would maximise the number of virulence phenotypes in the pathogen sample. In turn this may maximise the extent to which the isolates differ from one another so that they may detect larger numbers of different resistance phenotypes in the host and hence a greater number of resistant plants may be detected.

All the isolates used in the present study were obtained directly from different groundsel individuals in the field. Nothing was known about the resistance phenotypes of the hosts and in this respect the pathogen samples would be random and would tend to represent the more common isolates in the pathogen population.

7.4.2. The distribution of resistance between different locations in wild populations.

The work of Dinoor(1970) has provided a useful insight into the distribution of resistance in two species of wild oat (Avena sterilis and A. barbata) to two races of oat crown rust (Puccinia coronata avenae). In northern Israel 36.9% of the collections of A. sterilis were found to contain plants resistant to one or both of the rust races 264 and 276, whilst in southern Israel only 7.8% of the locations sampled were found to possess resistant individuals. One region, 25 km by 50 km, in northern Israel was found to have resistant individuals in 69.8% of the locations sampled. The distribution of resistance in another wild oat species Avena barbata was somewhat different. In the north of Israel only 6% of the locations were found to have seedling individuals with resistance to races 264 and 276 of oat crown rust. In southern Israel 35.3% of locations possessed

resistant A. barbata seedlings. One suggestion for the differences in the distributions of resistance between locations was that differences in the climate between the regions resulted in different selection on the host and that this was related to the time of year and stage of development that each species of host was at its most susceptible.

Studies by Burdon and Marshall (1981) on resistance of six species of Glycine to one single urediniospore isolate of the leaf rust fungus Phakopsora pachyrhizi indicated that there were no significant differences between the distribution of resistance in various populations of any of the host species. However, a maximum of only three individuals of each species in each location was tested. The small sample size makes the comparison of locations difficult, since only very obvious differences would be detected. It is not possible to be sure that any differences between populations were real or whether the differences were due to great variation in the host population as a whole. The results tended to indicate that there were greater numbers of susceptible plants in drier inland areas of New South Wales and Queensland. It was postulated that these differences could be due to the lower levels of the pathogen detected in drier areas, thus reducing the level of selection for resistance in the host.

It is difficult to determine the importance of environment on selection for race specific resistance in groundsel. Although powdery mildews are able to tolerate relatively wide ranges of temperature and humidity (Yarwood, 1957), powdery mildew epidemics on cereals are said to be more prevelent in dry weather conditions (Boughey, 1949 , and Cherewick, 1944). There is a higher rainfall and showers are more frequent at Glasgow than at the N.V.R.S. so one might expect the incidence of powdery mildew on groundsel to be greater at N.V.R.S. However, this does not appear to be the case. Observations in the

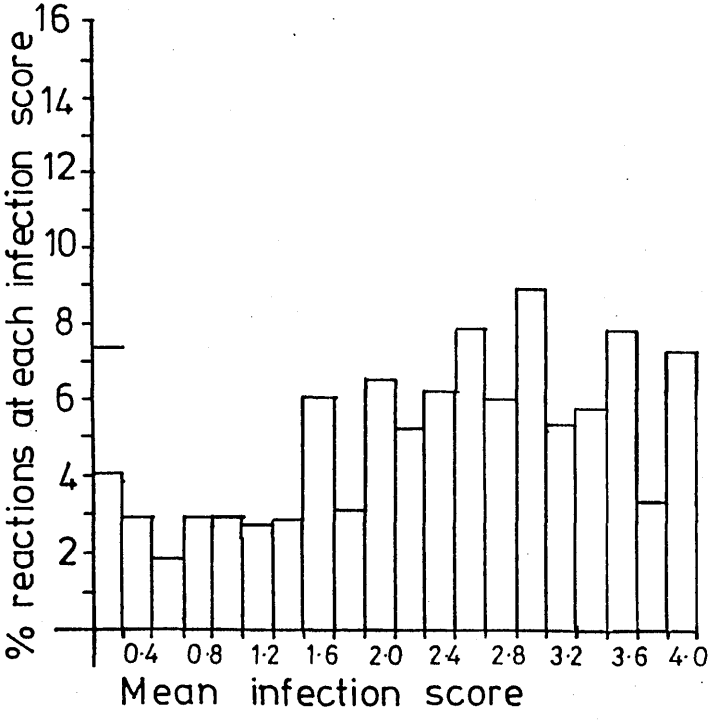
field indicated that the incidence of mildew was greater at Glasgow. Observations of mildew on the four 2 m x 4 m plots (Area GL1) before plants were harvested for seed in 1983, showed that the numbers of mildew infected plants were 27%, 56%, 35.5% and 86% on plots 1, 2, 3 and 4 respectively. At N.V.R.S. none of the plants on the 17 m x 17 m plot (Area N2) were observed to have mildew in 1983 and only 8% were infected with mildew in 1984. On the evidence of these observations one may then expect that selection for mildew resistance would be more important at Glasgow. However, the numbers of resistant plants and resistance phenotypes were greater at N.V.R.S than at Glasgow. Also, the number of resistant infection scores recorded over all tests with N.V.R.S. and Glasgow plants showed that 36 out of 1735 combinations or 2.1% were resistant in the case of the Glasgow plants whilst 208 out of a total of 2085 reactions tested or 10% were resistant in the case of the N.V.R.S. plants. It is possible to infer from this evidence that race specific resistance is functioning more effectively at N.V.R.S. than at Glasgow, since race specific resistance is more common but the incidence of the pathogen is lower at the N.V.R.S. However, it must be stressed that an extremely small proportion of the possible number of phenotypes of both the pathogen and the host were examined in this study. The sheer diversity in the system, although interesting, does make it difficult to determine whether differences detected in the populations are artifacts due to the particular isolates and plants used in the study.

It is also possible that the incidence of mildew in the field at N.V.R.S. may have been underestimated. An examination of the frequencies of the various mean infection scores obtained in each of the four combinations of isolate and plant populations presented in Figs 7.10 and 7.11 indicated that a higher proportion of the infection types in the Glasgow population were between 3 and 4 compared to the

Fig 7·10

% of reactions of each mean infection score for the N.V.R.S plants with each set of isolates.

N.V.R.S Isolates on N.V.R.S plants



Glasgow Isolates on N.V.R.S plants.

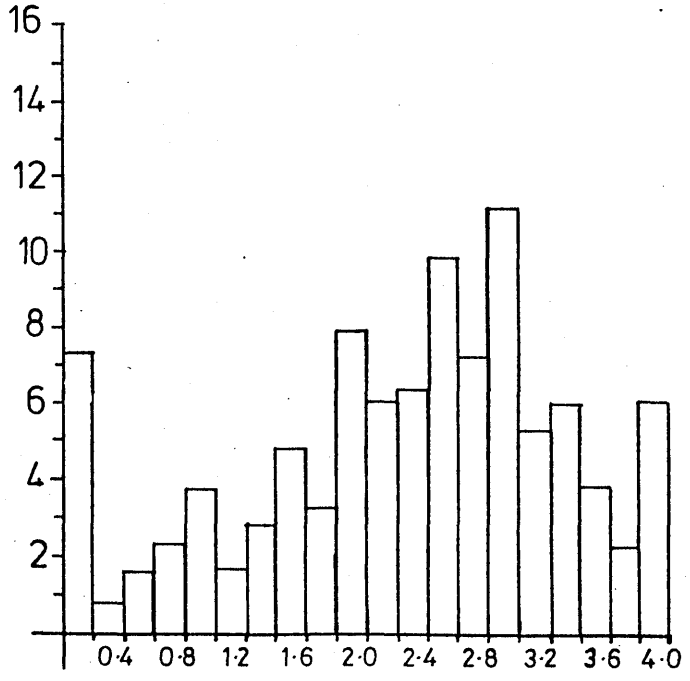
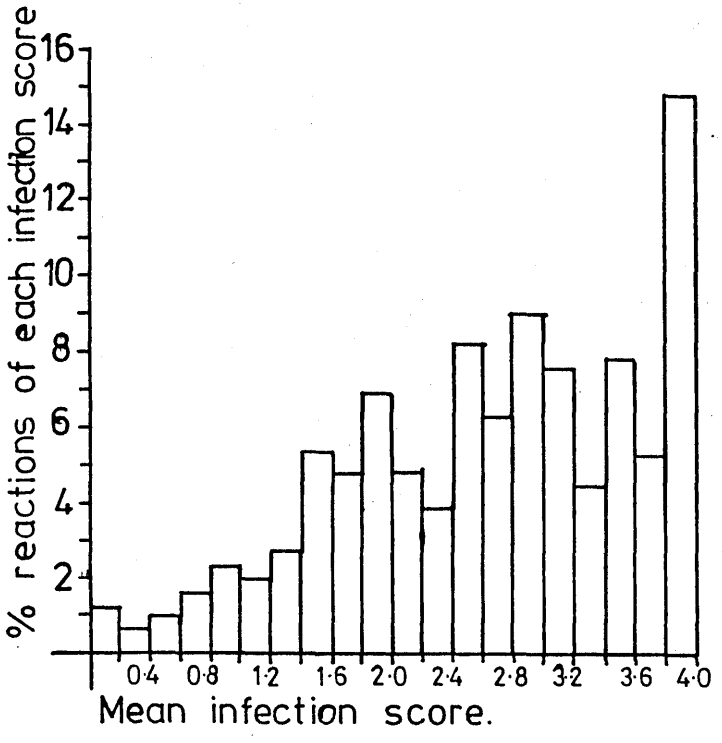


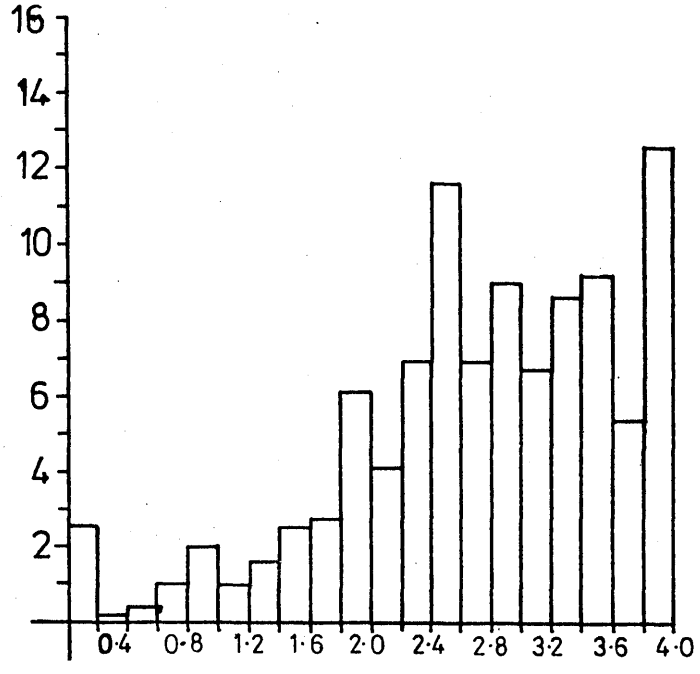
Fig 7.11

% of reactions of each mean infection score for the Glasgow plants with each set of isolates.

N.V.R.S Isolates on Glasgow plants.



Glasgow isolates on Glasgow plants.



N.V.R.S. population. Infection types below 3 would be harder to detect with the naked eye in the field so that these types of infected plants may be overlooked. Alternatively, the results may indicate that there are more plants at N.V.R.S. with partial resistance, or a relatively low level of susceptibility which may reduce the rate of progress of the mildew epidemic and so reduce the level of mildew incidence.

An examination of the different phenotypes detected in the groundsel populations tested with all 10 isolates revealed that 11 resistance phenotypes (excluding the phenotype which is susceptible to all the isolates) occurred in the 98 plants from Glasgow. The population at N.V.R.S was found to have 23 different resistance phenotypes. Seven of the phenotypes were common to both populations indicating that there are similarities between the two populations as well as dissimilarities. It is possible that the patterns of resistance phenotypes are basically similar in both populations with only but occasional differences, such as the high frequency of plants with resistance to isolate G9 in the 17 m x 17 m plot at N.V.R.S.

7.4.3. The distribution of resistance within a population

There were indications that as the size of area sampled increased then so did the number of different phenotypes detected in the groundsel population. At Glasgow only one resistance phenotype was detected in each of three individual 2 m x 4 m plots (Fig 7.2). Of the 100 plants tested from the 4 plots (Area GL1) only 3 resistance phenotypes were detected. Within the garden at Garscube (Area GL2, 100 m x 75 m) 52 plants were assessed and 3 phenotypes were detected using both the N.V.R.S. and Glasgow isolates (Fig 7.3). When the area of sampling was extended to 1.2 km^2 (Area GL3, Fig 7.4), 8 phenotypes were detected in the 46 plants assessed. At N.V.R.S. a similar pattern was observed with the sampling of area N3 (6 km^2) in 1984 revealing 16 different phenotypes (Fig 7.9). An increase in the number of phenotypes such as this would be expected where the distributions of various phenotypes are patchy.

Except for susceptibility to every isolate, the most common resistance phenotype in the N.V.R.S. population exhibited resistance to isolate G9 only. This phenotype was found predominantly within the 17 m x 17 m plot (Area N2) and the 1 m^2 plot (Area N1, Fig 7.1). Two other phenotypes were detected in area N2 in 1983 (Fig 7.6) and 5 others in 1984 (Fig 7.7). Eight additional resistance phenotypes were detected in the 1 m^2 area (N1). This illustrates the high degree of heterogeneity in the groundsel population, even within an area as small as 1 m^2. The groundsel population possesses a mixture of resistance phenotypes, some of which have resistance to several isolates.

7.4.4. The importance of race specific resistance in the pathosystem.

Robinson (1982a) postulated that in a highly evolved pathosystem, where

a gene-for-gene relationship applies, even where the number of genes involved is great, eventually each virulence phenotype in the pathogen will successfully establish itself on the matching resistance phenotype of the host. When each new resistance phenotype first appears it will have a selective advantage but after a period the resistance is likely to be overcome by the pathogen so that the phenotype no longer confers a selective advantage in hosts having that phenotype. Eventually the advantage of any particular resistance phenotype would tend to diminish. This appears to be the case in groundsel; the majority of the groundsel population is susceptible to the majority of the mildew population. Particular resistance phenotypes were generally detected only once or twice in the population. This does not necessarily mean that race specific resistance is not important in this pathosystem. The complexity of the system would tend to reduce the incidence of disease at the host population level.

A complex system could also be advantageous to the pathogen. If a highly pathogenic form of the pathogen were to develop, then it is likely that some hosts would still survive, decreasing the likelihood of the virulent form destroying the whole host population and hence the pathogens' food source. Increasing numbers of resistance genes in the host population may also select for complex virulence phenotypes capable of colonizing many different host phenotypes. However, there was no evidence that such selection leads to the development of a 'super race', ie a race possessing virulence genes matching all the resistance genes in the host. A 'super race' may occur in the mildew population but it does not appear to have a selective advantage or one would expect it to have become common and be detected even within the small samples such as those examined in this study. A 'super race' might only have a selective advantage if there was a high proportion

of plants with all possible genes for resistance. There was no evidence from this study for the existence of such plants at a high frequency in the groundsel population, although plants were found which had an apparently complex resistance phenotype.

Partial resistance and tolerance are probably also of considerable importance in the defence strategy. Groundsel is considered to be a primary colonizer. Once an individual has become established in an area it is to its advantage that it should produce a large number of progeny of a similar phenotype as rapidly as possible to ensure a rapid colonization of that site before competitors also colonize the area. Groundsel is primarily an inbreeder and produces on average 46 fruits per capitulum and 25 capitula per plant (ie about 1150 seed per plant) (Salisbury, 1943). The species is ,therefore, well adapted to primary colonization. Likewise a pathogen, such as E. fischeri may be considered to be a primary colonizer. Once it has become established on a host individual it would be advantageous to produce large numbers of similar progeny to ensure colonization of the host. It is also an advantage to the parasite in the long term to cause relatively little damage to the host so that the host may reproduce. The progeny of an individual plant will be of very similar phenotype to the parent and will tend to develop in close proximity to the parent, providing suitable hosts for the pathogen phenotypes colonizing the parent. It is possible that a balance has been achieved where both partial resistance and tolerance ensure that while conidia are produced to allow colonization of host progenies the damage caused by the pathogen is not so severe as to threaten the host to the detriment of both partners. The characteristics of partial resistance and tolerance may therefore provide advantages over high susceptibility for the pathogen as well as the host, since a host supply would be ensured. Even the most susceptible lines of groundsel

PLATE 5. Healthy(left) and heavily mildew infected (right) plants at maturity.

survive a mildew attack (Plate 5.) and reproduce so it is likely that even these 'highly susceptible' plants exhibit some partial resistance, or tolerance. Selection for tolerance and partial resistance would probably operate in a similar way, both allowing host progeny to develop in order to be exploited by the progeny of the pathogen.

CHAPTER 8. A STUDY OF THE FREQUENCY OF MILDEW, RUST AND APHID INFECTIONS IN A NATURAL POPULATION OF GROUNDSEL.

CONTENTS.

8.0 A STUDY OF THE FREQUENCY OF MILDEW, RUST AND APHID INFECTIONS IN A NATURAL POPULATION OF GROUNDSEL.

8.1. Introduction.

Disease epidemics in natural populations have often been reported to cause relatively little damage to their host populations (Burdon, 1978 , Segal et al, 1980 , Browning, 1974). However, studies by Kranz (1968a,1968b) demonstrated that this was not always the case. The extent of the damage caused by a pathogen depended on the particular host and parasite concerned. Some epidemics do lead to the devastation of the host. There are several well-known cases where pathogens have caused the destruction of natural host populations. For example, the non-indigenous pathogen Ceratocystis ulmi (Dutch Elm disease) devastated Elm species in North America and Europe (Holmes, 1962). At the other extreme, some parasites cause virtually no damage to their wild hosts. Verticillium albo-atrum is ubiquito- in groundsel yet produces virtually no symptoms (Matta and Kerling, 1964). Occasionally, a pathogen is considered to be of some benefit to the host. The systemic pathogen Epichloe typhina sterilizes its grass hosts by preventing the emergence of the inflorescence. The infected grasses tend to be more persistent than uninfected plants in pastures, supposedly having a greater vegetative vigour. It is postulated that this is because resources in the host are diverted away from flowering releasing a greater proportion for tiller production (Harper, 1977).

Where epidemics are relatively less severe in natural populations this has often been attributed to the greater diversity of natural plant communities (Burdon, 1978 , Burdon and Marshall, 1981). Not

only is there diversity of species in natural communities but, there may be tremendous diversity of genotypes within a particular species (Harlan, 1976).

The devastation caused by pathogens in crops has been attributed to the relative homogeneity of the host population (Vanderplank, 1963). The widespread culture of a single variety of, for example, a cereal tends to produce conditions favourable to an epidemic. Marshall (1977) also warned of the dangers of producing highly homogeneous crops from a relatively narrow gene base. This has led to the idea of using cereal variety mixtures and multilines to control epidemics of pathogens such as the powdery mildews. The incidence of powdery mildew was found to be reduced in both barley varietal mixtures (White, 1982., Wolfe et al 1981, Chin and Wolfe, 1984a,b) and wheat multilines (Fried et al 1979). The way the ultimate incidence of powdery mildew is thought to be reduced was different for the two pathosystems. In the barley mixture, where all components possessed resistance, there appeared to be little benefit at the beginning of the epidemic when the majority of the powdery mildew was from sources outside the crop. The most benefit was found in the mixture when the levels of inoculum from within the crop had reached a significant level but the crop canopy was not closed. During this time it is thought that the epidemic is curbed because a large proportion of the spores arrive on host genotypes they cannot infect (Wolfe and Minchin, 1979., Wolfe et al 1981). In the wheat multilines, where some proportion of the multiline was susceptible to all mildew races, the powdery mildew epidemic was checked by a reduction in the initial amount of inoculum present in the crop. The rate of the mildew epidemic was not decreased significantly until the resistant component of the multiline reached 75% (Fried et al, 1979).

Host density is also thought to be of importance in regulating

epidemic development. In many instances increased host density appears to increase the severity and rate of progress of an epidemic (Burdon, 1982b). Infection rates in celery by Cercospora apii (Berger, 1975) and infection of Lepidium sativum by Pythium irregulare (Burdon and Chilvers, 1975b) are increased by host density. One would expect host density to be of importance where propagules of the pathogen are only dispersed over short distances as with soil fungi. However, there is also evidence that increasing densities of barley in controlled environment experiments increase the rate of powdery mildew epidemics (Burdon and Chilvers, 1976a). Natural communities are usually made up of several different species of hosts and non-hosts, so effectively the density of hosts is reduced. It is possible that the effective reduction in host density may be partly responsible for the reduction in epidemic severity in natural populations.

Probably the most important determinant of the rate of progress of an epidemic is the climatic conditions. Unless the environmental conditions are favourable for the growth and reproduction of the pathogen on the host there will be no epidemic. In the 1800's before the germ theory of disease had been discovered, many diseases caused by pathogens were thought to be a direct consequence of environmental conditions (Walker, 1969) because they were so strongly associated with those conditions. The effects of climate on the progress of many disease epidemics have been studied in detail and the information gained has made it possible to produce accurate disease forecast systems. Temperature, humidity and rainfall are often the most important climatic factors determining an epidemic.

Little is known about the incidence of Erysiphe fischeri on Senecio vulgaris in a natural population or the environmental conditions that affect it. Ben-Kalio (1976) found that the optimum

temperature for conidial germination on glass slides was between 18°C and 28°C. Optimum temperatures for germ tube elongation lie between 12°C and 18°C. The development of the fungus on the host is inhibited below 6°C and above 28°C. Conidial germination was relatively unaffected by humidity; germination was found to occur at relative humidities between 0 and 100% with an optimum at 90%. Thus, it appears that potentially, epidemics may occur within a wide range of climatic conditions.

Conditions suitable for other powdery mildews are also diverse. Cereal powdery mildews tend to be severe in cool, moist temperate climates in contrast to very arid hot climates. Last (1963) observed that powdery mildew (Erysiphe graminis) on wheat and barley was rarely severe in spring. Later in summer as the temperatures rose to more favourable levels, disease incidence and severity were increasingly influenced by host nutrition and growth rate. Tapke (1953) found that cereals grown in the glasshouse during short days, with low light intensities and relatively cool temperatures, were more severly infected by powdery mildews than plants grown in longer, warmer and brighter days. The optimum temperature for wheat powdery mildew development was reported as 20°C by Futrell and Dickson (1954). However, plants appeared to be more susceptible at 14°C than 20°C and conidial production was also reported to be more profuse at 14°C than at 20°C by Last (1954).

Erysiphe cichoracearum on Trifolium spp (clover), has a higher optimum temperature for development of 24°C than most other mildews (Stavely and Hansen, 1966a). Thus, one may expect an epidemic on clover to occur at relatively high temperatures.

Other powdery mildews, such as Erysiphe cichoracearum on cucurbits, are favoured by dry atmospheric and soil conditions. The conidia are capable of germinating in relative humidities of below

20%, though as with cereal powdery mildews, moderate temperatures, reduced light intensities and fertile soils encourage the growth of E. cichoracearum (Yarwood, 1957).

Cherewick (1944) observed that the development of several species of Erysiphe was impeded if the colonies were sprinkled with water. Yarwood (1939) postulated that rainfall and water sprays inhibited mildew by damaging the conidiophores.

An investigation of the incidence of Erysiphe fischeri on four plots was carried out at Glasgow over the summer of 1983. It was hoped that some information would be gained on the natural incidence of mildew on groundsel. The time of year the epidemic progressed most rapidly and how this related to climatic factors such as rainfall and temperature, and factors such as host density were studied. It was also hoped that if a plot was subsequently found to have a low level of powdery mildew incidence then this may be related to higher levels of race specific resistance (See Chapter 7). However, it was subsequently demonstrated that the number of plants shown to have complete race specific resistance was too low on all plots to make valid comparisons. The incidence of rust (Puccinia lagenophora) and aphids (Myzus persicae) were also recorded on each sampling date as they also occurred commonly.

8.2. Materials and methods.

Populations of groundsel were studied in four plots, each of 2m by 4m, within the garden at Garscube, Glasgow (Area GL1). These were the same plots sampled for the assessment of resistance to powdery mildew.

Plots 1, 2, 3 and 4 were ploughed on 24.4.83 and then lightly raked on 10.5.83. Each of the plots was divided up into 32, 50 cm^2 quadrats, using canes and twine. The plots were examined 2, 4, 6, 8 and 12 weeks after the plots were raked. On each sampling date 15 quadrats on each plot were chosen using random numbers. For each quadrat the number of groundsel plants, their stage of development and whether or not they were colonized by mildew, rust or aphids was recorded.

For the purposes of the analysis the development of plants was classified into 7 stages as follows:

Stage 1. Plants with one or two true leaves.

Stage 2. Plants with three or four true leaves.

Stage 3. Plants with five or more true leaves but no visible sign of flowers in bud.

Stage 4. Plants producing flower buds.

Stage 5. Plants in flower.

Stage 6. Plants seeding.

Stage 7. Plants in later stages of seeding and in advanced stages of senescence.

Meteorological data from Glasgow (Abbotsinch) Airport, obtained from the Meteorological office, Edinburgh are given in Appendix Table 8.1 and were used as an indication of the weather conditions during

the investigation.

8.3. Results.

8.3.1. The development of the groundsel population.

The mean frequencies of groundsel plants on each plot for each recording date are given in Table 8.1 and Fig 8.1. Plots 1 and 2 produced fewer plants than plots 3 and 4. The frequency of groundsel on all plots tended to increase to a maximum 6 to 8 weeks after the plots were raked and then began to decline in August.

The distribution of groundsel on plot 3 was uneven, the frequency of plants detected at week six was lower than expected because the majority of the quadrats fell in thinly populated areas of the plot. On the other occasions the quadrats effectively represented all areas on the plot.

8.3.2. The frequency of groundsel infected with mildew.

The mean percentage frequencies of plants colonized with mildew on each plot at each recording date are given in Table 8.2 and Fig 8.2. The levels of mildew detected in the plots were very low until after the eighth week when there was a rapid increase in all plots. The rates of mildew increase from week 8 to week 12 were 6.5% (Plot 1), 14.5% (Plot 2), 9.1% (Plot 3) and 23% (Plot 4) per week. Plot 4 with the greatest density of groundsel possessed a higher proportion of plants infected with mildew than plot 1 with the smallest population of groundsel. Plot 3 showed a relatively low level of mildew in relation to the number of groundsel plants on the plot. This discrepancy may have resulted from the uneven distribution of the host.

The mean percentage of plants infected with mildew at each stage

Table 8.1 Mean frequency of groundsel per plot

Weeks after Exp start	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
2	0.7	0.23	0.1	0.07	6.6	0.93	7.8	0.93
4	2.5	0.62	4.9	0.64	16.1	2.64	16.7	1.70
6	2.7	0.43	6.5	0.58	11.1	1.36	19.5	2.49
8	2.2	0.38	7.7	0.48	16.5	2.32	18.3	1.96
12	1.7	0.36	6.4	0.47	9.4	1.19	12.7	1.05

Table 8.2 Mean % plants with mildew infection

Weeks after Exp start	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
2	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
4	0.0	0.00	0.0	0.00	0.9	0.94	0.0	0.00
6	0.0	0.00	0.0	0.00	0.0	0.00	1.8	1.27
8	2.2	2.17	0.0	0.00	0.4	0.44	1.5	0.65
12	27.6	10.28	58.3	4.86	36.6	5.00	93.6	2.08

Table 8.3 Mean % plants at each stage of development with mildew infection

Stage of development	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
1	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
2	0.0	0.00	0.0	0.00	0.0	0.00	2.9	2.18
3	0.0	0.00	0.0	0.00	0.0	0.00	4.5	2.59
4	6.3	5.33	0.0	0.00	1.4	1.35	4.4	3.11
5	22.2	14.70	0.0	0.00	1.7	1.72	16.3	5.53
6	28.8	13.54	59.8	6.16	21.7	5.53	52.5	8.88
7	0.0	0.00	56.7	7.82	33.7	7.67	89.3	5.80

Fig 8.1

Mean frequency of groundsel per quadrant on each plot against time.

● = Plot 1

■ = Plot 2

o = Plot 3

□ = Plot 4

| = Standard error

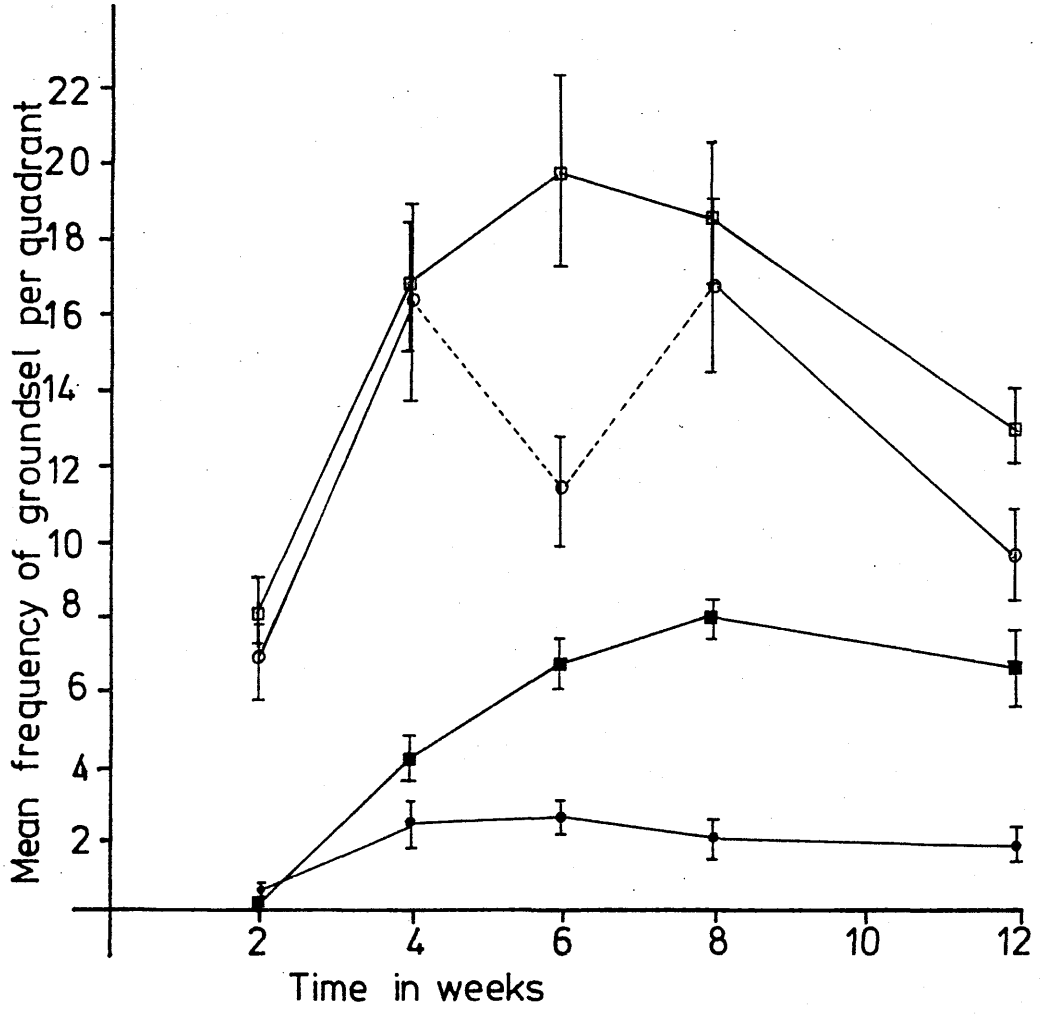


Fig 8·2

Mean % frequency of groundsel plants with mildew.

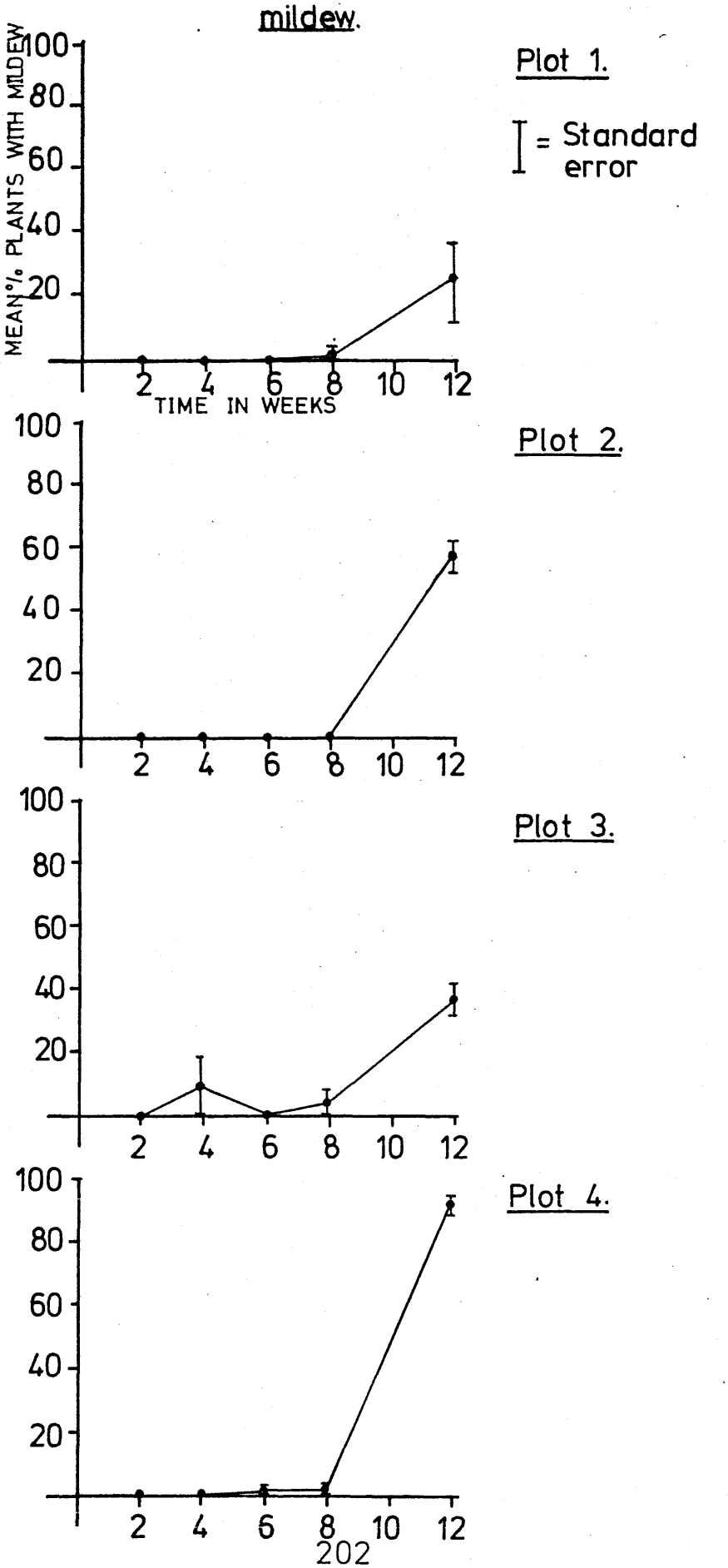
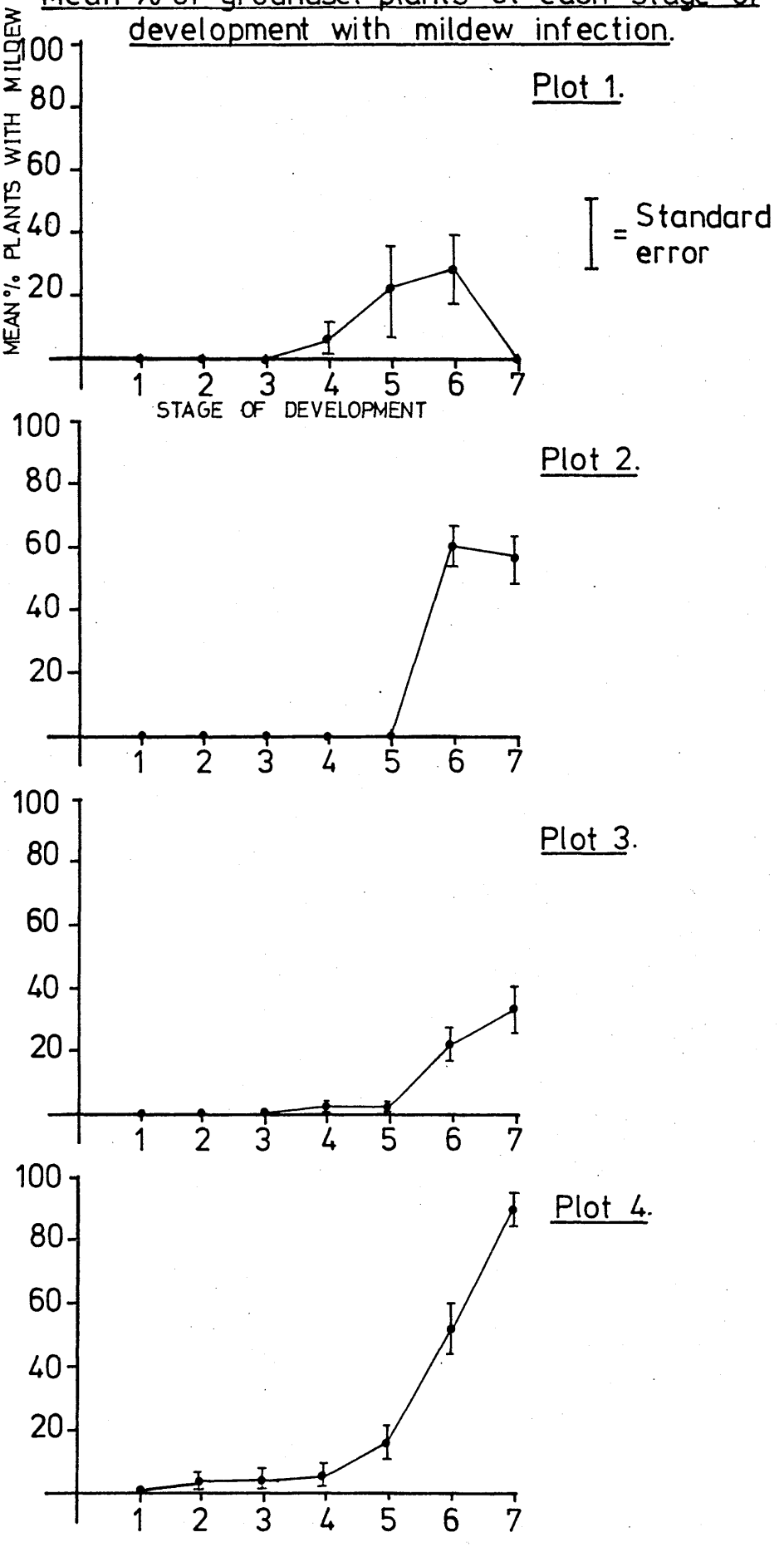


Fig 8.3

Mean % of groundsel plants at each stage of development with mildew infection.



of development is given in Table 8.3. The results are also presented graphically in Fig 8.3. Plants at stage 4 or earlier appear to be less susceptible than the later stages, when plants are in flower. However, the development of plants tended to be synchronised so that at each observation the majority of the plants were at a similar stage of development. The apparent resistance of the younger growth stages may have been due to a lack of inoculum early in the year.

8.3.3. The frequency of groundsel with rust infections.

Table 8.4 and Fig 8.4 gives the mean percentage of plants infected with rust (Puccinia lagenophora) on each plot. The first incidence of rust was recorded before that of the mildew but it was not until after week 8 that there was a substantial increase in the incidence of rust infection. There was a rapid increase of rust between week 8 and 12. The rates of rust increase during this period on plots 1, 2, 3 and 4 were 1.3%, 25%, 21.5% and 19.5% per week respectively. A suprisingly low number of plants on plot 1 became infected with rust, but this may have been related to the low frequency of groundsel on the plot, slowing the rate of development of the rust epidemic.

Table 8.5 and Fig 8.5 give the mean percentage of plants infected with rust at each stage of development on each plot. The plants appeared to be more susceptible to rust at or after flowering.

8.3.4. The incidence of plants infected with both rust and mildew.

The total number of plants recorded over the whole of the experimental period that were found to be colonized by mildew or rust or both are shown in Table 8.6.

Table 8.6. The numbers of plants with mildew and rust.

Plants with mildew only	Plants with rust only	Plants with both	Plants without infection	Total plants assessed
38	174	259	2084	2555

Table 8.4 Mean % plants with rust infection

Weeks after Exp start	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
2	0	0	0	0	0	0	0	0
4	0	0	0	0	0	0	0	0
6	0	0	1.1	1.08	6.7	3.27	0	0
8	0	0	0	0	6.7	3.34	11.3	3.22
12	5.2	3.64	100	0	91.6	3.33	39.4	4.10

Table 8.5 Mean % plants at each stage of development infected with rust

Stage of Development	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
1	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
2	0.0	0.00	0.0	0.00	0.0	0.00	2.0	1.96
3	0.0	0.00	1.9	1.85	3.2	2.24	4.1	2.43
4	0.0	0.00	0.0	0.00	0.9	0.90	0.5	0.39
5	0.0	0.00	0.0	0.00	10.7	4.97	18.1	5.47
6	10.4	7.00	100.0	0.00	59.7	10.90	76.2	7.99
7	0.0	0.00	100.0	0.00	91.5	18.17	83.2	5.82

Table 8.7 Mean % groundsel with aphids

Weeks after Exp start	Plot 1 Mean	Plot 1 Se	Plot 2 Mean	Plot 2 Se	Plot 3 Mean	Plot 3 Se	Plot 4 Mean	Plot 4 Se
2	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
4	8.3	6.06	1.8	12.4	13.7	4.19	25.3	4.60
6	0.00	0.00	1.5	1.16	12.4	2.93	32.4	4.56
8	9.4	6.80	11.7	3.96	73.2	6.13	28.9	4.35
12	63.0	11.31	29.7	6.35	17.1	4.93	22.8	4.75

Fig 8.4

Mean % of plants with rust infection.

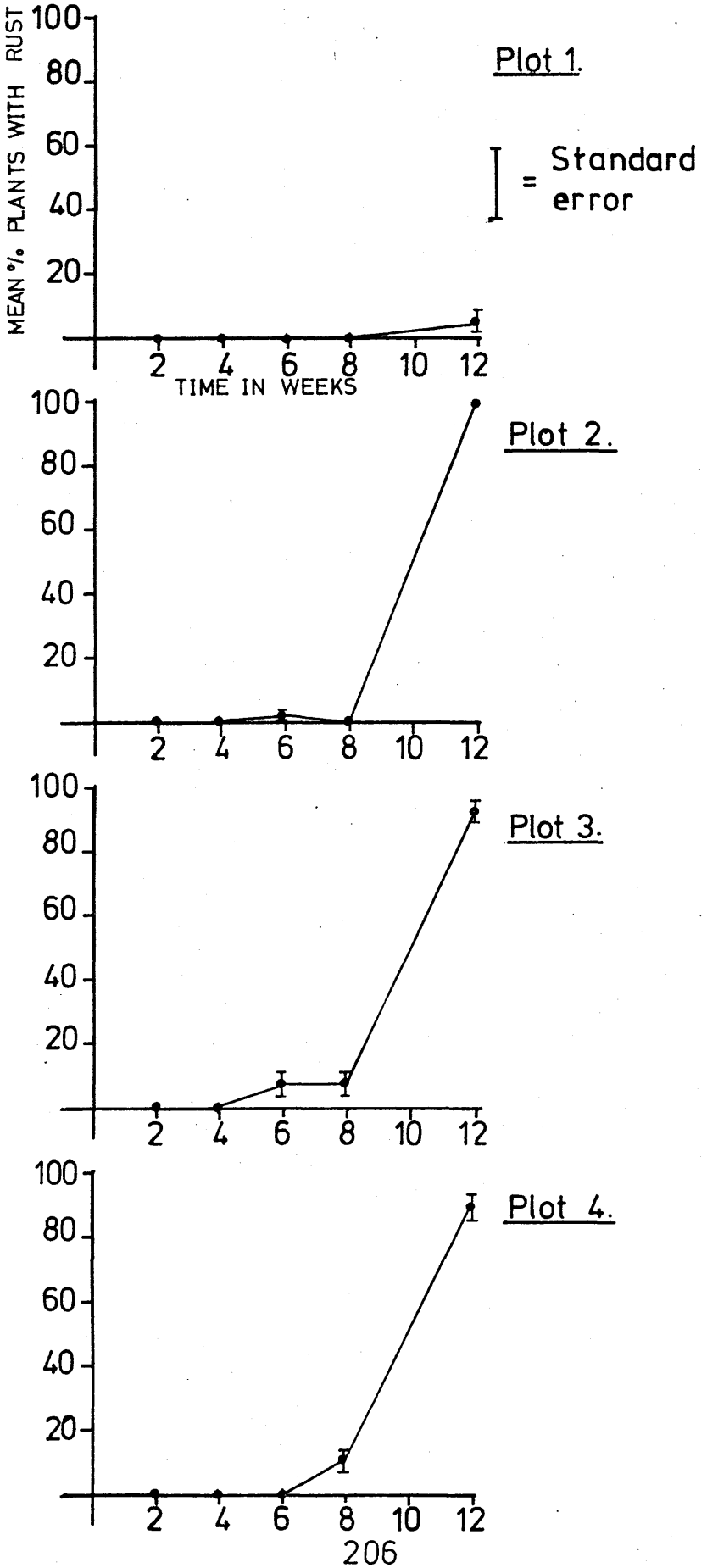


Fig 8.5

Mean % Plants with rust infection at each stage of development.

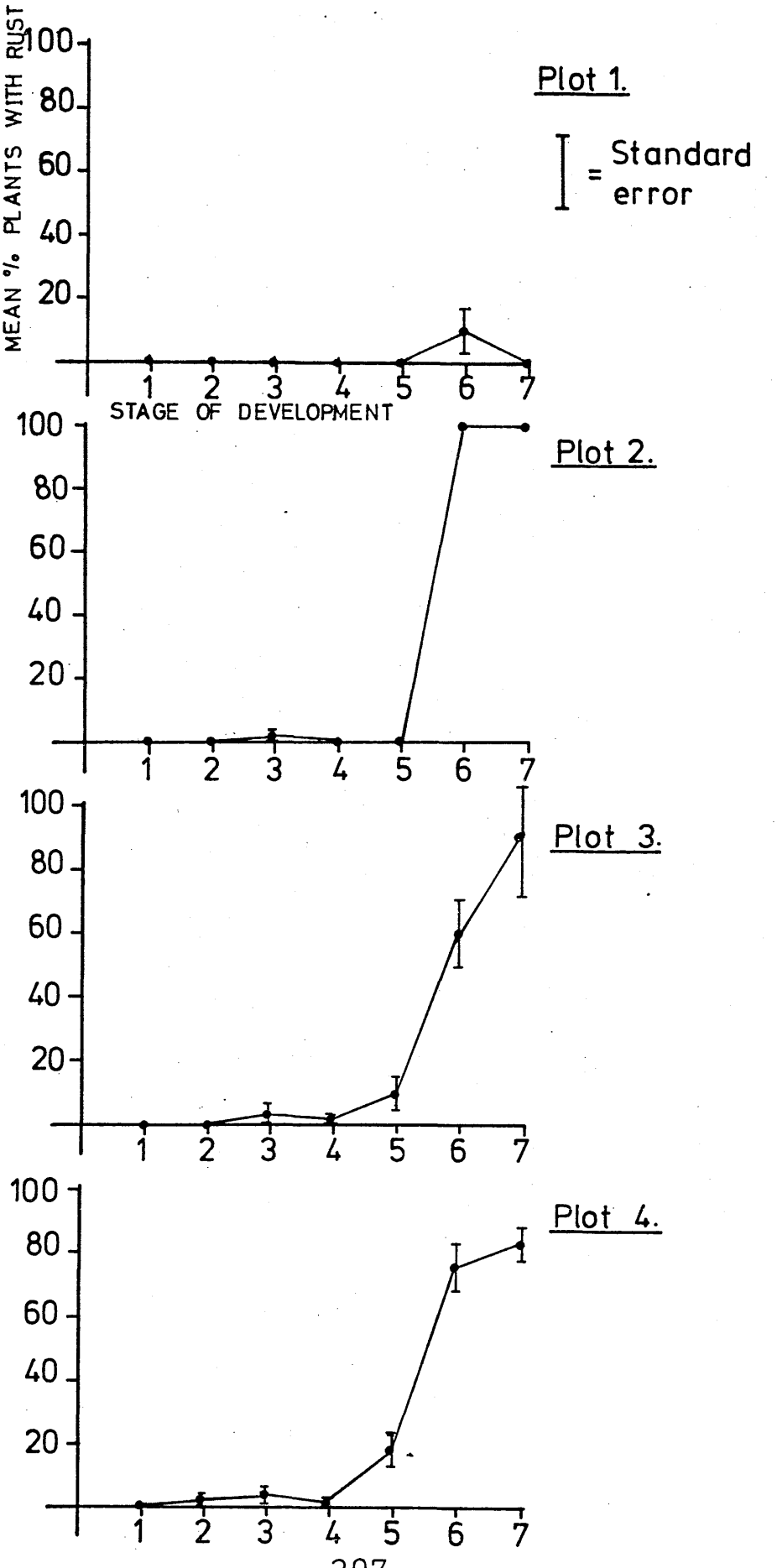


Fig 8.6

Mean % groundsel plants with aphid infection.

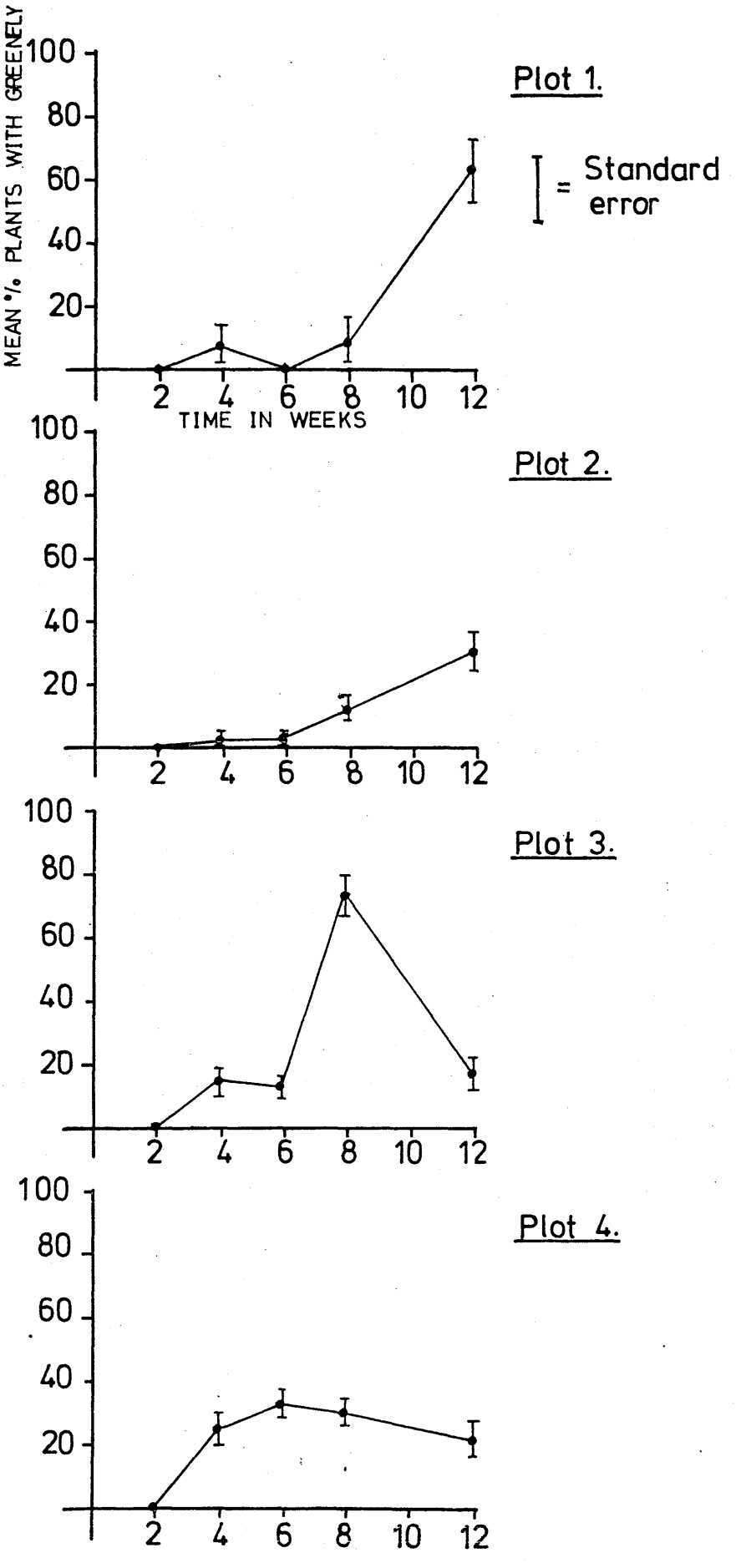
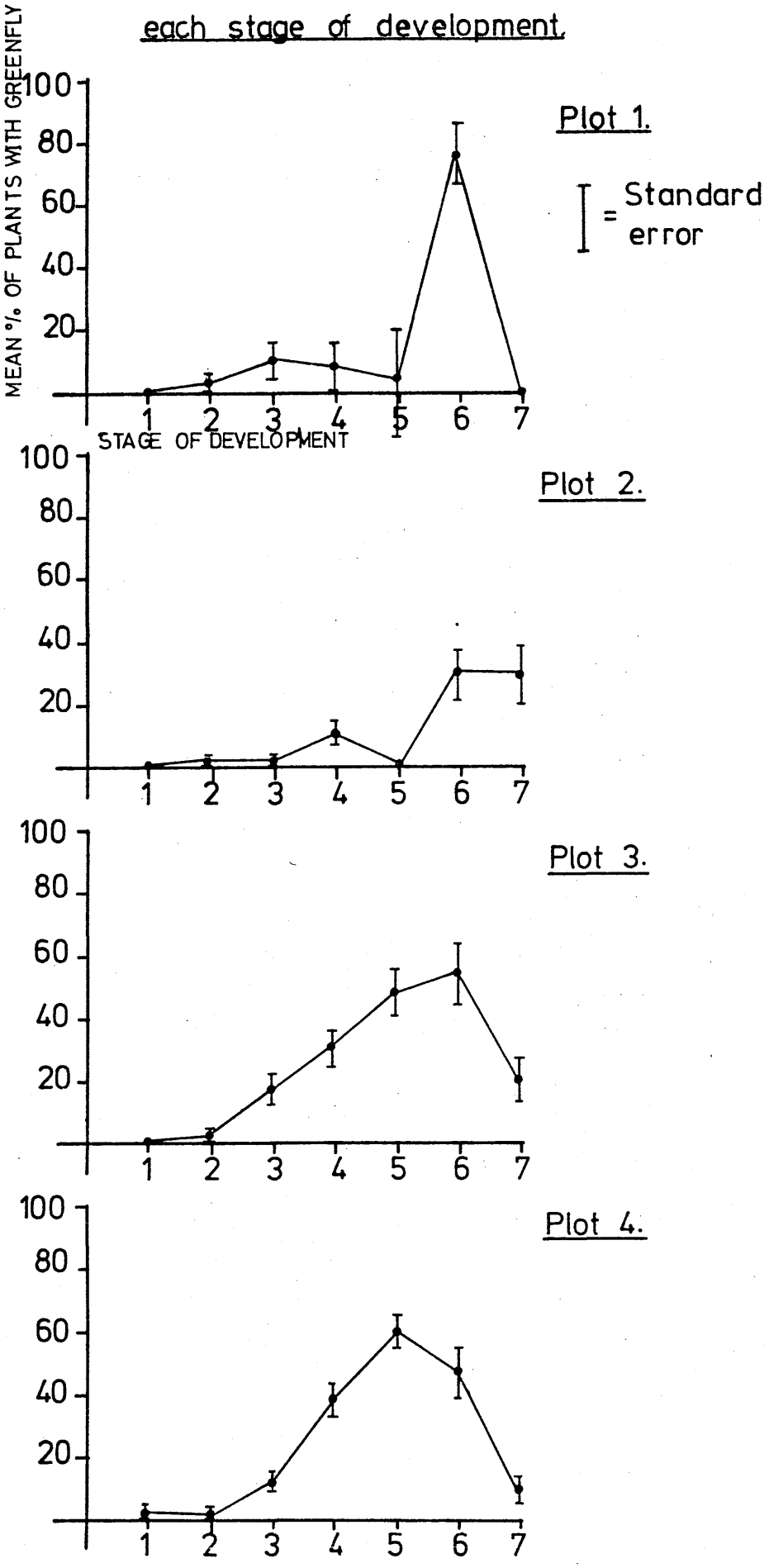


Fig 8.7

Mean % frequency of plants with aphids at each stage of development.



There was a highly significant ($P < 0.001$) association between rust and mildew infection ($\chi^2 = 1,179.0$).

8.3.5. The frequency of groundsel colonized by greenfly.

Table 8.7 and Fig 8.6 give the mean percentage of groundsel plants colonized with aphids on each plot on each recording date. The occurrence of aphids tended to be variable, which was to be expected since they are capable of being mobile and of showing host preference.

Aphids appeared earlier than either the mildew or rust and their incidence showed signs of levelling off in August towards the end of the investigation period. Table 8.8 and Fig 8.7 give the mean percentage of plants colonized by aphids at each stage of development. Generally, the incidence of aphids increased as plants developed, however, by the time the plants had reached stage 7 and were dying, the incidence of colonization had declined.

8.3.6. The effects of climatic conditions on the incidence of mildew, rust and greenfly.

The mean rainfall, mean maximum temperature and mean minimum temperature per day for the period before and including each recording date were calculated from the meteorological data and given in Table 8.9. The data is also presented graphically in Figs 8.8 and 8.9. Daily rainfall tended to decrease over the sampling period, whilst, mean maximum and minimum temperatures of the preceeding 14 days tended to increase.

Table 8.8 Mean % frequency at each stage of development with aphids

Stage of Development	Plot 1		Plot 2		Plot 3		Plot 4	
	Mean	Se	Mean	Se	Mean	Se	Mean	Se
1	0.0	0.00	0.0	0.00	0.0	0.00	2.0	1.45
2	2.5	2.50	2.3	1.57	2.7	2.63	1.6	1.05
3	9.5	6.56	1.9	1.85	17.8	5.74	11.8	2.57
4	7.8	7.76	11.4	3.71	30.4	6.06	38.8	5.45
5	4.4	17.57	0.0	0.00	48.5	7.73	59.8	5.20
6	76.0	11.94	29.2	8.37	54.7	10.62	48.0	7.77
7	0.0	0.00	30.1	9.94	19.3	7.52	8.5	3.89

Table 8.9 The mean rainfall, maximum temperature and minimum temperature per day, for the period before and including each sampling date

Weeks after Exp start	Mean rainfall/ day		Mean Max Temp/day		Mean Min Temp/day	
	Mean	Se	Mean	Se	Mean	Se
2	2.36	0.847	13.4	0.46	6.0	0.55
4	2.50	1.427	14.0	0.90	6.7	0.64
6	1.38	0.603	17.4	0.89	9.0	0.58
8	1.07	0.709	17.3	0.72	9.9	0.49
12	0.46	0.194	22.2	0.66	11.7	0.53

Fig 8·8

Mean rainfall over observation period.

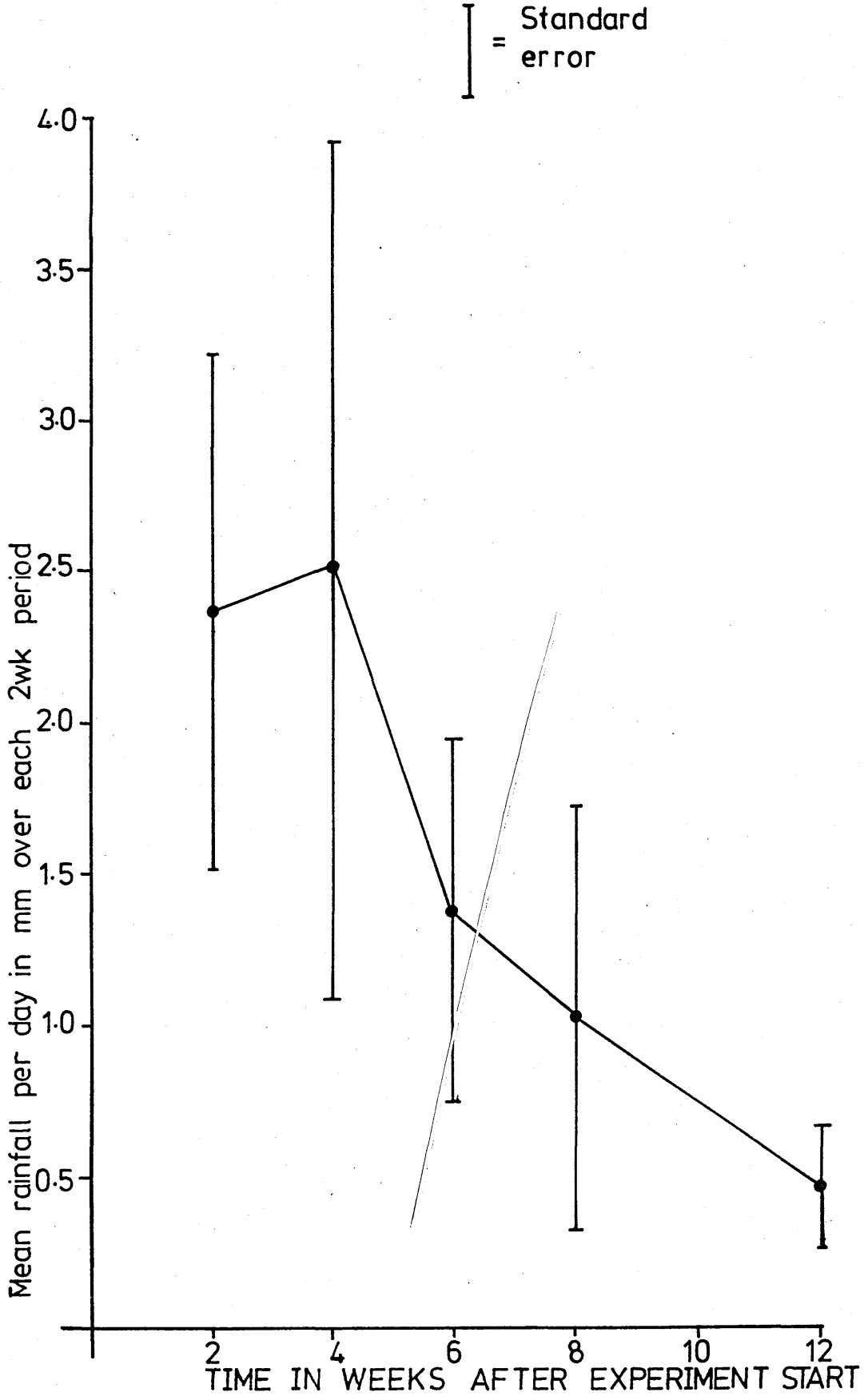
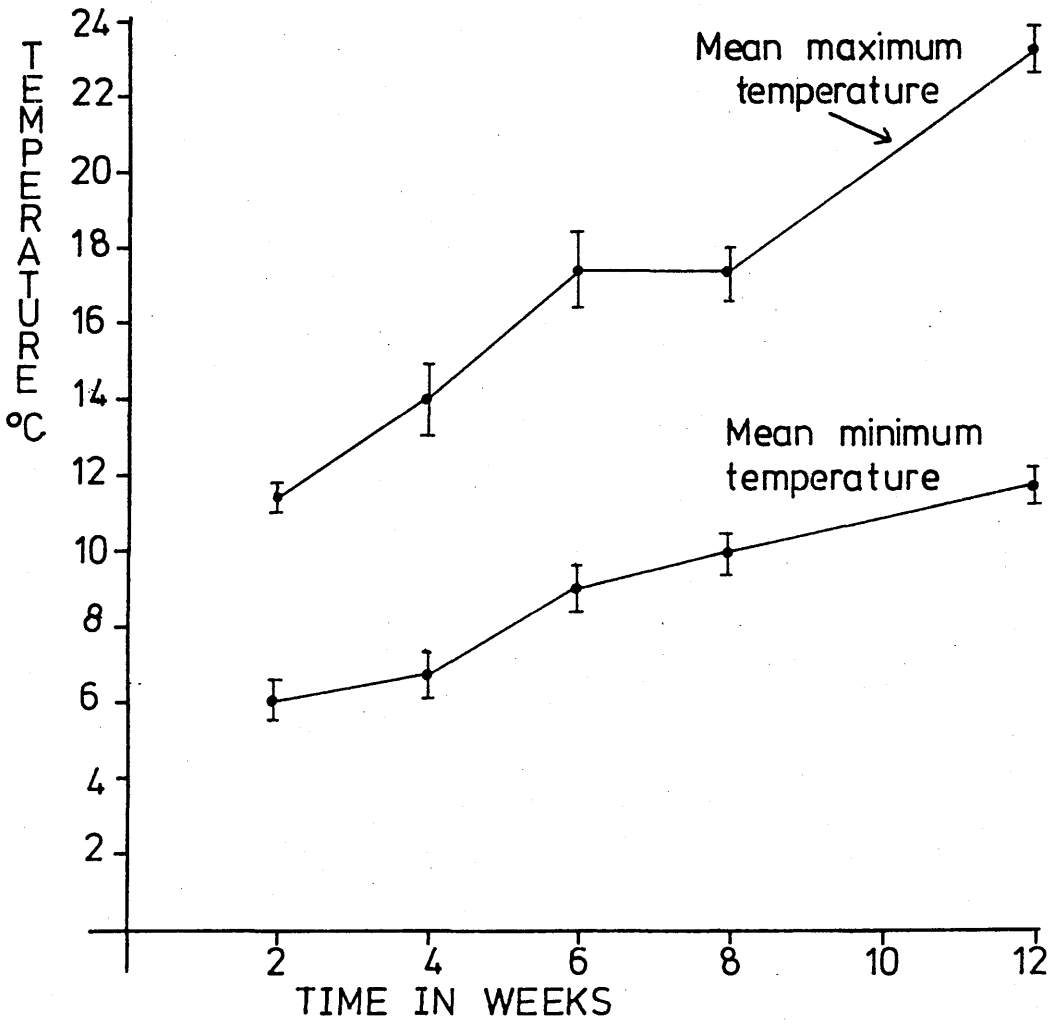


Fig 8.9.

Maximum and minimum temperature during each two week period.



8.4. Discussion.

The incidence of Erysiphe fischeri did not become substantial until relatively late in the season, in July and August. A combination of three possible factors may have delayed the onset of the epidemic:

1. The conidial inoculum available early in the season was probably very low.
2. Plants at an early stage of development may be less susceptible than older plants (See Chapter 3).
3. High rainfall and lower temperatures in early summer could have discouraged the development of an epidemic.

The sexual stage of Erysiphe fischeri has not been identified in Great Britain so it is assumed that the pathogen overwinters as mycelium or conidia on groundsel plants. Colonies of mildew on leaf discs on benzimidazole agar were found to remain capable of producing viable conidia for up to 6 wks in an illuminated cold room at 4°C. The length of time the mildew colony remained viable depended on how long the leaf segments survived. After a period of time the benzimidazole was no longer effective in preventing leaf senescence and the leaves eventually succumbed to bacterial rots. Colonies on leaf discs were also viable after they had been in the freezing compartment of a fridge at approximately 0°C for a week. Deep freezing at approximately -28°C destroyed the colonies and leaf discs. It appears that Erysiphe fischeri is capable of overwintering in Britain provided that the host also survives. Host and pathogen can survive at 0°C and it is likely they can withstand lower temperatures although not -28°C.

Relatively few groundsel plants tend to overwinter. The majority of

such plants are quite young, and are probably autumn-sown progeny of the main 'flush' of groundsel of the summer or seeds from the seed bank brought to the soil surface by autumn cultivations. At the beginning of spring there are relatively few groundsel plants available and these tend to be immature plants that have overwintered. Therefore, not only is there a tendency for initial mildew inoculum to be scarce but host tissue is also scarce and probably at a relatively resistant stage of development. Time is required for sufficient mildew inoculum to build up and initiate epidemics.

Rainfall was frequent early in the season; there were only 8 days in the first 6 weeks of the trial when it did not rain. During the second six weeks, when mildew developed, there were 16 days without rain. Maximum and minimum temperatures were favourable to mildew at all times although they were relatively low at the beginning of the season. Rainfall was probably the most important environmental factor affecting epidemic development. It is noteworthy that rain made examination for mildew difficult since mildew would tend to be washed from the plants. Observations made on plants soon after rain may have underestimated the amount of mildew present. It appears that E. fischeri responds in a similar way to climatic factors as E. cichoracearum (Yarwood, 1957 , Cherewick, 1944) which is discouraged by rain.

There was some evidence indicating that greater densities of groundsel were more favourable to more rapid mildew epidemic development. Burdon (1982b) has also found evidence of this in other pathosystems. However, the results produced in the present study are based on field observations where inoculum levels were not controlled. Although the plots were within 100 m of each other it is highly likely that they did not receive the same amount of mildew inoculum. The frequency of other plant species on the plots were not recorded:

numbers of non-host species in the population may affect the spread of the epidemic.

The numbers of plants infected with both mildew and rust were far more frequent than expected. This was suprising since rust was expected to be more frequent in wet conditions in contrast to mildew.

The rust epidemic started slightly earlier than the mildew epidemic. This was partly to be expected as rust spores require free water to germinate. The incidence of rust was greater on mature groundsel but other casual observations indicated that seedlings did become infected with rust more frequently than they did with mildew. The incidence of rust tended to be greater than the incidence of mildew. This may have been a reflection of an earlier onset of the epidemic, but it may also indicate that the groundsel populations were more susceptible to rust. Rust may in fact impose stronger selection for resistance in the host than mildew, since it tends to produce more damaging infections and can be lethal to the seedling host. Paul and Ayres (1984) studied the effects of Puccinia lagenophora and water stress on photosynthesis and the water relations of groundsel. Infection with rust increased the detrimental effects of water stress on photosynthesis and water use efficiency in the host. Therefore, under drier summer conditions rust is more likely to be detrimental to the host population.

The incidence of Myzus persicae in the plots was more variable than the incidence of rust or mildew. Aphids appeared before rust or mildew. There was a tendency for aphid infestations to decline when plants became senescent. The incidence of aphids has been reported to behave in a similar way on other host species (Van Emden et al,1969). Aphids are capable of showing a preference for different host tissues and Jepson (1982) found that apterous Myzus persicae on sugar beet made repeated leaf to leaf movements. These movements were related to

particular stages of development of the host. Aphids tended to settle on the youngest plants when essential amino acids and sugars were at the highest concentrations or on the plants at a later stage of growth when maximum rates of photosynthesis were occurring. On lettuce, Eenink and Dieleman (1980) found that younger plants were more resistant to Myzus persicae than older plants. The information from the plots of groundsel suggested a similar pattern. The sample dates on which the highest incidence of aphids occurred coincided with plant maturity.

Aphids had been a problem in the glasshouses at Glasgow and occasionally in the growth rooms. Observations indicated that groundsel was most likely to be killed by aphids after flowering. Occasionally plants were destroyed at the seedling stage. Groundsel is far more affected by aphids than by rust or mildew, with infestations often resulting in the death of the host. One may, therefore, expect that there would be stronger selection for resistance to aphids in groundsel than to either rust or mildew. There was some evidence that some groundsel lines were more severely affected than others suggesting that there may be some specificity in the relationship between the host and the aphid. Differences in resistance to different biotypes of ahid have been reported in other host species including lettuce (Eenink and Dieleman, 1982); it is possible that a similar situation may occur in groundsel.

CHAPTER 9. THE GERMINATION AND DEVELOPMENT OF ERYSIPHE FISCHERI SPORELINGS.

CONTENTS.

9.0 THE GERMINATION AND DEVELOPMENT OF ERYSIPHE FISCHERI SPORELINGS.

9.1 Introduction

The germination of powdery mildew conidia and the development of sporelings have been extensively studied in barley and wheat. The germination of Erysiphe graminis hordei and E. graminis tritici on their respective hosts, is essentially very similar. After the production of a germ tube, the sporelings may develop through a number of easily identifiable stages. The stages have been described by Ellingboe (1972), and McCoy and Ellingboe (1966) for barley mildew. Masri and Ellingboe (1966) described similar stages for wheat powdery mildew, and were as follows:

1. Formation of appressorial initials.
2. Maturation of appressoria.
3. Formation of a penetration peg which penetrates the host cuticle and epidermal cell wall.
4. Formation of haustorium in the epidermal cell.
5. Formation of a secondary hypha which elongates and is capable of initiating secondary appressoria and haustoria.

In barley powdery mildew, additional structures called primary germ tubes have also been observed (Woolacott and Archer, 1984, Kunoh et al, 1978). These develop at the opposite end of the conidium from the appressorial forming germ tube. The small initial primary germ tube produces no appressorium but it does form a contact with the host. It produces a papilla, the production of which is thought to be critical for the successful penetration of the host from the appressorial forming germ tube.

The germination of cucumber powdery mildew (E. cichoracearum) is similar to that of the cereal powdery mildews, but germination is slower, taking 14 h for the germ tube to emerge, as opposed to 1 h. The haustoria have a different appearance, the finger-like processes, observed in the cereals, curl around the central body in E. cichoracearum to form a round body that is granular in appearance (Staub et al, 1974).

The development of powdery mildews on resistant plants has been studied in an effort to determine mechanisms of disease resistance and also to distinguish the action of different resistance genes. Sporeling development on non-hosts is halted at different stages depending on the powdery mildew studied and the non-host species. Staub et al (1974) observed that the development of E. cichoracearum was halted just after the appressorial stage, without penetration of the non-host. Johnson et al (1982) suggested that the further germling development was able to progress on the non-host, the closer the host and non-host species were related taxonomically.

The effect of complete or 'major' genes for resistance has been extensively studied on the development of cereal powdery mildews. Masri and Ellingboe (1966a) found that different genes for resistance in barley and wheat halted the development of powdery mildew germlings at different stages. Often more than one stage of development was affected by each resistance gene. In most cases, the development of the germling was not affected until the haustorial stage. Depending on the gene involved, haustorial development was delayed, distorted, or a hypersensitive response was initiated in the host that restricted further development of the sporeling. However, some genes were not expressed until the mildew colony was ready to sporulate. Sporulation was then inhibited completely or severely reduced.

White and Baker (1954), investigating powdery mildew of barley, found that the proportion of sporelings forming haustoria was related

to the ultimate infection score of a particular variety. Masri and Ellingboe (1966) found that the proportion of sporelings forming elongating secondary hyphae was also related to the ultimate infection score. The frequency of elongating secondary hyphae could be used to distiguish the two host/ parasite genotypes P1/Pm1 and P4/Pm4 in the wheat and Erysiphe graminis tritici system. Stuckey and Ellingboe (1974) found that the frequency of elongating secondary hyphae could be used to distinguish the reactions of six different resistance genes, thought to be allelic, in barley to E. graminis hordei. Slesinski and Ellingboe (1970) attempted to distinguish between the three compatible host/parasite genotypes possible with a gene-for-gene interaction in wheat. The incompatible genotype, P1/Pm1 gave a significantly lower proportion of sporelings producing elongating secondary hyphae. The numbers of sporelings producing elongating secondary hyphae with the three compatible genotypes p1/Pm1, p1/pm1 and P1/pm1, was similar.

The effects of race non-specific resistance on conidial germination has also been studied. Carver and Carr (1977) studied the resistance of several oat cultivars and wild relatives of oats to E. graminis avenae. As with major resistance in wheat and barley, resistance was first expressed at the infection peg stage where there was a significant reduction in the numbers of spores producing infection pegs.

The effects of partial resistance in barley on the germination of E. graminis hordei were found to be due to the failure of the majority of the conidia to produce secondary elongating hyphae (Asher and Thomas, 1983). Thus, colony establishment was arrested at a very early stage of development, a characteristic of many major genes for resistance. Hypersensitivity and host cell collapse was mainly associated with major gene resistance. However, hypersensitivity did

appear in some partially resistant reactions and, suprisingly, in the most susceptible reactions, suggesting it to be a generalised resistance mechanism. The difference between partial and major gene resistance appeared to be only in the magnitude of their effects on each stage of germling development including the frequency of sporelings producing elongating secondary hyphae.

An investigation was made of sporeling development of E. fischeri, isolate G8, on three groundsel lines 8a, 4h and 9a. Line 8a was highly susceptible, 4h was partially resistant and 9a was highly resistant to this isolate. In this way, the stage of infection at which resistance was expressed could be determined and a comparison made between the effects of partial and complete resistance on sporeling development.

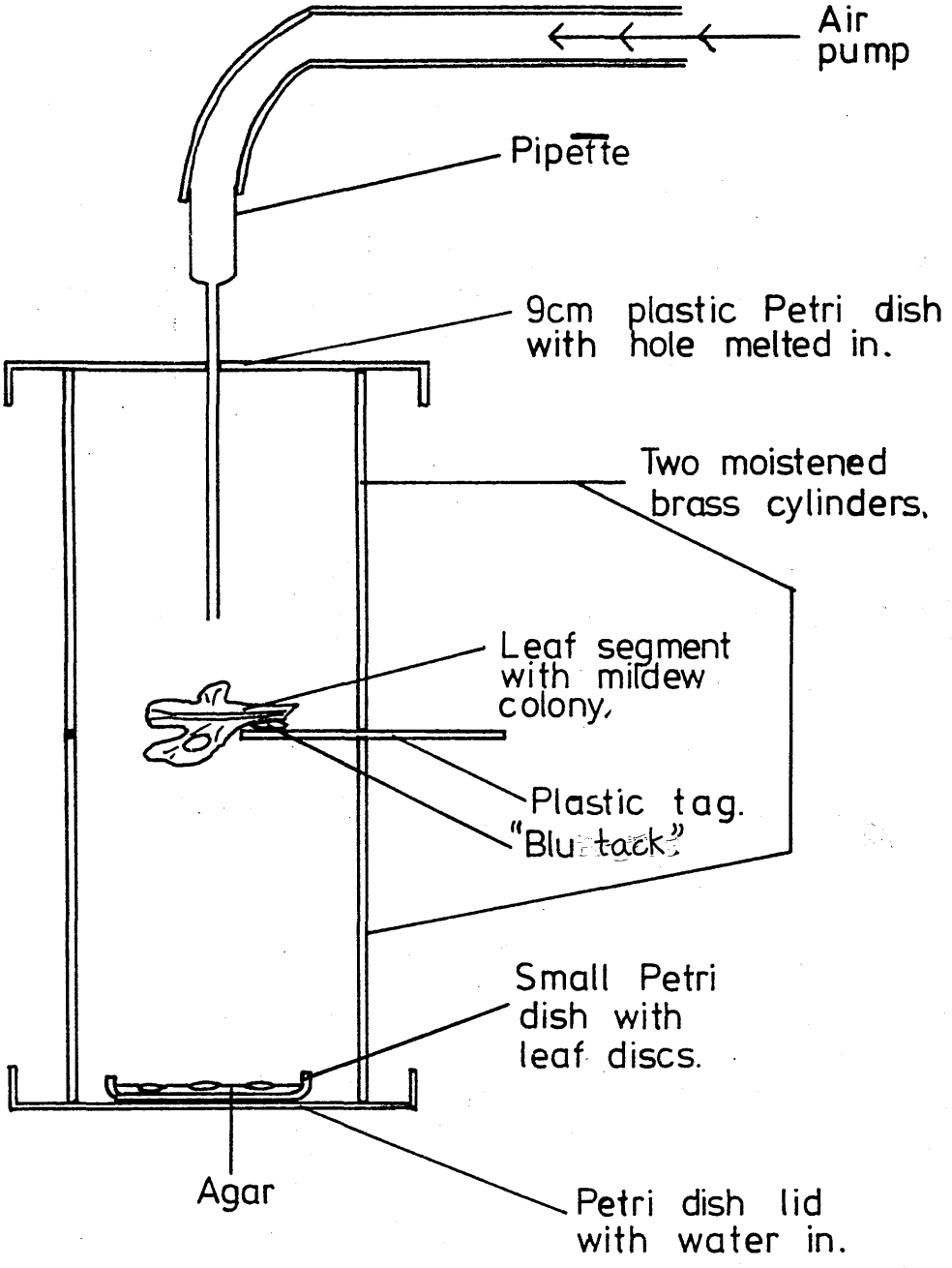
9.2. Materials and methods.

Approximately 24 h before inoculations were to be made, conidia were gently blown from suitable colonies of isolate G8 using a pipette. This ensured that the spores used to inoculate leaf discs were no more than 24 h old so that % viability was likely to be high. Thirty-six leaf discs from each of the lines 4h, 8a and 9a were cut from mildew free plants using a 5 mm cork borer. Three discs of each line were placed at random in small Pyrex Petri dishes approximately 4 cm in diameter. Each dish contained benzimidazole agar. Thus, twelve dishes, each with a total of nine leaf discs were prepared.

The discs were inoculated using a small version of a settling tower, Fig 9.1. The settling tower consisted of two brass tubes 6 cm in diameter and 10 cm in height placed on top of one another. The brass tubes were run under a cold tap to reduce static charge on them, before they were assembled. The Petri dish to be inoculated was

Fig 9.1

Inoculation tower



placed in the bottom half of a 9 cm plastic Petri dish with a little water in. The brass tubes were placed over the small Petri dish but within the larger dish. A 9 cm Petri lid with a hole in the centre was placed on top of the tower as a lid. A leaf segment with a colony of isolate G8 was attached to a flat plastic plant tag and slid between the two brass tubes so that the colony was facing upwards and over the centre of the dish to be inoculated (See Fig 9.1). A long pipette was attached to the rubber hose of the outward airflow of an air pump which had a timer attached. The air flow from the pump was gentle to avoid the tendency of spores to clump. The pipette was lowered carefully into the settling tower so that it was about 3 cm above the mildew colony. The colony was given 5 one second bursts of air. The tower was then left for the conidia to settle for at least 20 minutes.

The lids were placed on the small Petri dishes and incubated at 15°C with a 12 h photoperiod. At 24 h, 48 h and 74 h after inoculation, 4 Petri dishes were removed. The leaf discs were removed immediately and fixed in isopropanol by a method similar to that of Staub et al (1974). The leaf discs were placed in a watch glass on a drop of water, just big enough to prevent the discs from drying out and the watch glass placed in a crystallising dish with a small amount of isopropanol in the bottom. The lid was placed on the crystallising dish so that the atmosphere surrounding the discs became saturated with isopropanol. Isopropanol is highly inflammable so the dishes were placed in a fume cupboard. Isopropanol was added to the dishes as necessary until the leaves were cleared. This usually took 2 - 4 days depending on the plant line used.

Once the leaf discs were cleared they were transferred to glass slides and stained with a drop of 1% Aniline blue solution. Some conidia were successfully examined using Nomarsky interference contrast optics, but in the majority of cases staining was required.

The margins of the discs were avoided by only examining the area of disc that was at least one microscope field from the edge of the disc. The area examined encompassed 24 fields on each disc, and 12 discs of each plant line for each incubation period were recorded.

After the leaf discs were removed from the Petri dishes for fixing, the agar was immediately examined under a light microscope at x 16 magnification to observe any conidia germinating on the agar. The stages of development were recorded from fifteen fields of view per Petri dish.

9.3. Results

9.3.1 The stages of sporeling development.

Conidial germination and sporeling development of Erysiphe fischeri followed a pattern similar to that reported for the cereal powdery midews. However, there was an important exception. There were no indications that the first germ tube functioned as a primary germ tube as has been observed in E. graminis hordei (Woolacott and Archer, 1984). Where conidia made successful infections the pattern of germination was as follows.

1. A small germ-tube initial, otherwise known as a primordium, developed.
2. A germ-tube then developed from the initial.
3. An appressorium was then produced.
4. The appressorium then produced a peg.
5. A haustorium then developed, which was usually round with a ragged edge and had a granular appearance.
6. Secondary hyphae were then produced, indicating a successful infection.
7. Later these hyphae generally produced secondary haustoria and started to branch, forming a proper colony.

Multiple germ-tubes were common on the susceptible groundsel line, 8a. The second germ tube was not observed until after 48 h, obviously some hours after the first one. This indicated that additional germ tubes may be produced sequentially, not simultaneously.

Abnormal germination tended to be more frequent on the agar than on the leaf discs. Germ tubes had a tendency to branch on agar and produced appressoria with processess, which were presumably abortive

attempts to produce infection pegs and haustoria.

The total number of conidia reaching each stage of sporeling development on each of the plant lines 8a, 4h, 9a and on the agar surface are given in Figs 9.2, 9.3 and 9.4 for the incubation periods 24 h, 48 h and 74 h respectively. Chi squared analysis was carried out on the data to compare values where appropriate.

9.3.2. The germination frequency.

A conidium was said to have germinated when it had formed a primordium. There was no significant difference between the numbers of conidia germinated on the three plant lines 8a, 4h and 9a.

Germination frequency on the agar controls was significantly greater than on any of the plant lines, after 24 h and 48 h of incubation. However, by 74 h there was no significant difference between any of the plant lines and the agar, see Table 9.1.

Table 9.1 Number of conidia germinating on agar in comparison to each plant line.

Time	Comparison	χ^2	Probability.
24 h	AG v 8a	4.1	<0.05
24 h	AG v 4h	11.5	<0.01
24 h	AG v 9a	10.5	<0.01
48 h	AG v 8a	7.2	<0.01
48 h	AG v 4h	5.0	<0.05
48 h	AG v 9a	8.1	<0.01

AG = Agar surface.

9.3.3. Number of conidia producing primordia only.

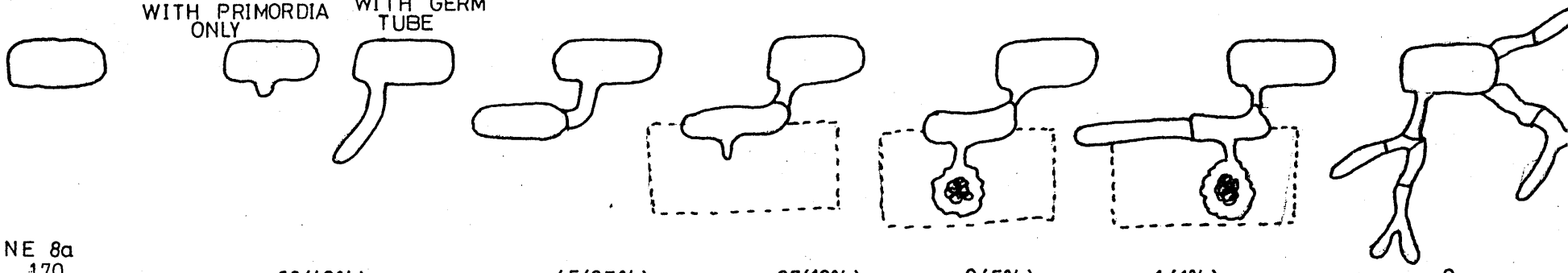
There were no significant differences between the number of conidia producing primordia on the three plant lines at 24 h or 74 h incubation. However, at 48 h incubation there were significant differences between lines 8a and 9a ($\chi^2 = 15.1$, $P < 0.001$), and between 4h and 9a ($\chi^2 = 8.9$, $P < 0.01$), but not 8a and 4h.

Initially there were no significant differences between the

Fig 9·2

NUMBER OF SPORELINGS REACHING EACH STAGE OF DEVELOPMENT. 24h INCUBATION

ALL PERCENTAGES CALCULATED USING TOTAL NUMBER OF CONIDIA APPLIED

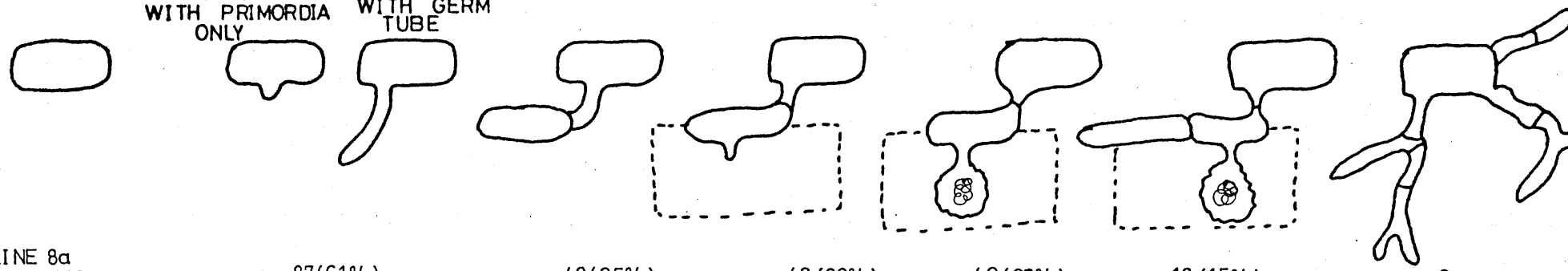


LINE 8a 170	68(40%)	12(7%)	56(33%)	45(27%)	27(16%)	9(5%)	1(1%)	0
LINE 4h 170	53(31%)	19(11%)	34(20%)	20(12%)	12(7%)	2(1%)	0	0
LINE 9a 98	29(30%)	6(6%)	23(23%)	19(19%)	10(10%)	2(2%)	0	0
AGAR 74	40(54%)	8(11%)	32(43%)	3(4%)	0	0	0	0

Fig 9·3

NUMBER OF SPORELINGS REACHING EACH STAGE OF DEVELOPMENT 48h INCUBATION

ALL PERCENTAGES CALCULATED USING TOTAL NUMBER OF CONIDIA APPLIED

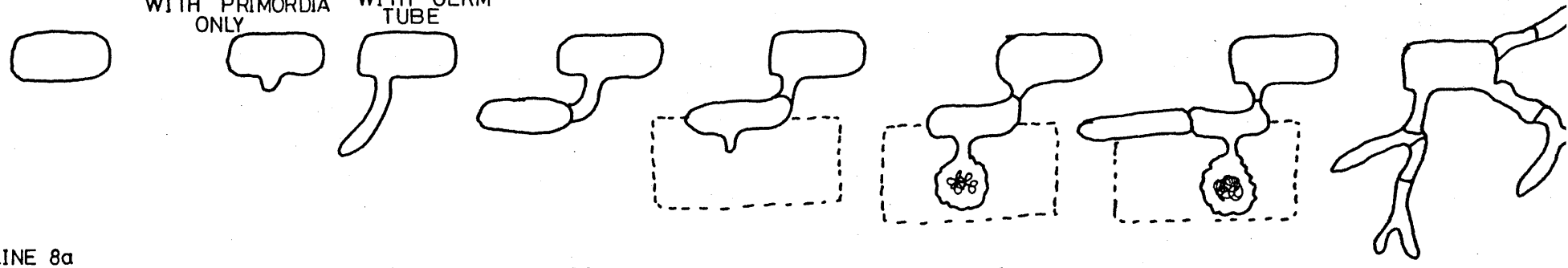


LINE 8a 142	87(61%)	30(21%)	57(40%)	49(35%)	43(30%)	40(28%)	12(15%)	0
LINE 4h 150	97(65%)	23(15%)	74(49%)	49(33%)	33(22%)	22(15%)	3(2%)	0
LINE 9a 80	46(58%)	2(3%)	44(55%)	35(44%)	22(28%)	12(15%)	0	0
AGAR 69	55(80%)	5(7%)	50(72%)	12(17%)	3(4%)	0	0	0

Fig 9·4

NUMBER OF SPORELINGS REACHING EACH STAGE OF DEVELOPMENT 74h INCUBATION.

ALL PERCENTAGES CALCULATED USING TOTAL NUMBER OF CONIDIA APPLIED



LINE 8a 152	112 (74%)	16 (11%)	96 (63%)	70 (46%)	58 (38%)	52 (34%)	42 (28%)	40 (26%)
LINE 4h 149	117 (79%)	23 (15%)	94 (63%)	67 (45%)	46 (31%)	27 (18%)	7 (5%)	5 (3%)
LINE 9a 126	107 (85%)	18 (14%)	89 (71%)	66 (52%)	42 (33%)	24 (19%)	1 (1%)	1 (1%)
AGAR 61	50 (82%)	1 (2%)	49 (80%)	10 (16%)	1 (2%)	0	0	0

number of conidia producing primordia on the agar surface or any of the plant lines. By 48 h there were significantly fewer conidia with primordia on the agar controls than on 8a ($\chi^2 = 6.5$, $P < 0.05$) but not 4h and 9a, indicating that more spores were beginning to germinate on the susceptible line, 8a. However, a closer examination of the data suggests that germination was slower on line 8a than on any of the other lines since 35% of the total number of conidia germinated on line 8a had only reached the primordial stage whilst on lines 4h, 9a and the agar surface the percentages of spores remaining at the primordial stage were 21%, 6% and 10% respectively. By 74 h incubation there were no significant differences between the proportion of conidia with primordia on any of the plant lines but there were significantly fewer conidia remaining at the primordial stage on the agar than on any of the lines ($\chi^2 = 8.8$, $P < 0.05$).

9.3.4. Number of conidia producing germ tubes.

After 24 h incubation, significantly more conidia had produced germ tubes on the susceptible line 8a than on the intermediate 4h ($\chi^2 = 7.3$, $P < 0.01$). There were no significant differences between 8a and 9a, or 4h and 9a. By 48 h there was no significant difference between the three plant lines and this was maintained at 74 h.

The numbers of conidia producing germ tubes were significantly greater on the agar controls than on 4h ($\chi^2 = 14.1$, $P < 0.001$) and 9a ($\chi^2 = 7.6$, $P < 0.01$), but not on 8a at 24 h. By 48 h the number on the agar controls was significantly greater than on any of the plant lines (8a, $\chi^2 = 17.4$, $P < 0.001$: 4h, $\chi^2 = 9.4$, $P < 0.01$: 9a, $\chi^2 = 4.9$, $P < 0.05$). By 74 h there was no significant difference between the agar controls and any of the lines.

9.3.5. The number of conidia producing multiple germ tubes.

Table 9.2 gives the numbers of spores producing 1, 2, 3 and 4 or more germ tubes at each incubation period.

Table 9.2. The total number of spores producing 1, 2, 3 and 3 or more germ-tubes.
Values for multiple germ tubes include the data from 1,2 or 3 germ tubes.

	PLANT LINE			
	V. Susc 8a	Interm 4h	V. Res 9a	Agar AG
Time = 24h				
Total no of spores	170	170	98	74
1 germ tube	56(33%)	34(20%)	23(24%)	32(43%)
2 germ tubes	0	0	1 (1%)	0
3 germ tubes	0	0	0	0
4 or more germ tubes	0	0	0	0
Time = 48 h				
Total no of spores	142	150	80	69
1 germ tube	57(40%)	74(49%)	44(55%)	50(72%)
2 germ tubes	26(18%)	6(4%)	1(1%)	1(2%)
3 germ tubes	10(7%)	1(1%)	0	0
4 or more germ tubes	0	0	0	0
Time = 74 h				
Total no of spores	152	149	126	61
1 germ tube	96(63%)	94(63%)	89(71%)	49(80%)
2 germ tubes	51(34%)	14(9%)	2(2%)	2(3%)
3 germ tubes	36(24%)	6(4%)	1(1%)	0
4 or more germ tubes	20(13%)	3(2%)	1(1%)	0

V. susc = Very susceptible, Interm = Intermediate, V. res = Very resistant.

Conidia with multiple germ tubes were first observed in the 48 h of incubation treatment. Conidia on the susceptible line 8a tended to produce more germ tubes than those on the other two lines or on the agar. This indicates that a second germ tube is more likely to develop when the first has made a successful penetration.

At 48 h there were significant differences between 8a and 9a (χ^2

= 13.9, P = 0.001), and 8a and 4h (χ^2 = 13.9, P < 0.001), but not between 4h and 9a, in the number of conidia with 2 germ tubes. Similar differences were found with respect to conidia that produced 3 germ tubes ie more conidia on 8a produced 3 germ-tubes than on 4h (χ^2 = 8.2, P < 0.01) but this time no conidia on 9a produced 3 germ tubes. At 48 h none of the conidia had produced more than three germ tubes.

By 74 h there were significant differences between all three lines with respect to the production of two germ-tubes. The highest proportion of conidia with two germ tubes was found on 8a, with slightly fewer on 4h and fewer still on 9a and the agar. The numbers of conidia producing 3 germ tubes and 4 or more germ tubes were significantly greater on 8a than 9a or 4h. However, there was no significant difference between 4h and 9a. See the Table 9.3.

Table 9.3. Chi squared values for comparisons of numbers of germ tubes.

Time	Comparison	χ^2	Probability
74 h	8a v 4h (2 germ tubes)	31.6	<0.001
74 h	8a v 9a (2 germ tubes)	45.6	<0.001
74 h	4h v 9a (2 germ tubes)	6.3	<0.05
74 h	8a v 9a (3 germ tubes)	31.3	<0.001
74 h	8a v 4h (3 germ tubes)	24.2	<0.001
74 h	8a v 9a (4 or more)	15.1	<0.001
74 h	8a v 4h (4 or more)	13.7	<0.001

9.3.6. Number of applied conidia reaching the stage of appressorium formation.

By 24 h, a significantly higher proportion of the applied conidia had produced an appressorium on line 8a than line 4h (χ^2 = 11.9, P < 0.01). There were no significant differences between 8a and 9a, or 4h and 9a. By 48 h of incubation there were no significant differences between the number of sporelings producing appressoria on any of the plant lines.

At 24 h, 48 h and 74 h significantly more sporelings germinating

on the agar controls produced appressoria than on any of the plant lines. See Table 9.4.

Table 9.4. Chi squared values for comparisons of numbers of appressoria.

Time h	Plant lines	χ^2	Probability.
24	00 v 8a	4.0	<0.05
24	00 v 4h	24.2	<0.01
24	00 v 9a	8.6	<0.01
48	00 v 8a	6.6	<0.05
48	00 v 4h	5.5	<0.05
48	00 v 9a	11.9	<0.001
74	00 v 8a	16.3	<0.001
74	00 v 4h	15.2	<0.001
74	00 v 9a	22.1	<0.001

9.3.7. Numbers of applied conidia reaching the stage of peg formation. By 24 h there were significant differences among the numbers of applied conidia producing pegs on the plant lines. More pegs were produced on line 8a than on 4h (χ^2 = 6.5, $P < 0.05$). Surprisingly, sporelings on 9a produced more pegs than on 8a or 4h, although the difference was only significant between 4h and 9a (χ^2 = 11.2, $P < 0.001$).

At 48 h there was no significant difference between any of the plant lines. At 74 h differences between the lines were again apparent. More sporelings had developed pegs on 9a than 8a (χ^2 13.1, $P < 0.001$) and on 4h (χ^2 = 5.6, $P < 0.05$). The least number of pegs were produced on 4h.

The sporelings germinating on the agar rarely produced pegs. This indicates that the presence of plant tissue may be necessary to stimulate the development of pegs.

9.3.8. Number of applied conidia reaching the stage of haustorium development.

No structures resembling haustoria were produced by sporelings on the agar.

Sporelings on all plant lines had produced haustoria by 24 h but there was no significant difference between plant lines. By 48 h, significantly more applied applied conidia resulting in sporelings on line 8a had reached the stage of producing an haustorium than on line 4h ($\chi^2 = 7.9$, $P < 0.01$) and 9a ($\chi^2 = 5.0$, $P < 0.05$). The percentage of sporelings producing haustoria on lines 9a and 4h was about the same being 15%. At 74 h, again there was a significant difference between the plant lines. More sporelings on 8a produced haustoria than on 4h ($\chi^2 = 10.1$, $P < 0.01$) and on 9a ($\chi^2 = 8.0$, $P < 0.01$). The percentage of applied conidia resulting in sporelings producing haustoria on 4h and 9a were 18% and 19% respectively; effectively there was no significant difference between them.

9.3.9. The number of applied conidia reaching the stage of producing secondary hyphae.

None of the sporelings on agar produced secondary hyphae. By 24 h very few sporelings on the leaves were producing secondary hyphae. Only one out of 170 applied conidia had developed to the stage of secondary hyphae on line 8a. There was no secondary hyphal production on 4h or 9a. At 48 h a few more sporelings had produced secondary hyphae, 11% on 8a and 2% on line 4h. This difference was significant ($\chi^2 = 7.6$, $P < 0.01$). None of the sporelings on 9a had produced secondary hyphae. By 74 h, 1 out of 126 applied conidia on line 9a had developed to the stage of secondary hyphal production. A substantial proportion of the applied conidia on 8a (28%) had developed to this stage by the same time, however, only 5% of applied

conidia had reached this stage on line 4h. The differences in the proportions of applied conidia reaching the stage of secondary hyphal production were significant between 8a and 4h ($\chi^2 = 29.1$, $P < 0.001$) and 8a and 9a ($\chi^2 = 38.0$, $P<0.001$) but not between 4h and 9a.

9.3.10. Number of applied conidia resulting in colony formation. Colonies in which hyphae had started to branch, with the branches producing haustoria, were not observed until 74 h after inoculation. Differences between plant lines were significant. More colonies were produced on line 8a than 4h ($\chi^2 = 98.4$, $P < 0.001$), and 9a ($\chi^2 = 35.7$, $P < 0.001$), but there was no significant difference between 4h and 9a. Applied conidia on line 8a produced colonies the most frequently (26%) followed by line 4h (3%) and then 9a (1%).

9.4. Discussion.

Conidial germination and sporeling development of Erysiphe fischeri followed a pattern similar to the description of cereal powdery mildew germination given by Ellingboe (1972). There were some important differences, for example, primary germ tubes that do not form appressoria have been observed in germinating E. graminis hordei and E. graminis tritici by Kunoh et al (1977) and E. graminis avenae by Carver and Carr (1977). No such primary germ tubes were observed in germinating E. fischeri conidia.

E. fischeri conidia frequently produced multiple germ tubes all of which could potentially produce an appressorium. Multiple germ tubes have been observed during the infection process of E. graminis hordei and E. graminis tritici, but only one germ tube produced an appressorium (Kunoh et al, 1977, Kunoh and Ishizakih, 1976). Other powdery mildews, such as E. betae (Drandarevski, 1978), Sphaerotheca humuli fuliginea (Sitterly, 1978) and S. humuli of hops (Royle, 1978) produce multiple germ tubes each potentially able to form an appressorium, in a similar way to E. fischeri.

Multiple germ tubes were more common on the susceptible line 8a than on 4h, 9a or the agar surface. Multiple germ tubes were not generally formed until 48 h and 74 h after inoculation, indicating that successful penetration by the first formed tube was necessary for the production of a second germ tube. A similar situation has been observed in Sphaerotheca humuli fuliginea on cucurbits where the first haustorium becomes established before additional germ tubes are produced (Sitterly, 1978). However, Kunoh et al (1977) observed that the number of germ tubes produced by E. graminis hordei and E. graminis tritici was determined as early as 4 h after inoculation indicating that successful penetration did not determine multiple

germ tube formation as only one germ tube forms an appressorium this is hardly suprising.

Germination of powdery mildews has been observed to be stimulated by the presence of host tissue (Yarwood, 1957). Staub et al (1974) observed that a higher proportion of E. graminis hordei conidia germinated on host barley tissue than on non-host cucumber tissue, whilst equal proportions of E. cichoracearum conidia germinated on cucumber and non-host barley. Suprisingly, germination of E. fischeri was initially more rapid on the agar surface than on the host tissue, but by 74 h incubation there was no difference. This may have been a reflection of a more readily available water supply from the agar, or the benzimidazole supplement to the agar stimulated germination. Relatively few conidia germinating on the agar surface produced appressoria, although 16% of conidia produced appressoria which is comparatively high compared to observations of other powdery mildews (Yarwood, 1957, Drandarevski, 1978).

Resistance in the partially resistant line 4h and the completely resistant line 9a was not clearly expressed until the haustorial stage. Significantly fewer haustoria were produced by sporelings on lines 4h and 9a than on 8a. Differences between the two resistant lines did not become apparent until secondary hyphae were produced. More sporelings produced secondary hyphae on line 4h than 9a. However, the difference was not significant, which was probably due to the low numbers of sporelings reaching hyphal production making analysis difficult. Only one sporeling out of the 126 applied conidia reached this stage on line 9a; it is possible that this may have been a contaminant or a mutant. The number of conidia producing multiple germ tubes was significantly greater on 4h and 9a, indirectly suggesting that more penetrations were successful on 4h and this criterion could be taken to distinguish the resistance of the two

lines.

Resistance to other powdery mildews has been reported to be expressed at early stages of infection, the exact stage of germination depending on the system and the resistance gene involved. In the majority of cases, resistance to E. graminis is not expressed until the haustorial stage (Ellingboe, 1972). Carver and Carr (1977) observed that various levels of race non-specific resistance were expressed as early as peg formation, a large proportion of E. graminis avenae sporelings were halted on various oat varieties at this stage. Resistance gene Rb in hop to Sphaerotheca humuli was associated with fewer sporelings developing 2 or more germ tubes and fewer resulting in sporulating colonies. Resistance gene R2 on the other hand, was expressed much earlier, only one germ tube was allowed to develop and no haustoria were produced (Royle, 1978). Ellingboe (1972), and Stuckey and Ellingboe (1974) considered that the proportion of sporelings producing secondary hyphae can be used to distinguish resistance and susceptibility, and different resistance alleles in barley.

There was some evidence from the data in Figs 9.2, 9.3 and 9.4 that the resistance of 4h and 9a could be distinguished before the haustorial and secondary hyphal stages. A slightly greater proportion of sporelings was arrested at the appressorial and peg formation stages on line 4h than line 9a. The differences were not significant but this does suggest resistance in the partially resistant line was expressed earlier. It is possible that the partial resistance of 4h is race non-specific. Race non-specific resistance is often considered to take effect earlier than complete race specific resistance. Race non-specific resistance observed by Carver and Carr (1977) in oat was expressed as early as peg formation. Work on the rust fungus Puccinia hordei by Niks (1983) has demonstrated that race

non-specific resistance in barley varieties such as Vada was expressed as a reduction in the number of sporelings successfully producing haustoria. The major gene for resistance Pa7 did not become effective until after haustorial formation when the host cell collapsed. Germination of sporelings on plants with both forms of resistance indicated that the two types of resistance acted sequentially on germination, indicating that it may be useful to combine both forms of resistance within the one variety. Resistance conferred by gene Pa3 acted earlier on conidial germination than Pa7. It was therefore difficult to distinguish the effects of partial resistance and this gene, apart from the fact that Pa3 elicited a hypersensitive reaction in the host (Niks and Kuiper, 1983).

Hypersensitivity, ie host cell collapse and necrosis, was not observed in either line 9a or 4h. Hypersensitivity has often been associated with race specific resistance but this may not necessarily be the case (Crute et al, 1985). There are several examples where genes controlling race specific resistance operate without hypersensitivity. Johnson et al (1979) found that not all the reactions resulting in incompatibility in Algerian barley to culture CR3 of Erysiphe graminis hordei resulted in hypersensitivity. Masri and Ellingboe (1966) also found that resistance in Algerian, and with Goldfoil barley varieties, having resistance genes Mla and Mlg respectively, did not express hypersensitivity to E. graminis hordei.

Only a small number of groundsel lines was examined with a single isolate, so it is impossible to determine whether the observations were typical of all groundsel and mildew interactions; far more lines would need to be tested with more isolates to establish the apparent absence of hypersensitivity and the expression of different resistance genes at different stages of sporeling development.

CHAPTER 10. GENERAL CONCLUSIONS AND FURTHER STUDIES.

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10.0. GENERAL CONCLUSIONS AND FURTHER STUDIES.

It became increasingly evident throughout the project that the *Senecio vulgaris*/ *Erysiphe fischeri* pathosystem is extremely complex. The pathogen and host appear to have evolved together to reach a state that approaches coexistence, as described by Harper (1977). The host is rarely damaged substantially by the pathogen and is able to reproduce even when it is heavily infected. Various forms of resistance have developed in the system. The variability of host and pathogen phenotypes, together with their interaction with the environment and factors such as plant age, have produced a system that is so complex that it is difficult to envisage that the system would not be able to buffer any dramatic changes in virulence in the pathogen or resistance in the host.

10.1. Factors contributing to complexity.

Some important factors contributing to the complexity of the pathosystem include:

10.1.1. Race specificity

Race specific resistance was demonstrated to be operating in the system. A minimum of fourteen genes for resistance and matching avirulence genes could explain the reaction patterns found. Fourteen genes can give rise to a maximum of 16,384 different resistance phenotypes in the host and virulence phenotypes in the pathogen. It is highly likely that only a small proportion of the total number of genes in the system was detected. Thus, the potential variability within the pathosystem for race specificity alone is enormous.

10.1.2. Heterogeneity.

The host population was composed of a highly heterogeneous mixture. At N.V.R.S. ten resistance phenotypes were detected within an area of just 1 m^2 using only five isolates. The pathogen population was also highly heterogeneous: out of 24 isolates studied, eighteen were found to be different when tested on 50 inbred groundsel lines. Thus, the system resembles a highly complex cultivar mixture.

10.1.3. Susceptibility appears more frequent than complete resistance.

The majority of the groundsel plants from both Glasgow and N.V.R.S. were susceptible to the majority of the mildew isolates. At first it appeared that complete race specific resistance did not contribute much to the host's defence strategy. However, if one considers the situation at the population level, a large number of different resistance and virulence phenotypes could tend to lead to stability. The probability of a pathogen phenotype developing which is universally highly virulent on all phenotypes of the host is low.

If more isolates had been used for testing it is possible that the majority of the plants would prove to have resistance to at least some components of the pathogen population. This is suspected because mildew collected from plants known to have resistance to the more common mildew isolates proved to be avirulent on lines previously thought to be susceptible to all mildew isolates.

10.1.4. There were some differences between populations.

The frequency of plants with complete race specific resistance to at least one isolate was greater at N.V.R.S. This was primarily due to a high frequency of resistance to an isolate from Glasgow (G9) in the N.V.R.S. plant population. This illustrated that there were some marked differences between the two host populations. The frequency of

resistance to the remaining nine isolates was also slightly higher at N.V.R.S. indicating that selection for race specific resistance may be greater under drier and therefore more favourable conditions for mildew. The number of different resistance phenotypes detected in the N.V.R.S. plants was also greater, showing that there was greater variability within this population. There was also evidence that more of the N.V.R.S. plants were resistant to the N.V.R.S. isolates than to the Glasgow isolates, if resistance to isolate G9 was disregarded. This indicates again, that there may be greater selection for resistance in the N.V.R.S. population.

10.1.5. Complex virulence.

The majority of the mildew isolates were complex for virulence and had relatively few avirulence genes. Since the host population is highly heterogeneous, selection has probably been directed towards complex virulence enabling the pathogen to colonize a wide range of host phenotypes. It is also expected that, in an asexually reproducing population, genes for virulence will build up as selection is for the whole phenotype, not just particular genes that are more likely to be exposed individually in a sexually reproducing population. However, there was no evidence that a 'super race' with matching virulence to every resistance gene in the host had developed, or at least it was not common in the mildew population. This suggests that either there is no selective advantage for a 'super race', or the 'super race' is actually less fit than races without all the virulence genes.

10.1.6. Partial resistance.

In addition to the operation of complete race specific resistance, partial resistance was also detected. Partial resistance was relatively common and was often detected in plants which also had

complete race specific resistance. Partial resistance could be race specific or non race-specific and its presence often confounded data and made it difficult to interpret. Partial resistance does appear to play an important role in the defence strategy of the host and may also have certain advantages for the pathogen which is still able to reproduce on the host. In turn, this may lead to stability in the pathosystem.

10.1.7. The environment.

Not only do climatic factors have to be favourable for mildew to occur, but there is also an interaction between the environment and host and pathogen genotype. Temperature and plant age were found to affect the expression of resistance and susceptibility of certain plant lines to certain isolates adding yet more complexity to the system. Genes in the host that are expressed only under one set of conditions will impose selection on the pathogen for shorter periods of time making it less likely that virulence in the pathogen for the resistance phenotype will build up.

10.1.8. Resistance is expressed early in the infection process.

Resistance in both a completely resistant and a partially resistant line is most markedly expressed at the haustorial stage of development. The difference between complete and partial resistance became more apparent when the sporelings produced secondary hyphae. Virtually no sporelings produced secondary hyphae on the completely resistant line, whilst a small proportion on the partially resistant line reached this stage. It is possible that partial resistance may also be expressed at later stages of colony development, as seen by characteristics such as slower growth rate and a lower production of conidia.

10.2. Possible applications to plant breeding and crop management.

The groundsel/ powdery mildew pathosytem appears to operate like a highly complex cultivar mixture. Unfortunately, it is impractical for the plant breeder to totally mimic the natural situation. However, it does seem evident that a breeding programme that aims at including partial resistance and race non-specific resistance, within cultivars that have race specific resistance, may lead to longer lasting and more effective crop protection. Possibly a cultivar mixture may prove more successful than a multiline which uses different resistance genes in the same genetic background. A cultivar mixture is more likely to be based on a wider gene background. A variable genetic background may be responsible for the differential expression of genes in different environmental conditions.

It is possibly dangerous to assume that variability will inevitably lead to stable and lasting crop protection. Likewise, cultivars relying on apparently race non-specific resistance alone may not in reality prove durable in the absence of race specificity. Several forms of resistance have probably evolved to work together in the natural situation.

Geographic cultivar diversification schemes for airborne pathogens, apart from being difficult to enforce, may not prove to be successful for the individual farmer or on the national scale. Although a barrier to an epidemic is created it may not last a whole growing season due to the efficient dispersal mechanisms of airborne pathogen propagules.

10.3. Further studies.

Generally more studies of wild pathosystems may lead to a better understanding of the function of resistance and explain why resistance utilized in crops is not always lastingly effective and how this situation can be avoided. A study of resistance to more destructive pathogens may prove interesting in comparison with the strategy of coexistence.

Further studies on the groundsel/ powdery mildew pathosystem could include:

1. Additional inheritance studies in the host to establish more firmly the presence of a 'gene-for-gene' relationship and how many genes are responsible for a particular phenotype.

2. Additional studies to establish the frequency and importance of partial resistance.

3. Studies on the effects of the environment on more resistance phenotypes.

4. Competition studies to determine selective advantages of plants with partial and complete resistance in comparison with susceptible plants in the presence and absence of mildew.

5. Studies on the effects of other pests and pathogens and their influence on the groundsel/ powdery mildew pathosytem.

6. A more extensive examination of the early infection process of more mildew isolates on more groundsel lines with a view to distinguishing the action of different resistance and virulence genes.

7. A more extensive study of the variability in the pathogen using a large number of mildew isolates on a smaller number of plants.

APPENDIX 4.1 Mean infection score of each Glasgow isolate on 50 inbred groundsel lines

Plant line	Isolate G1	G2	G3	G4	G5	G6	G7	G8	G9	G10	G11	G12
1c	2.00	0.44	2.17	2.04	3.58	2.50	1.75	2.50	1.50	2.92	2.42	3.83
1e	3.33	3.67	2.92	1.50	3.83	2.67	2.83	3.42	3.42	2.75	2.33	3.42
1f	2.92	2.94	3.67	2.83	4.00	3.58	2.83	4.00	3.67	3.58	3.83	4.00
1g	2.17	2.00	1.92	2.75	4.00	3.25	3.42	3.75	2.08	3.08	4.00	3.00
1h	2.21	0.72	1.46	1.33	2.75	2.83	2.33	3.08	1.09	1.50	3.33	2.50
1i	1.75	0.28	1.25	2.67	3.08	2.17	1.33	3.25	1.17	1.46	2.17	3.17
1m	0.83	0.61	1.42	2.50	3.83	2.50	0.92	4.00	2.92	3.83	3.75	2.00
1n	1.33	1.72	1.83	2.58	3.50	2.42	2.67	3.33	2.17	2.50	3.58	2.08
1p	4.00	2.11	2.25	3.47	3.67	2.33	2.75	4.00	1.83	2.75	3.67	1.33
1s	1.44	0.61	3.08	3.08	3.58	3.67	2.67	4.00	3.00	2.83	3.58	3.17
2a	2.89	1.44	2.58	3.17	3.83	3.83	3.17	3.33	3.33	3.58	3.67	2.92
2d	2.06	2.83	1.20	3.33	3.83	1.83	2.58	4.00	0.78	0.94	3.42	1.28
2e	2.67	2.28	1.83	2.50	3.17	2.00	1.00	3.58	1.33	1.56	2.83	0.50
2i	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3f	0.93	0.33	0.75	2.08	2.17	2.58	1.33	1.17	0.92	2.08	1.75	0.58
3g	2.17	1.83	0.83	4.00	3.08	1.25	2.00	3.42	1.44	1.06	2.42	0.50
4a	0.10	0.44	1.25	0.96	2.50	1.33	0.67	2.75	1.25	2.33	1.42	1.29
4h	0.00	0.00	0.00	3.50	4.00	0.00	0.00	2.50	0.27	1.33	3.25	0.00
5a	0.47	0.67	1.10	1.50	2.50	2.58	1.11	4.00	0.39	1.11	3.25	0.73
6b	0.00	0.00	0.00	0.00	0.00	0.25	0.00	0.00	0.00	0.00	0.00	0.21
6d	0.50	1.17	0.83	2.75	3.67	0.92	1.56	2.08	0.28	2.06	1.83	0.44
6f	0.73	0.53	1.75	3.67	3.75	0.75	1.58	3.83	0.75	3.83	3.42	1.25
7a	3.11	1.00	2.17	3.08	3.67	3.58	2.58	4.00	2.17	3.75	3.83	4.00
7b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7c	1.11	2.50	1.61	3.08	4.00	1.75	2.06	4.00	2.22	2.33	3.42	1.28
7d	0.00	0.00	0.00	0.00	0.00	0.83	0.00	0.00	1.17	0.00	0.00	1.42
7f	1.22	2.11	4.00	2.25	4.00	4.00	1.08	4.00	1.58	3.83	3.83	3.58
8a	2.67	3.00	1.22	3.00	3.00	0.92	2.72	3.67	2.06	3.06	3.00	2.00
8g	2.39	3.56	1.22	3.33	3.67	2.67	3.22	3.75	2.06	2.33	2.50	1.94
9a	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9c	0.00	0.00	0.00	0.00	0.00	1.17	0.00	0.13	1.00	0.00	0.00	1.17
9d	0.00	0.00	0.00	0.00	0.00	0.92	0.00	0.25	2.67	0.00	0.00	0.50
9g	0.63	0.00	0.00	0.25	1.17	0.00	0.00	1.92	0.00	0.00	1.13	0.07
10j	1.50	2.28	2.56	1.92	2.08	2.17	2.06	2.75	2.06	2.33	2.17	1.89
11a	1.94	0.94	1.92	2.92	3.50	2.67	2.58	3.67	3.00	2.75	3.67	3.67
11e	2.00	2.94	1.72	2.83	3.50	2.25	0.94	3.33	1.17	3.61	3.33	1.50
11i	0.00	0.00	0.00	0.00	0.00	2.33	0.00	1.58	2.92	0.00	0.38	2.33
14b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15c	3.78	3.11	3.67	4.00	3.83	2.58	3.67	4.00	3.58	3.17	4.00	2.58
15j	2.83	3.28	2.25	2.33	3.67	3.25	3.25	3.67	1.75	1.33	3.67	1.33
16d	2.28	1.22	2.25	3.61	3.50	2.42	2.75	2.67	1.71	1.08	2.92	2.25
16f	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17h	0.94	1.06	1.17	3.33	2.92	0.92	1.92	3.33	1.67	1.63	1.83	0.42
18i	2.39	2.39	2.75	2.83	2.75	3.00	1.75	3.17	2.50	3.08	3.25	2.67
19b	3.00	1.44	2.50	3.56	2.83	3.00	3.25	3.42	2.50	2.08	2.83	0.92
23f	0.67	0.33	1.00	3.89	3.58	1.75	3.33	3.08	0.46	2.00	3.75	1.04
23g	2.11	0.72	2.67	3.89	3.50	1.75	3.00	3.67	1.75	1.00	3.00	1.75
23i	0.33	1.89	2.50	3.83	3.42	1.75	3.67	3.67	1.25	2.42	3.58	3.08
24f	0.00	0.94	0.96	2.50	3.17	1.00	0.96	4.00	3.08	4.00	2.92	3.42
24j	2.78	2.50	4.00	4.00	3.42	1.83	3.42	3.67	3.50	1.00	3.58	0.83

APPENDIX 4.2 Mean infection score of each NVRS isolate on 50 inbred groundsel lines

Plant line	Isolate N1	N2	N3	N4	N5	N6	N7	N8	N9	N10	N11	N12
1c	1.33	2.08	3.83	2.17	3.33	1.83	2.25	2.50	2.04	2.00	1.50	1.08
1e	2.50	3.75	4.00	3.67	4.00	3.50	3.83	3.42	1.88	2.67	1.58	3.75
1f	0.42	4.00	4.00	3.58	3.83	3.50	3.25	3.58	3.08	4.00	1.42	3.42
1g	1.75	3.58	4.00	2.67	4.00	4.00	3.83	3.33	0.83	3.33	0.67	3.17
1h	1.46	3.42	3.08	2.42	3.67	3.17	1.42	3.33	1.25	3.00	0.50	1.58
1i	1.25	2.08	3.42	2.67	3.83	3.17	1.42	3.33	0.75	2.50	1.21	2.75
1m	1.92	2.75	4.00	0.00	3.08	2.33	2.58	2.42	0.75	3.83	1.21	3.42
1n	1.50	3.67	2.83	2.17	3.42	1.83	1.00	2.33	2.17	0.92	1.08	2.17
1p	2.08	3.75	4.00	4.00	3.50	3.67	2.58	3.67	1.58	3.08	0.50	2.67
1s	1.42	2.00	3.83	2.17	4.00	2.17	2.08	3.42	0.92	2.00	1.33	2.33
2a	2.17	3.83	4.00	2.08	3.08	2.42	3.25	3.33	2.17	2.58	0.75	1.00
2d	1.50	3.50	3.42	2.17	3.50	3.08	1.92	2.33	1.83	2.92	1.13	2.00
2e	1.08	3.25	3.33	2.58	3.67	3.33	1.33	3.50	0.92	2.25	1.50	2.17
2i	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
3f	0.88	1.67	2.17	0.38	1.75	1.00	0.75	1.58	1.13	0.92	0.46	0.96
3g	1.29	3.25	2.75	2.00	1.33	1.33	2.25	2.17	1.33	2.42	1.04	1.92
4a	1.29	1.42	1.92	1.58	2.42	1.75	0.50	1.33	0.67	1.08	0.42	1.50
4h	0.21	0.25	0.92	1.75	3.33	2.50	0.83	0.25	1.08	0.38	0.00	0.25
5a	1.50	2.25	3.25	3.42	3.00	2.50	2.92	2.75	1.54	1.83	1.21	1.17
6b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
6d	2.33	1.75	3.67	1.75	3.83	3.25	0.63	2.75	1.08	2.50	1.17	2.33
6f	0.79	2.17	2.67	0.00	2.17	1.17	0.50	2.42	1.75	1.67	0.25	2.08
7a	2.42	4.00	4.00	2.58	4.00	2.92	2.50	2.67	2.25	2.58	0.83	1.92
7b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
7c	1.25	3.50	3.58	3.25	4.00	3.08	3.33	3.08	2.08	3.33	0.42	2.58
7d	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.50	0.00
7f	2.58	3.42	4.00	4.00	3.25	3.83	2.25	3.42	1.42	3.08	0.67	3.50
8a	0.83	4.00	4.00	3.67	3.67	3.58	3.50	3.50	2.00	2.92	1.25	2.58
8g	1.83	4.00	4.00	3.83	4.00	4.00	3.83	3.17	1.83	4.00	1.33	3.25
9a	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9c	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9d	0.00	0.00	0.00	0.13	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
9g	0.00	0.13	0.92	0.38	0.38	0.75	0.25	0.33	0.00	0.33	1.17	0.54
10j	0.13	0.25	1.92	0.33	0.38	0.25	0.21	0.50	0.00	0.38	0.00	0.38
11a	2.25	3.58	3.33	2.58	3.83	2.67	3.08	3.33	1.50	2.50	1.67	2.08
11e	2.08	3.17	3.58	2.00	4.00	3.83	2.83	2.50	3.58	3.83	1.25	2.92
11i	0.00	1.08	0.67	1.25	1.58	0.50	0.21	0.38	0.08	0.33	0.67	0.33
14b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
15c	1.08	3.75	4.00	3.42	3.58	4.00	3.08	3.58	3.58	3.83	2.25	2.17
15j	0.67	3.58	4.00	3.08	4.00	4.00	3.08	3.25	2.25	2.42	1.58	3.58
16d	2.00	4.00	4.00	1.83	4.00	3.67	3.42	3.50	2.67	2.50	0.67	3.67
16f	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
17h	1.83	2.08	3.42	2.92	3.75	2.17	1.83	2.50	1.08	1.92	1.25	0.92
18i	2.33	2.17	4.00	2.83	3.08	2.25	2.58	2.33	1.33	1.83	1.75	2.17
19b	1.42	3.50	3.50	3.08	2.92	1.83	1.75	2.67	0.50	1.75	1.00	2.33
23f	3.00	3.08	3.83	1.92	3.67	3.83	3.50	3.08	1.08	3.00	1.42	3.08
23g	1.17	3.83	4.00	1.75	4.00	3.83	3.00	4.00	0.67	3.17	1.08	2.92
23i	2.75	4.00	3.67	1.75	4.00	3.42	3.08	3.33	0.83	2.50	0.67	3.42
24f	0.83	3.33	4.00	1.67	3.83	2.58	1.33	3.00	0.92	3.50	0.67	3.17
24j	0.83	3.00	2.75	3.17	3.67	3.83	2.17	3.50	1.08	3.67	2.67	2.42

APPENDIX Table 5.1 Mean infection scores produced by mildew populations obtained from 'trap' groundsel lines tested on 10 lines

	Tested on lines	Plants exposed on date shown for no of weeks shown					
		10.9.84 for 3 wks		10.9.84 for 4 wks		8.10.84 for 4 wks	
		Mean	Se	Mean	Se	Mean	Se
Mildew from line 4h	4h	2.1	0.34	2.7	0.60	1.8	0.59
	6b	0.0	0.00	0.0	0.00	1.9	0.65
	7b	0.0	0.00	2.6	0.08	0.0	0.00
	7d	0.0	0.00	0.0	0.00	3.5	0.32
	8a	3.2	0.34	3.0	0.39	2.4	0.80
	8g	2.0	0.42	2.7	0.40	2.9	0.24
	9a	0.0	0.00	0.0	0.00	0.0	0.00
	9c	0.0	0.00	1.5	0.50	3.1	0.20
	9g	0.7	0.26	0.0	0.00	0.0	0.00
	11i	0.1	0.13	3.5	0.22	4.0	0.00
Mildew from line 7b	4h	1.6	0.35	–	–	–	–
	6b	0.9	0.25	–	–	–	–
	7b	1.0	0.26	–	–	–	–
	7d	1.5	0.28	–	–	–	–
	8a	2.0	0.33	–	–	–	–
	8g	0.0	0.00	–	–	–	–
	9a	0.8	0.25	–	–	–	–
	9c	1.3	0.29	–	–	–	–
	9g	0.0	0.00	–	–	–	–
	11i	1.6	0.39	–	–	–	–
Mildew from line 7d	4h	1.5	0.38	–	–	–	–
	6b	1.0	0.41	–	–	–	–
	7b	0.5	0.20	–	–	–	–
	7d	1.7	0.26	–	–	–	–
	8a	2.0	0.18	–	–	–	–
	8g	0.1	0.10	–	–	–	–
	9a	1.4	0.42	–	–	–	–
	9c	1.4	0.37	–	–	–	–
	9g	0.4	0.28	–	–	–	–
	11i	1.7	0.32	–	–	–	–
Mildew from line 8a	4h	0.3	0.17	3.6	0.27	3.2	0.54
	6b	0.0	0.00	0.0	0.00	0.0	0.00
	7b	0.0	0.00	0.0	0.00	0.0	0.00
	7d	0.0	0.00	0.0	0.00	0.8	0.34
	8a	3.1	0.17	2.4	0.80	2.7	0.84
	8g	3.1	0.20	4.0	0.00	3.7	0.21
	9a	0.0	0.00	0.0	0.00	0.0	0.00
	9c	0.0	0.00	0.0	0.00	0.0	0.00
	9g	0.3	0.17	0.0	0.00	0.8	0.53
	11i	0.3	0.17	0.3	0.25	0.7	0.44

APPENDIX Table 5.1 continued

	Tested on lines	Plants exposed on date shown for no of weeks shown					
		10.9.84 for 3 wks		10.9.84 for 4 wks		8.10.84 for 4 wks	
		Mean	Se	Mean	Se	Mean	Se
Mildew from line 8g	4h	2.5	0.37	0.0	0.00	1.8	0.56
	6b	0.0	0.00	0.0	0.00	0.0	0.00
	7b	0.0	0.00	0.0	0.00	0.0	0.00
	7d	0.0	0.00	0.0	0.00	0.0	0.00
	8a	2.3	0.37	3.8	0.25	3.1	0.41
	8g	3.3	0.26	3.3	0.25	3.8	0.17
	9a	0.0	0.00	0.0	0.00	0.0	0.00
	9c	0.1	0.13	0.5	0.32	0.3	0.25
	9g	0.0	0.00	0.0	0.00	0.3	0.25
	11i	0.9	0.34	0.5	0.32	1.3	0.56
Mildew from line 9a	4h	1.9	0.38	–	–	–	–
	6b	0.4	0.21	–	–	–	–
	7b	1.2	0.23	–	–	–	–
	7d	1.8	0.23	–	–	–	–
	8a	2.9	0.30	–	–	–	–
	8g	0.1	0.08	–	–	–	–
	9a	2.1	0.34	–	–	–	–
	9c	2.2	0.25	–	–	–	–
	9g	0.2	0.16	–	–	–	–
	11i	2.4	0.35	–	–	–	–
Mildew from line 9g	4h	–	–	–	–	2.5	0.81
	6b	–	–	–	–	0.0	0.00
	7b	–	–	–	–	0.0	0.00
	7d	–	–	–	–	0.0	0.00
	8a	–	–	–	–	3.3	0.67
	8g	–	–	–	–	2.5	0.63
	9a	–	–	–	–	0.0	0.00
	9c	–	–	–	–	0.0	0.00
	9g	–	–	–	–	0.0	0.00
	11i	–	–	–	–	0.0	0.00
Mildew from line 11i	4h	0.9	0.35	0.0	0.00	–	–
	6b	0.0	0.00	0.9	0.44	–	–
	7b	0.0	0.00	0.0	0.00	–	–
	7d	0.0	0.00	3.2	0.42	–	–
	8a	2.1	0.40	0.3	0.25	–	–
	8g	1.6	0.43	0.5	0.32	–	–
	9a	0.0	0.00	1.7	0.76	–	–
	9c	0.0	0.00	2.3	0.48	–	–
	9g	0.0	0.00	0.0	0.00	–	–
	11i	1.0	0.26	0.4	0.00	–	–

APPENDIX Table 6.1 Mean infection scores for each plant line with each of the Glasgow isolates

	Isolate														
	G8			G9			G10			G11			G12		
Plant Line	Mean	Se	LSD at 0.05	Mean	Se	LSD at 0.05	Mean	Se	LSD at 0.05	Mean	Se	LSD at 0.05	Mean	Se	LSD at 0.05
4h	0.9	0.13	0.25	0.0	0.00	0.00	1.3	0.13	0.26	3.5	0.12	0.23	3.6	0.07	0.14
6b	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00
7b	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00
7d	0.1	0.05	0.10	2.1	0.12	0.24	0.1	0.05	0.10	0.0	0.00	0.00	0.1	0.03	0.07
8a	3.1	0.12	0.27	3.4	0.08	0.16	3.5	0.10	0.20	3.1	0.16	0.13	3.2	0.13	0.26
8g	3.2	0.11	0.22	3.2	0.11	0.21	3.5	0.10	0.20	3.3	0.13	0.26	3.1	0.14	0.27
9a	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00
9c	0.1	0.05	0.10	2.3	0.12	0.24	0.1	0.04	0.08	0.0	0.03	0.05	0.0	0.00	0.00
9g	0.2	0.07	0.13	0.4	0.10	0.20	0.7	0.13	0.25	0.9	0.16	0.30	0.4	0.11	0.22
11i	1.0	0.13	0.26	3.5	0.11	0.17	0.7	0.10	0.20	0.3	0.09	0.18	0.3	0.08	0.16

APPENDIX Table 6.2 Mean infection score of each plant line with each NVRS isolate

Plant Line	Isolate N1 Mean	Se	LSD at 0.05	Isolate N4 Mean	Se	LSD at 0.05	Isolate N5 Mean	Se	LSD at 0.05	Isolate N7 Mean	Se	LSD at 0.05	Isolate N11 Mean	Se	LSD at 0.05
1m	2.7	0.22	0.42	0.6	0.16	0.31	2.2	0.25	0.49	2.2	0.20	0.39	1.5	0.20	0.39
1n	2.4	0.22	0.42	1.1	0.21	0.41	1.7	0.26	0.51	2.5	0.28	0.55	2.0	0.28	0.55
2a	2.7	0.23	0.45	2.8	0.18	0.36	2.6	0.25	0.49	2.7	0.21	0.42	2.6	0.21	0.42
3f	0.7	0.19	0.38	0.8	0.20	0.39	1.5	0.23	0.45	1.1	0.20	0.39	1.5	0.20	0.39
4a	1.9	0.26	0.16	0.9	0.21	0.42	1.4	0.27	0.52	2.1	0.19	0.37	1.4	0.19	0.37
4h	0.6	0.15	0.29	3.0	0.13	0.25	3.5	0.18	0.35	1.0	0.16	0.31	0.6	0.16	0.31
6b	0.1	0.06	0.11	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.10	0.19	0.2	0.10	0.19
6d	2.2	0.21	0.42	2.7	0.18	0.35	2.6	0.24	0.48	2.2	0.17	0.34	2.4	0.17	0.34
6f	2.1	0.18	0.35	0.7	0.16	0.26	1.5	0.24	0.46	1.7	0.20	0.39	1.0	0.20	0.39
7b	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00
7d	0.4	0.12	0.23	0.6	0.19	0.37	0.0	0.00	0.00	0.1	0.18	0.35	0.9	0.18	0.35
8a	3.2	0.16	0.31	2.0	0.31	0.60	2.8	0.25	0.48	3.1	0.19	0.38	3.3	0.19	0.38
8g	3.0	0.20	0.39	2.7	0.22	0.44	2.9	0.18	0.34	2.5	0.19	0.38	3.0	0.19	0.38
9a	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00	0.0	0.00	0.00
9c	0.1	0.05	0.10	0.3	0.10	0.20	0.0	0.00	0.00	0.0	0.00	0.00	0.5	0.14	0.27
9d	0.0	0.00	0.00	0.2	0.10	0.19	0.0	0.00	0.00	0.1	0.07	0.13	1.0	0.19	0.37
9g	0.0	0.00	0.00	0.0	0.00	0.00	0.2	0.10	0.19	0.2	0.11	0.22	0.1	0.07	0.13
10j	1.1	0.26	0.51	1.0	0.22	0.43	0.7	0.20	0.40	1.1	0.25	0.49	1.1	0.25	0.49
11i	0.4	0.13	0.26	1.2	0.20	0.88	1.1	0.19	0.37	0.5	0.14	0.28	2.2	1.02	2.00
19b	2.1	0.27	0.52	1.5	0.25	0.49	2.6	0.19	0.38	2.1	0.24	0.47	1.6	0.24	0.46

APPENDIX Table 6.3 Ranking order of susceptibility of each plant line with each isolate

Plant line	Isolate									
	G8	G9	G10	G11	G12	Ni	N4	N5	N7	N11
4h	7	10	5	2	1	9	4	3	6	8
7d	5.5	1	5.5	9.5	7.5	4	3	9.5	7.5	2
8a	7	2	1	8	5.5	4	10	9	5.5	3
8g	4	3	1	2	5	6	9	8	10	7
9c	4	1	5	7	9	6	3	9	9	2
9g	7	4	2	1	3	9.5	9.5	6	5	8
11i	5	1	6	9	10	8	3	4	7	2

APPENDIX Table 6.4 Spearmans ranking correlation coefficients for each plant line with each other plant line

Plant line	Plant line 4h	7d	8a	8g	9c	9g	11i
4h							
7d	S -0.773						
8a	NS -0.567	NS -0.467					
8g	NS -0.006	NS +0.015	NS +0.464				
9c	S -0.637	S +0.894	NS +0.367	NS +0.249			
9g	NS +0.485	NS -0.403	+0.270	S +0.667	NS -0.212		
11i	NS -0.552	S +0.676	NS +0.124	NS -0.176	S +0.758	NS -0.352	

$$r = 1 - \frac{(6\ d^2)}{n(n^2-1)}$$

r at P = 0.05 = 0.632

r at P = 0.01 = 0.765

Appendix Table 7.1a. Morphological characters of plants grown in the glasshouse at Glasgow.

NR = Non-radiate, G = Green, DG = Dark green, P = Purple, SL = Slightly, H = Hairy, GLAB = Glabrous, Leaf type see Fig 7.1.

	Progeny	Flower type	Height in cm	No of nodes	Leaf type	Leaf colour	Leaf hairy	Stem colour	Stem hairy
Parent	A	NR	18.5	14	2	-	SL H	G	H
1	B	NR	21.0	12	2	-	SL H	G	H
	C	NR	26.5	16	2	-	SL H	SL P	H
	D	NR	28.0	22	1	-	SL H	SL P	SL H
	E	NR	26.0	14	2	-	GLAB	G	H
	F	NR	25.0	15	2	-	SL H	G	H
	G	NR	26.5	15	2	-	-	G	H
	H	NR	23.0	17	1	-	SL H	SL P	SL H
Parent	A	NR	22.0	-	2	-	GLAB	G	H
2	B	NR	17.0	14	2	G	GLAB	G	H
	C	NR	15.0	-	2	G	H	G	H
	D	NR	21.0	-	2	G	GLAB	G	H
	E	NR	8.0	12	2	G	H	G	H
	F	NR	17.0	12	2	G	GLAB	G	H
	G	NR	16.0	-	2	G	GLAB	G	SL H
Parent	A	NR	38.0	12	2	-	-	G	H
3	B	NR	34.0	16	2	-	-	G	H
	C	NR	33.5	14	5	-	-	G	H
	D	NR	31.0	16	5	-	-	-	H
	E	NR	28.0	17	5	-	-	G	H
	F	NR	21.5	14	5	-	-	G	H
	G	NR	31.0	15	5	-	-	G	H
	H	NR	29.0	-	5	-	-	G	H
	I	NR	40.0	16	3	-	-	G	H
Parent	A	NR	18.0	17	3	G	GLAB	G	GLAB
4	B	NR	19.0	19	3	G	GLAB	G	GLAB
	C	NR	20.0	16	3	DG	GLAB	G	GLAB
	D	NR	22.5	16	3	G	GLAB	G	GLAB
	E	NR	22.5	17	3	DG	GLAB	G	SL H
	F	NR	21.0	15	3	DG	GLAB	G	GLAB
	G	NR	19.5	17	3	G	GLAB	G	GLAB
	H	NR	13.5	-	3	-	-	G	H
Parent	A	NR	31.0	14	3	-	-	SL P	H
5	B	NR	27.0	21	3	-	-	SL P	H
	C	NR	31.0	24	3	-	-	SL P	GLAB
	D	NR	39.5	19	3	-	-	SL P	SL H
	E	NR	29.0	16	3	-	-	SL P	H
	F	NR	37.0	18	3	-	-	SL P	H
Parent	A	NR	10.0	14	4	G	SL H	G	H
6	B	NR	13.5	10	4	G	GLAB	G	H
	C	NR	17.0	14	4	G	GLAB	G	H
	D	NR	28.0	-	4	-	-	G	H
	E	NR	35.0	15	4	-	GLAB	G	SL H
	F	NR	32.0	-	4	-	GLAB	G	SL H

Appendix Table 7.1b. Analysis of variance for plant height and number of nodes for the plants grown in the glasshouse in Glasgow.

Plant height

	SS	df	MS	FPR	
Height	1487.49	5	743.75	26.33	= Significant
Residual	1186.39	39	28.25		at P = 0.001
Total	2673.88	44			

Number of nodes

	SS	df	MS	FPR	
No of nodes	115.46	5	23.09	4.07	= Significant
Residual	170.18	30	5.67		at P = 0.01
Total	285.64	35			

Appendix Table 7.1c. Morphological characters of the progeny families grown in the cabinets at N.V.R.S.

	Progeny	Flower	Height in cm	No of nodes	Leaf type	Leaf colour	Leaf hairy	Stem colour	Stem hairy
Parent 1	A	NR	9.5	10	4	G	-	G	SL H
	B	NR	12.0	11	4	-	-	G	SL H
	C	NR	11.5	12	2	-	-	G	SL H
	D	NR	13.0	11	4	-	-	G	SL H
	E	NR	13.5	10	2	-	-	SL P	SL H
	F	NR	15.0	12	4	-	-	G	SL H
	G	NR	11.5	10	4	-	-	G	SL H
	H	NR	13.0	10	4	-	-	G	SL H
	I	NR	9.0	10	2	-	-	SL P	SL H
Parent 2	A	NR	9.0	12	3	-	-	G	SL H
	B	NR	8.5	13	4	-	-	SL P	SL H
	C	NR	7.0	12	3	-	-	SL P	SL H
	D	NR	5.0	9	3	-	-	SL P	SL H
	E	NR	9.0	11	3	-	-	SL P	SL H
	F	NR	8.0	12	3	-	-	SL P	SL H
	G	NR	9.5	11	4	-	-	SL P	SL H
	H	NR	11.0	9	3	-	-	SL P	SL H
	I	NR	7.5	9	3	-	-	SL P	SL H
	J	NR	6.0	8	2	-	-	SL P	SL H
	K	NR	8.0	11	2	-	-	SL P	SL H
	L	NR	8.5	9	2	-	-	SL P	SL H
	M	NR	8.0	8	2	-	-	SL P	SL H
	N	NR	7.5	10	3	-	-	SL P	SL H
	O	NR	9.0	9	3	-	-	SL P	SL H
	P	NR	7.0	11	3	-	-	SL P	SL H
Parent 3	A	NR	4.5	10	1	G	H	DG	H
	B	NR	3.0	7	1	G	H	DG	H
	C	NR	3.5	8	1	G	H	DG	H
	D	NR	5.5	10	1	G	H	DG	H
	E	NR	2.5	9	1	G	H	G	H
	F	NR	4.5	10	1	G	H	DG	H
	G	NR	2.5	9	1	G	H	DG	H
	H	NR	4.5	11	1	G	H	DG	H
	I	NR	6.0	11	1	DG	H	DG	H
	J	NR	6.0	8	1	G	H	SL P	H
	K	NR	4.5	8	1	G	H	G	H
	L	NR	8.0	12	1	G	H	DG	H
	M	NR	5.0	12	1	DG	H	DG	H
	N	NR	3.5	8	1	G	H	DG	H

Appendix Table 7.1d. Analysis of variance for plant height and number of nodes for the progenies grown in the cabinets at N.V.R.S.

Plant height	SS	df	MS	FPR	
Plant height	308.33	2	154.16	62 =	Significant
Residual	89.48	36	2.49		at P = 0.001
Total	379.80	38			

Number of nodes	SS	df	MS	FPR	
No of nodes	8.27	2	4.14	1.95	Not signif
Residual	76.50	36	2.13		
Total	84.77	38			

Appendix Table 7.2 Mean infection scores of progeny family 3

	Isolate				
Progeny	N1	N4	N5	N7	N11
A	0.00	0.69	0.00	0.00	0.00
B	0.00	1.19	0.50	0.00	0.75
C	1.56	1.19	1.13	0.38	0.00
D	0.81	1.88	2.06	0.69	0.56
E	0.51	1.00	1.69	1.13	2.00
F	0.00	1.69	0.19	0.00	0.19
G	0.00	1.88	0.25	0.00	0.75
H	0.00	0.00	0.38	0.00	0.38
I	0.19	0.00	0.75	0.00	0.00
J	1.25	2.00	2.50	0.56	1.13
K	1.25	1.69	2.19	1.25	0.19
L	0.19	1.38	0.38	0.19	0.00
M	0.31	1.50	2.44	0.56	0.38
N	1.13	1.63	3.44	1.06	0.75
0	0.00	0.00	0.94	0.00	0.38
Original test					
	0.00	1.75	3.83	0.38	0.13

0.6 or less = Resistant

More than 0.6 = Susceptible

APPENDIX Table 7.3 Mean infection scores and standard deviations of control tests

Plant Line	Isolate G8 control tests									
	Inoc -		Inoc -		Inoc -		Inoc 24.5.84		Inoc 27.6.82	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	2.5	0.00	-	-	2.4	0.92	0.2	0.41	0.8	0.82
6b	0.0	0.00	0.0	0.00	0.6	0.67	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	-	-	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.7	0.52	-	-	-	-	3.4	1.02	3.1	0.74
8g	3.8	0.61	-	-	-	-	2.7	0.26	2.8	0.61
9a	0.0	0.00	0.0	0.00	-	-	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.3	0.61	0.2	0.41	0.0	0.00	0.0	0.00
9g	1.9	1.50	-	-	0.0	0.00	0.4	1.02	0.0	0.00
11i	1.6	0.92	-	-	-	-	0.9	1.07	1.0	0.77
	Inoc 6.7.84		Inoc 30.7.84		Inoc 8.8.84		Inoc 15.8.84		Inoc 24.8.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	1.7	1.29	1.4	0.80	0.0	0.00	1.2	0.98	1.6	0.92
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.5	0.78	0.3	0.61
8a	3.7	0.52	3.8	0.41	3.6	0.66	3.0	1.55	1.3	1.47
8g	2.9	1.56	2.3	1.03	3.7	0.52	3.4	0.67	3.7	0.52
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.3	0.61	0.5	0.78
9g	0.0	0.00	0.5	0.77	0.3	0.61	0.0	0.00	0.0	0.00
11i	1.7	1.29	0.0	0.00	0.0	0.00	1.8	0.52	1.3	1.13
	Inoc 5.9.84		Inoc 17.9.84		Inoc 9.10.84		Inoc 17.10.84			
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd		
4h	0.5	0.78	1.1	1.24	0.5	0.78	0.5	0.78		
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.5	0.78		
8a	3.6	0.67	4.0	0.00	2.5	0.00	2.6	0.20		
8g	4.0	0.00	3.5	0.55	2.5	0.00	3.3	0.61		
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.5	0.78		
9g	0.0	0.00	0.0	0.00	0.0	0.00	0.9	1.07		
11i	0.3	0.61	0.5	0.78	0.0	0.00	1.8	0.52		

APPENDIX Table 7.3 continued

Plant Line	Isolate G9 control tests									
	Inoc -		Inoc 13.4.84		Inoc 22.5.84		Inoc 28.6.84		Inoc 9.7.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	0.3	0.73	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6b	0.0	0.00	2.5	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	-	-	0.0	0.00	0.0	0.00	0.0	0.00
7d	1.2	1.37	0.8	0.75	2.3	0.68	1.8	0.99	2.3	1.17
8a	2.1	1.59	-	-	2.9	0.20	3.2	0.68	4.0	0.00
8g	2.1	1.70	-	-	2.3	1.17	2.7	0.26	2.9	0.20
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	1.0	1.23	2.6	0.58	2.2	0.52	2.5	0.55	3.3	0.61
9g	0.0	0.00	1.7	1.44	1.1	1.24	0.0	0.00	0.0	0.00
11i	2.9	0.20	0.0	0.00	4.0	0.00	3.7	0.52	3.8	0.41
	Inoc 29.7.84		Inoc 8.8.84		Inoc 14.8.84		Inoc 23.8.84		Inoc 4.9.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	2.0	0.48	2.0	1.16	2.6	0.59	2.3	1.13	2.7	0.26
8a	3.2	0.41	3.7	0.52	2.8	0.27	4.0	0.00	3.3	0.75
8g	3.0	0.55	2.3	1.86	3.5	0.55	3.8	0.61	3.8	0.41
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	1.6	0.92	2.8	0.26	2.8	0.82	2.8	0.26	2.5	0.00
9g	0.0	0.00	1.9	1.02	0.0	0.00	0.3	0.61	0.8	0.82
11i	3.3	0.52	3.5	0.55	3.6	0.67	4.0	0.00	3.5	0.55
	Inoc 13.9.84		Inoc 10.10.84		Inoc 18.10.84					
	Mean	Sd	Mean	Sd	Mean	Sd				
4h	0.0	0.00	0.0	0.00	0.0	0.00				
6b	0.0	0.00	0.5	0.78	0.0	0.00				
7b	0.0	0.00	0.0	0.00	0.0	0.00				
7d	2.7	0.26	2.4	0.49	1.8	0.52				
8a	3.3	0.76	3.5	0.55	3.7	0.52				
8g	3.8	0.41	3.0	0.00	3.8	0.41				
9a	0.0	0.00	0.0	0.00	0.0	0.00				
9c	2.5	0.00	1.0	0.78	2.6	0.58				
9g	0.8	0.82	0.0	0.00	0.7	1.08				
11i	3.5	0.55	4.0	0.00	3.7	0.52				

APPENDIX Table 7.3 continued

Plant Line	Isolate G10 control tests									
	Inoc –		Inoc –		Inoc 3.2.84		Inoc 11.4.84		Inoc 25.5.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	1.3	1.09	–	–	3.8	0.41	–	–	0.4	0.66
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.1	1.29	–	–	–	–	–	–	2.5	0.95
8g	2.3	1.80	–	–	3.8	0.41	–	–	2.5	0.55
9g	0.0	0.00	0.0	0.00	–	–	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.0	0.00	2.0	0.78	0.0	0.00	1.1	1.24
11i	0.0	0.00	–	–	0.0	0.00	–	–	0.3	0.61
	Inoc 29.6.84		Inoc 9.7.84		Inoc 28.7.84		Inoc 6.8.84		Inoc 13.8.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	1.5	0.00	1.3	1.13	0.3	0.61	1.3	1.13	0.5	0.78
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.3	0.61
8a	2.9	0.80	3.8	0.41	3.4	0.67	4.0	0.00	2.5	1.34
8g	2.7	0.26	2.4	1.32	4.0	0.00	3.8	0.61	2.9	0.59
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.9	1.07	0.5	0.78	1.5	0.95	0.8	1.26
11i	0.8	0.82	0.8	0.82	0.8	0.82	1.0	0.78	0.0	0.00
	Inoc 23.8.84		Inoc 3.9.84		Inoc 12.9.84		Inoc 9.10.84		Inoc 22.10.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	2.2	1.08	1.8	0.99	2.2	0.52	1.2	0.98	1.3	1.13
6b	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.8	0.82	0.0	0.00	0.0	0.00	0.3	0.61	0.0	0.00
8a	3.7	0.52	4.0	0.00	3.2	1.03	4.0	0.00	3.3	0.61
8g	3.8	0.61	3.8	0.41	3.8	0.41	3.7	0.52	3.8	0.41
9a	0.0	0.00	0.0	0.0	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.0	0.0	0.00	0.5	0.78	0.0	0.00
9g	0.3	0.61	0.0	0.00	0.0	0.00	1.5	1.23	1.3	1.13
11i	1.5	0.00	0.5	0.78	0.0	0.00	1.0	0.78	1.2	0.98

APPENDIX Table 7.3 continued

Plant Line	Isolate G11 control tests									
	Inoc –		Inoc –		Inoc 8.2.84		Inoc 9.4.84		Inoc 31.5.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	3.3	0.61	–	–	3.2	1.03	4.0	0.00	2.8	0.82
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	–	–	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.0	0.55	–	–	–	–	–	–	3.8	0.41
8g	2.5	1.55	–	–	2.7	0.26	–	–	3.5	0.55
9a	0.0	0.00	0.0	0.00	–	–	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	1.1	1.24	1.2	0.98	1.7	0.41	1.3	1.13	0.0	0.00
11i	0.5	0.78	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00
	Inoc 25.6.84		Inoc 9.7.84		Inoc 1.8.84		Inoc 10.8.84		Inoc 15.8.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	4.0	0.00	3.8	0.41	1.9	1.16	4.0	0.00	3.7	0.52
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.5	0.55	3.8	0.41	0.4	1.53	2.3	1.84	2.4	1.96
8g	3.0	0.55	3.8	0.41	1.8	1.57	4.0	0.00	3.3	0.52
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.5	0.78	1.9	1.49	0.5	0.78	0.8	1.29	0.5	0.78
11i	0.0	0.00	1.6	0.92	0.0	0.00	0.5	0.28	0.0	0.00
	Inoc 24.8.84		Inoc 16.7.84		Inoc 17.9.84		Inoc 8.10.84		Inoc 17.10.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	3.3	0.52	3.8	0.61	3.8	0.41	4.0	0.00	3.1	1.63
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.3	0.82	3.7	0.52	2.2	1.08	3.7	0.52	3.8	0.61
8g	4.0	0.00	3.3	0.75	3.8	0.61	2.7	0.61	3.3	1.63
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.3	0.61	0.7	1.08	0.0	0.00	0.8	1.29	2.1	1.02
11i	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.5	0.78

APPENDIX Table 7.3 continued

Plant Line	Isolate G12 control tests									
	Inoc -		Inoc -		Inoc 10.2.84		Inoc 10.4.84		Inoc 23.5.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	2.6	0.20
6b	0.0	0.00	0.4	0.67	0.0	0.00	1.8	0.99	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	-	-	0.0	0.00
7d	1.4	0.80	-	-	2.2	1.08	2.7	0.26	0.0	0.00
8a	2.0	1.56	-	-	2.1	1.02	-	-	3.6	0.67
8g	1.9	1.53	-	-	2.1	1.02	-	-	2.3	1.17
9a	0.0	0.00	0.0	0.00	-	-	0.0	0.00	0.0	0.00
9c	1.2	0.98	-	-	1.8	0.99	1.8	1.48	0.0	0.00
9g	0.1	0.33	0.0	0.00	1.5	1.23	1.4	1.56	0.8	0.82
11i	2.3	0.68	-	-	1.8	1.44	1.3	1.77	0.0	0.00
	Inoc 26.6.84		Inoc 5.7.84		Inoc 31.7.84		Inoc 9.8.84		Inoc 13.8.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	3.5	0.55	0.7	0.27	3.5	0.55	3.7	0.52	3.3	0.52
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.3	0.61	0.0	0.00	0.3	0.61	0.0	0.00	0.0	0.00
8a	3.0	0.95	0.5	0.21	3.4	0.67	2.4	1.96	3.0	0.78
8g	2.4	1.20	0.2	0.08	2.3	1.26	2.7	2.07	2.9	0.20
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.5	0.78	0.0	0.00	0.0	0.00	1.1	1.24	0.5	0.78
11i	0.0	0.00	0.0	0.00	0.0	0.00	0.8	0.82	0.3	0.61
	Inoc 23.8.84		Inoc 5.9.84		Inoc 17.9.84		Inoc 10.10.84		Inoc 22.10.84	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
4h	3.8	0.41	3.8	0.61	3.7	0.52	3.8	0.41	3.3	0.75
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	4.0	0.00	3.3	1.08	3.1	0.49	3.3	0.52	2.3	1.13
8g	3.5	0.55	4.0	0.00	4.0	0.00	3.7	0.52	3.3	0.82
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.0	0.00	0.0	0.00	0.8	1.29	1.3	1.37
11i	1.3	1.13	0.0	0.00	0.0	0.00	0.3	0.61	0.0	0.00

APPENDIX Table 7.3 continued

Plant Line	Isolate N1 control tests									
	Inoc 21.11.84		Inoc 5.12.84		Inoc 11.2.85		Inoc 26.2.85		Inoc 13.3.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	1.9	1.02	-	-	2.0	1.64	2.7	0.26	4.0	0.00
1n	0.0	0.00	3.4	1.24	0.8	0.82	2.5	0.55	1.7	1.29
2a	2.2	1.72	-	-	4.0	0.00	2.8	1.48	2.2	1.08
3f	0.0	0.00	1.8	1.48	1.3	1.13	1.4	0.80	1.5	1.64
4a	0.3	0.61	2.3	1.29	3.3	0.75	3.2	0.68	2.6	0.20
4h	0.0	0.00	0.4	0.67	-	-	0.0	0.00	0.5	0.78
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	2.3	0.68	-	-	3.2	0.68	1.8	0.99	3.2	1.03
6f	0.3	0.61	1.3	1.47	3.3	0.75	2.3	1.29	2.2	0.52
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.3	0.61	0.0	0.00	1.5	0.00
8a	0.8	1.29	-	-	3.8	0.41	2.3	1.13	3.3	0.75
8g	1.8	1.44	-	-	3.8	0.41	2.6	0.20	3.3	0.75
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.0	0.00	0.5	0.78	0.0	0.00	0.0	0.00
10j	0.0	0.00	0.3	0.61	2.0	0.55	0.0	0.00	0.0	0.00
11i	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.5	0.78
19b	0.3	0.61	2.6	1.56	3.3	0.99	0.5	0.78	3.0	0.78
	Inoc 13.5.85		Inoc 28.5.85		Inoc 16.7.85		Inoc 29.7.85			
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd		
1m	2.8	0.82	1.8	1.37	2.1	1.39	2.7	0.98		
1n	2.7	0.26	2.7	1.66	2.5	1.55	1.5	1.23		
2a	3.3	0.75	3.1	1.63	2.3	1.13	2.2	0.52		
3f	0.5	0.78	0.3	0.61	0.0	0.00	2.6	0.20		
4a	2.3	1.57	0.5	0.78	0.9	1.42	0.5	0.78		
4h	1.4	0.80	0.5	0.78	0.5	0.78	0.0	0.00		
6b	0.2	0.41	0.0	0.00	0.3	0.61	0.0	0.00		
6d	2.5	0.95	1.8	1.37	1.8	1.08	2.4	0.49		
6f	2.6	0.97	1.2	0.98	2.2	0.52	1.2	1.37		
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
7d	0.0	0.00	0.3	0.61	0.0	0.00	0.3	0.61		
8a	3.8	0.41	3.2	0.68	3.3	0.61	2.8	1.13		
8g	3.8	0.41	1.7	1.29	3.8	0.41	2.8	0.61		
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
9c	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00		
9d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
9g	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
10j	2.4	0.74	2.8	0.98	0.0	0.00	0.8	1.29		
11i	0.7	1.08	0.5	0.78	0.3	0.61	0.3	0.61		
19b	3.2	0.41	0.7	1.08	3.0	0.95	1.3	1.47		

APPENDIX Table 7.3 continued

Plant Line	Isolate N4 control tests									
	Inoc 12.11.84		Inoc 26.11.84		Inoc 11.2.85		Inoc 25.2.85		Inoc 13.3.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.7	1.10
1n	2.0	0.55	–	–	1.3	1.13	0.9	1.07	0.4	1.02
2a	2.1	1.02	–	–	2.6	1.24	3.0	0.78	3.0	0.78
3f	0.5	0.78	0.3	0.61	1.7	1.40	0.7	1.08	0.7	1.08
4a	1.6	1.32	–	–	1.0	0.78	0.3	0.61	0.3	0.61
4h	1.8	0.99	–	–	–	–	2.3	0.41	2.8	0.61
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	1.8	0.99	–	–	0.3	0.61	2.0	1.10	2.9	0.97
6f	0.0	0.00	0.0	0.00	1.0	0.78	0.0	0.00	0.0	0.00
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	2.2	1.08
8a	3.7	0.52	–	–	3.4	0.67	0.4	1.02	2.5	1.98
8g	3.8	0.41	–	–	3.2	0.68	2.1	1.69	4.0	0.00
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.3	0.61
9d	0.0	0.00	0.3	0.61	0.0	0.00	0.0	0.00	0.8	0.82
9g	0.0	0.00	0.8	0.82	0.0	0.00	0.0	0.00	0.0	0.00
10j	0.5	0.78	0.2	0.41	1.7	0.41	0.0	0.00	0.0	0.00
11i	1.3	0.61	–	–	0.3	0.61	0.7	0.85	0.4	1.02
19b	3.1	0.92	–	–	1.8	0.52	2.3	1.84	1.7	1.40
	Inoc 15.5.85		Inoc 28.5.85		Inoc 3.7.85		Inoc 17.7.85		Inoc 30.7.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	0.3	0.61	1.2	0.98	0.7	1.08	0.8	1.29	0.8	1.29
1n	1.6	1.53	1.3	1.13	2.0	1.10	1.8	2.04	1.8	2.04
2a	3.5	0.78	2.6	0.97	3.7	0.52	2.1	1.16	2.1	1.16
3f	0.8	1.29	0.4	1.02	1.2	0.98	1.1	1.24	1.1	1.24
4a	0.0	0.00	2.3	0.82	2.4	0.49	1.6	1.32	1.6	1.32
4h	3.5	0.55	3.3	0.75	3.3	0.99	2.5	0.55	2.5	0.55
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	3.0	1.23	3.3	0.61	3.2	0.68	1.8	0.52	1.8	0.52
6f	0.5	0.78	0.9	1.07	0.3	0.61	0.5	0.78	0.5	0.77
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.3	0.61	0.0	0.00	0.5	0.78	0.5	0.77
8a	1.0	0.78	3.3	0.75	3.0	0.95	2.1	1.69	2.1	1.69
8g	2.4	0.49	2.9	0.97	3.6	0.67	2.6	1.46	2.6	1.46
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.3	0.61	0.0	0.00	1.1	1.24	0.0	0.00	0.0	0.00
9d	0.0	0.00	0.0	0.00	0.5	0.78	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.0	0.00	0.3	0.61	0.0	0.00	0.0	0.00
10j	2.3	0.61	2.0	1.23	0.3	0.61	0.0	0.00	0.0	0.00
11i	0.5	0.78	1.7	0.41	2.2	1.21	1.2	0.98	1.2	0.98
19b	0.4	1.02	1.4	1.24	1.9	0.67	2.3	1.40	2.3	1.40

APPENDIX Table 7.3 continued

Plant Line	Isolate N5 control tests									
	Inoc 13.11.84		Inoc 26.11.84		Inoc 11.2.85		Inoc 27.2.85		Inoc 13.3.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	3.1	0.74	–	–	2.4	0.74	2.7	0.61	3.0	0.55
1n	3.4	0.66	–	–	1.0	1.23	2.3	0.68	0.3	0.61
2a	3.1	0.92	–	–	3.8	0.41	3.1	1.07	0.4	1.02
3f	1.8	0.99	–	–	0.8	0.82	1.4	1.24	2.4	1.20
4a	2.4	0.49	–	–	0.8	1.26	2.3	1.84	1.2	1.57
4h	3.3	0.52	–	–	–	–	4.0	0.00	3.8	0.41
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	3.8	0.41	–	–	4.0	0.00	3.8	0.41	2.5	1.55
6f	2.2	0.52	–	–	1.1	1.24	0.5	0.78	0.8	0.82
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	3.7	0.53
8a	3.7	0.52	–	–	4.0	0.00	3.2	1.60	2.1	1.69
8g	4.0	0.00	–	–	4.0	0.00	3.0	1.23	4.0	0.00
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.3	0.61	0.5	0.77	0.5	0.78	0.5	0.78	0.0	0.00
10j	0.0	0.00	0.8	0.82	3.7	0.52	0.0	0.00	0.0	0.00
11i	1.6	0.92	–	–	0.0	0.00	2.2	1.08	0.0	0.00
19b	2.9	0.20	–	–	3.1	1.63	2.3	1.60	2.3	1.40
	Inoc 15.5.85		Inoc 28.5.85		Inoc 15.7.85		Inoc 29.7.85			
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd		
1m	1.8	1.33	0.3	0.61	2.9	1.24	2.6	1.36		
1n	0.4	1.02	3.3	0.52	2.1	1.16	0.0	0.00		
2a	3.3	0.75	3.5	0.55	2.5	0.55	1.0	1.22		
3f	1.5	1.34	0.3	0.61	1.8	1.08	2.7	0.26		
4a	0.0	0.00	2.5	0.95	1.0	0.77	1.0	1.22		
4h	2.3	1.60	4.0	0.00	3.5	0.77	4.0	0.00		
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
6d	1.4	1.24	2.1	1.16	3.1	0.92	1.5	0.95		
6f	2.4	1.32	1.3	1.13	2.6	0.97	1.8	0.99		
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
8a	2.9	1.24	2.8	1.57	3.1	0.49	2.6	1.36		
8g	2.1	1.02	2.8	0.26	2.6	0.58	2.2	1.08		
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.3	0.61		
9d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00		
9g	0.0	0.00	0.5	0.77	0.0	0.00	0.3	0.61		
10j	2.5	0.55	0.9	1.07	0.0	0.00	0.8	0.82		
11i	1.7	0.68	0.8	0.82	1.0	0.77	0.7	1.08		
19b	3.0	0.55	2.4	0.92	3.0	0.55	2.6	0.58		

APPENDIX Table 7.3 continued

Plant Line	Isolate N7 control tests									
	Inoc 14.11.84		Inoc 26.11.84		Inoc 12.2.85		Inoc 4.3.85		Inoc 18.3.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	2.6	0.58	-	-	2.4	1.20	1.8	0.99	2.3	1.40
1n	1.0	0.77	-	-	2.3	1.13	1.6	1.32	1.5	1.22
2a	3.3	0.99	-	-	3.6	0.66	2.4	1.20	1.6	1.32
3f	0.3	0.61	1.3	0.61	1.8	0.99	0.8	0.82	1.8	0.99
4a	0.5	0.77	-	-	2.1	1.69	1.3	1.13	2.3	1.13
4h	0.0	0.00	1.7	0.41	-	-	0.3	0.61	1.8	1.08
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	0.3	0.61	1.0	0.77	3.5	0.55	1.3	1.13	1.8	0.52
6f	0.5	0.77	-	-	1.0	0.77	1.8	1.08	1.9	1.02
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.5	0.55	-	-	3.8	0.61	3.6	0.66	2.5	1.97
8g	3.8	0.41	-	-	3.8	0.61	2.6	0.80	3.3	0.61
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9d	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.3	0.61
9g	0.0	0.00	0.5	0.77	0.8	0.82	0.0	0.00	0.7	1.08
10j	0.2	0.41	0.3	0.61	3.3	0.61	1.1	1.24	0.0	0.00
11i	0.2	0.41	0.3	0.61	0.0	0.00	0.0	0.00	0.3	0.61
19b	1.8	1.37	-	-	3.8	0.61	1.7	1.40	1.6	0.92
	Inoc 13.5.85		Inoc 28.5.85		Inoc 4.7.85		Inoc 17.7.85		Inoc 30.7.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	1.3	1.47	3.7	0.52	1.7	1.03	2.2	0.75	1.3	1.66
1n	3.3	0.61	4.0	0.00	1.8	0.52	2.0	1.55	1.2	1.83
2a	3.1	0.74	3.4	0.66	3.6	0.66	3.1	0.92	2.4	0.49
3f	0.7	0.75	1.4	0.97	0.3	0.61	1.1	1.24	1.9	1.02
4a	2.6	0.97	2.5	0.55	0.0	0.00	1.5	1.22	1.2	1.37
4h	1.1	1.24	1.8	0.52	1.5	1.22	0.0	0.00	0.0	0.00
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	3.4	0.66	2.0	1.64	1.8	1.44	2.2	0.75	1.3	1.13
6f	1.2	0.98	1.3	1.13	0.9	1.07	2.2	1.08	1.3	1.13
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.0	0.00	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00
8a	3.8	0.61	3.3	1.08	2.6	0.97	2.6	1.36	2.5	1.34
8g	2.1	0.66	3.3	0.99	3.4	0.66	0.9	1.07	1.6	1.96
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9d	0.3	0.61	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9g	0.0	0.00	0.5	0.77	0.2	0.41	0.0	0.00	0.5	0.77
10j	1.5	1.22	2.8	1.13	0.8	0.82	0.0	0.00	0.0	0.00
11i	1.4	0.80	0.0	0.00	0.8	0.82	0.8	0.82	1.0	0.77
19b	3.6	0.66	0.8	0.82	2.3	1.13	3.0	0.95	0.5	0.77

APPENDIX Table 7.3 continued

Plant Line	Isolate N11 control tests									
	Inoc 21.11.84		Inoc 5.12.84		Inoc 12.2.85		Inoc 4.3.85		Inoc 18.3.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	0.0	0.00	2.4	0.49	2.5	0.00	0.9	1.07	2.2	1.08
1n	1.1	1.24	-	-	0.9	1.07	1.6	1.32	0.8	0.82
2a	0.8	0.82	-	-	3.6	0.66	3.0	0.55	1.5	1.22
3f	0.0	0.00	0.9	1.43	1.3	1.13	2.0	0.55	1.7	1.29
4a	0.4	1.02	-	-	2.9	1.24	1.8	1.13	1.8	0.99
4h	0.0	0.00	0.0	0.00	-	-	0.3	0.61	0.3	0.61
6b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
6d	1.2	0.98	-	-	4.0	0.00	1.8	0.99	2.1	0.66
6f	-	-	-	-	0.8	1.25	0.7	1.08	0.5	0.77
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	0.3	0.61	0.8	0.82	0.5	0.77	0.0	0.00	1.7	1.29
8a	1.3	1.47	-	-	4.0	0.00	2.0	1.34	3.4	0.66
8g	1.3	1.47	-	-	3.8	0.61	3.3	0.61	3.8	0.41
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	0.0	0.00	0.0	0.00	0.0	0.00	0.4	0.66	0.7	1.08
9d	0.0	0.00	0.0	0.00	0.9	1.07	0.9	1.07	1.3	1.37
9g	1.2	0.98	-	-	0.0	0.00	0.3	0.61	0.0	0.00
10j	0.0	0.00	0.0	0.00	3.0	0.55	0.3	0.61	0.0	0.00
11i	0.7	1.08	-	-	0.5	0.77	1.5	1.22	1.4	0.80
19b	1.0	0.77	-	-	4.0	0.00	2.0	1.10	0.5	0.77
	Inoc 14.5.85		Inoc 28.5.85		Inoc 5.7.85		Inoc 17.7.85		Inoc 30.7.85	
	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd	Mean	Sd
1m	1.2	0.98	1.5	1.34	2.0	0.55	2.0	0.77	1.0	1.55
1n	2.4	1.53	4.0	0.00	1.9	1.39	1.2	0.98	0.8	0.82
2a	1.6	0.92	4.0	0.00	2.8	0.26	2.7	0.26	0.8	0.82
3f	1.3	1.13	1.7	1.40	2.1	0.66	0.8	0.82	0.8	0.82
4a	1.6	0.92	1.1	1.24	0.7	1.08	0.8	0.82	0.8	0.82
4h	1.4	0.80	0.5	0.77	0.8	0.82	0.7	1.08	0.3	0.61
6b	0.8	0.82	0.0	0.00	0.0	0.00	0.3	0.61	0.0	0.00
6d	3.2	1.03	3.0	0.55	2.3	0.99	1.8	0.52	1.7	1.29
6f	1.7	1.03	0.9	1.43	1.3	0.61	1.2	0.98	1.3	1.37
7b	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
7d	1.0	0.77	0.5	0.77	1.3	0.61	1.4	0.80	0.3	0.61
8a	3.3	1.08	3.8	0.41	3.4	1.02	3.8	0.41	3.0	1.55
8g	2.5	1.34	2.5	1.34	3.2	0.41	2.8	0.82	1.8	1.63
9a	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00	0.0	0.00
9c	1.0	0.77	0.0	0.00	0.0	0.00	0.3	0.61	0.3	0.61
9d	1.6	0.92	0.3	0.61	0.3	0.61	0.8	0.82	0.3	0.61
9g	0.0	0.00	0.3	0.61	1.3	0.61	0.0	0.00	0.3	0.61
10j	2.7	1.13	2.4	0.49	0.7	1.08	0.0	0.00	0.0	0.00
11i	2.8	0.82	3.1	0.49	3.3	0.61	2.0	0.55	1.9	1.02
19b	1.8	0.99	1.0	0.77	1.4	1.56	2.8	1.57	0.8	1.29

APPENDIX Table 7.4 Mean infection scores obtained for the Glasgow plants

Plant No. Area GL3 1983	Glasgow Isolates G8	G9	G10	G11	G12	NVRS Isolates N1	N4	N5	N7	N11
GL3 01	3.83	3.58	3.67	4.00	2.83	3.75	2.75	3.17	4.00	3.33
GL3 02	4.00	3.83	4.00	3.67	3.33	3.33	4.00	3.67	3.83	3.67
GL3 03	3.42	2.83	3.42	2.75	3.17	3.83	3.42	2.50	2.58	2.50
GL3 04	1.83	2.67	2.92	3.83	2.50	2.42	4.00	3.83	3.67	2.58
GL3 05	3.58	2.42	3.33	3.17	1.83	3.17	4.00	4.00	3.75	4.00
GL3 06	2.50	3.08	2.42	2.25	2.58	3.67	3.83	4.00	3.83	2.92
GL3 07	3.67	0.00	3.17	2.67	1.08	2.92	3.67	4.00	3.17	2.92
GL3 08	2.50	2.67	2.33	2.17	1.75	0.46	2.25	2.42	2.00	2.08
GL3 09	1.67	3.25	2.67	3.42	2.58	2.58	4.00	3.42	4.00	3.08
GL3 10	4.00	3.83	4.00	4.00	3.42	3.33	3.83	3.83	4.00	4.00
GL3 11	3.83	3.33	3.25	4.00	3.83	4.00	1.67	2.92	2.08	2.42
GL3 12	3.25	2.25	3.33	3.58	2.42	4.00	3.42	3.42	4.00	3.50
GL3 13	3.50	3.25	4.00	2.75	3.33	3.42	2.17	3.42	3.83	3.17
GL3 14	3.50	3.50	3.83	2.75	2.58	3.00	3.58	3.17	3.58	3.25
GL3 15	2.17	1.08	1.42	2.25	2.58	1.75	3.08	2.17	3.33	2.75
GL3 16	3.67	3.67	3.00	3.08	3.08	3.58	1.58	2.58	3.33	2.75
GL3 17	2.33	2.00	2.83	2.75	2.58	2.33	2.00	2.75	2.92	2.58
GL3 18	4.00	0.83	3.50	4.00	2.67	3.08	3.33	3.00	3.33	3.50
GL3 19	3.83	2.00	2.33	3.00	3.50	1.75	3.42	3.17	3.75	3.58
GL3 20	3.75	3.42	4.00	3.75	2.50	3.67	2.83	4.00	3.75	3.33
GL3 21	2.83	2.33	2.75	2.75	2.42	1.63	4.00	3.83	3.50	2.83
GL3 22	2.75	2.50	1.92	2.25	2.50	3.00	2.25	2.58	2.08	2.42
GL3 23	3.67	3.25	3.83	2.83	4.00	4.00	3.50	3.83	3.58	3.25
GL3 24	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
GL3 25	4.00	3.83	3.25	3.50	3.83	3.17	1.92	2.50	0.50	2.50
GL3 26	4.00	2.75	3.67	3.00	3.33	2.17	2.50	1.13	2.75	2.67
GL3 27	4.00	3.67	3.33	2.83	2.58	1.92	2.42	3.00	2.25	2.67
GL3 28	3.17	3.08	3.50	3.67	3.25	3.33	1.92	2.25	3.08	3.58
GL3 29	3.17	0.75	2.25	3.33	3.33	3.67	3.58	3.83	3.83	4.00
GL3 30	0.29	3.58	0.08	0.00	4.00	0.58	2.17	0.38	0.63	2.50
GL3 31	2.67	0.92	2.00	2.17	2.25	1.29	3.08	0.38	1.58	1.21
GL3 32	3.33	2.83	3.17	2.75	2.17	2.75	3.83	2.33	1.83	3.25
GL3 33	4.00	3.50	4.00	3.75	3.00	3.33	3.50	3.17	3.83	4.00
GL3 34	1.08	1.67	0.92	0.50	0.92	2.42	2.58	3.42	3.83	3.50
GL3 35	3.83	3.58	4.00	3.58	3.42	4.00	4.00	4.00	4.00	3.58
GL3 36	4.00	1.83	3.83	3.33	3.17	2.83	2.92	3.17	3.83	2.25
GL3 37	3.25	3.25	1.67	2.92	1.83	1.83	2.00	1.67	1.08	2.13
GL3 38	2.08	2.67	3.00	2.75	2.08	3.08	3.67	3.67	4.00	4.00
GL3 39	3.50	3.67	3.50	3.25	3.75	3.00	3.50	3.42	2.92	3.67
GL3 40	3.75	3.25	3.83	2.50	2.00	4.00	3.83	3.83	2.75	3.42
GL3 41	4.00	1.58	3.17	3.83	0.00	4.00	4.00	3.67	4.00	4.00
GL3 42	3.83	1.00	2.25	2.42	1.50	3.25	3.42	3.67	3.67	3.83
GL3 43	3.17	2.58	2.50	2.42	2.25	2.08	1.67	1.67	1.67	2.42
GL3 44	2.08	2.58	1.67	2.50	3.50	1.13	2.92	3.17	0.13	4.00
GL3 45	4.00	2.92	3.08	2.67	1.92	3.17	3.75	4.00	3.67	3.33
GL3 46	2.42	2.50	3.75	2.83	3.17	2.08	3.17	3.17	3.00	3.08
GL3 47	3.17	2.67	1.67	1.25	2.75	–	–	–	–	–

APPENDIX Table 7.4 continued (Glasgow plants)

Plant No. Area GL2 1983	Glasgow Isolates G8	G9	G10	G11	G12	NVRS Isolates N1	N4	N5	N7	N11
GL2 01	1.25	1.92	2.00	1.08	2.25	1.58	1.04	2.42	2.42	1.83
GL2 02	2.50	2.75	2.08	1.00	2.25	3.17	0.96	2.17	2.92	1.50
GL2 03	4.00	4.00	3.50	3.50	3.42	2.92	2.17	2.67	2.92	2.58
GL2 04	1.75	1.25	2.42	2.00	2.08	2.17	0.75	1.75	1.83	1.42
GL2 05	2.50	1.92	2.25	2.67	2.33	1.79	2.17	1.42	2.42	1.50
GL2 06	1.42	1.58	0.67	1.50	1.75	1.38	0.92	1.17	1.29	1.21
GL2 07	3.17	2.92	3.50	2.00	1.58	2.92	1.50	3.25	3.83	2.67
GL2 08	2.50	2.42	0.92	2.25	2.25	1.50	1.00	1.58	3.17	2.25
GL2 09	3.17	3.17	2.92	2.50	3.50	3.17	3.42	3.58	3.42	2.00
GL2 10	3.04	2.63	2.25	2.50	1.08	1.58	1.25	1.46	1.88	1.50
GL2 11	3.42	0.54	2.92	2.25	0.00	4.00	3.83	3.50	3.67	2.92
GL2 12	2.50	1.91	1.67	0.67	0.67	2.00	1.08	1.67	1.75	0.92
GL2 13	2.17	1.42	1.83	2.25	1.00	1.58	1.67	2.50	2.00	1.83
GL2 14	3.83	2.58	3.00	2.58	3.42	3.00	1.13	1.50	2.08	1.83
GL2 15	2.75	2.33	3.17	1.83	3.00	2.75	2.83	2.17	2.67	2.42
GL2 16	3.83	3.42	2.50	3.17	3.67	1.50	1.50	2.21	3.42	1.29
GL2 17	3.17	2.17	3.50	2.67	3.00	0.88	2.08	2.00	2.42	2.75
GL2 18	3.33	3.83	4.00	3.67	3.83	4.00	3.17	2.58	3.17	4.00
GL2 19	3.25	2.50	2.50	2.58	3.33	3.17	4.00	3.50	3.00	1.50
GL2 20	4.00	2.50	3.25	3.67	3.75	3.00	2.33	3.25	4.00	3.33
GL2 21	3.17	0.00	2.58	3.17	0.00	2.75	2.83	2.08	3.83	4.00
GL2 22	3.25	3.33	3.50	3.25	2.50	0.83	1.92	1.63	2.75	2.42
GL2 23	3.83	3.67	3.25	3.42	2.83	3.75	4.00	2.75	4.00	3.00
GL2 24	3.17	2.42	2.33	2.33	2.33	1.54	1.38	2.42	2.50	2.08
GL2 25	3.08	1.83	2.92	1.73	3.00	1.17	0.50	1.58	2.67	1.38
GL2 26	3.17	3.00	2.67	2.50	2.42	2.67	0.67	2.75	3.17	1.96
GL2 27	3.33	2.17	4.00	2.92	3.83	4.00	3.17	3.33	4.00	3.75
GL2 28	2.92	3.00	3.50	2.33	2.67	2.75	1.75	2.33	2.92	3.17
GL2 29	1.58	2.50	2.75	3.33	3.50	2.17	0.96	1.92	1.83	2.50
GL2 30	1.58	1.92	2.08	1.00	0.67	1.67	0.75	2.67	3.00	1.58
GL2 31	3.00	2.17	2.92	2.75	3.58	1.67	2.08	2.83	2.67	1.92
GL2 32	2.75	2.00	4.00	0.92	3.67	2.58	1.58	3.33	4.00	2.92
GL2 33	3.00	3.08	4.00	1.92	2.75	1.67	2.08	1.33	3.17	2.75
GL2 34	2.00	3.42	1.83	2.42	3.42	1.50	1.58	0.88	3.17	2.42
GL2 35	3.33	3.50	3.33	3.08	3.83	2.50	2.42	2.33	3.83	3.00
GL2 36	3.83	3.08	4.00	2.25	2.83	3.33	2.75	2.83	3.00	3.42
GL2 37	2.92	2.92	3.67	1.75	1.33	2.50	0.92	0.75	3.08	2.33
GL2 38	1.33	2.83	2.33	3.00	3.08	4.00	3.67	2.00	3.67	3.17
GL2 39	3.25	2.50	3.67	3.67	3.67	3.83	3.00	2.92	3.25	3.08
GL2 40	2.75	3.33	3.33	2.17	2.92	3.00	1.75	2.25	1.83	0.50
GL2 41	3.42	3.58	2.42	2.42	2.92	2.92	3.58	4.00	3.67	2.83
GL2 42	2.17	2.17	1.33	1.67	3.50	1.83	3.00	1.13	0.63	0.38
GL2 43	1.92	2.50	1.67	3.25	3.08	1.04	0.79	2.67	1.75	1.33
GL2 44	2.00	2.83	2.33	2.17	3.33	2.75	2.00	2.08	2.67	1.00
GL2 45	1.50	3.25	2.50	2.25	2.75	1.58	0.75	1.38	2.25	0.92
GL2 46	4.00	2.50	3.17	2.83	2.50	1.67	1.71	3.50	2.17	2.42
GL2 47	2.17	1.25	2.75	1.75	2.75	2.50	1.50	1.83	2.33	2.42
GL2 48	2.17	1.25	3.25	2.67	3.58	1.75	2.75	1.63	1.83	1.33
GL2 49	2.25	1.92	2.25	2.00	2.58	3.83	2.92	3.58	4.00	3.83
GL2 50	2.33	3.25	2.83	1.83	1.83	1.58	2.92	1.92	3.67	2.25
GL2 51	2.42	3.83	2.50	3.00	2.92	3.58	2.33	2.75	3.50	2.83
GL2 52	3.42	4.00	1.33	2.25	1.42	2.08	3.17	3.17	2.75	2.25

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Area GL1 (Plot 1)	Isolate G8	G9	G10	G11	G12
PI 01	2.58	3.25	2.83	1.67	2.42
PI 02	3.25	3.58	3.33	2.33	3.42
PI 03	2.25	3.25	3.25	1.67	1.25
PI 04	3.00	3.42	3.25	2.42	2.83
PI 05	3.33	3.67	2.67	2.50	2.17
PI 06	2.08	2.17	3.08	1.50	2.17
PI 07	2.83	2.08	2.83	3.00	2.33
PI 08	3.25	3.50	3.58	2.42	3.17
PI 09	2.33	3.00	3.67	2.67	1.67
PI 10	3.67	3.42	3.83	2.92	2.67
PI 11	4.00	0.50	3.83	3.75	0.00
PI 12	3.42	0.00	3.08	2.58	0.00
PI 13	1.17	2.17	1.29	1.67	2.50
PI 14	3.50	3.58	3.25	3.50	4.00
PI 15	2.50	2.42	2.00	1.08	1.17
PI 16	3.67	3.08	3.42	2.33	2.50
PI 17	3.25	2.83	3.83	3.00	3.08
PI 18	2.83	3.25	3.58	2.75	3.75
PI 19	3.50	3.25	3.58	3.17	2.25
PI 20	2.75	2.67	2.33	1.58	2.33
PI 21	2.50	2.58	3.75	3.17	2.58
PI 22	3.17	3.08	2.00	1.17	2.50
PI 23	2.92	3.33	1.38	1.33	1.42
PI 24	3.83	2.83	2.67	1.50	2.58
PI 25	2.58	3.33	3.67	1.92	2.92

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Area GL1 (Plot 2)	Isolate G8	G9	G10	G11	G12
P2 01	3.08	3.67	2.75	2.67	2.58
P2 02	3.50	3.00	3.17	3.67	3.67
P2 03	3.00	3.67	3.67	3.00	3.17
P2 04	3.33	2.58	3.33	2.25	2.33
P2 05	2.33	2.83	3.25	2.17	2.58
P2 06	3.08	3.08	3.00	2.67	2.67
P2 07	3.00	2.17	3.00	2.17	0.67
P2 08	1.83	2.50	3.83	2.33	2.33
P2 09	3.33	3.17	3.08	2.67	2.75
P2 10	3.25	2.75	3.58	1.33	2.58
P2 11	3.17	3.50	1.92	2.17	2.25
P2 12	1.50	1.75	3.17	2.92	3.50
P2 13	1.42	3.08	3.25	2.58	2.83
P2 14	3.33	3.08	3.75	1.42	3.17
P2 15	2.00	1.50	3.00	2.67	1.83
P2 16	2.00	1.17	2.67	2.17	1.92
P2 17	2.08	1.67	2.17	1.08	0.67
P2 18	1.92	1.33	2.58	0.75	2.08
P2 19	2.75	2.00	2.33	1.83	3.00
P2 20	2.00	2.75	2.00	1.83	2.00
P2 21	3.42	2.83	3.83	3.83	4.00
P2 22	2.08	0.67	2.17	2.50	2.08
P2 23	3.50	2.42	3.83	3.83	4.00
P2 24	2.25	0.25	2.58	2.00	1.42
P2 25	2.58	1.17	3.00	2.92	3.08

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Area GL1 (Plot 3)	Isolate G8	G9	G10	G11	G12
P3 01	1.92	1.67	1.75	1.08	1.75
P3 02	3.08	3.67	3.83	3.00	3.67
P3 03	2.17	1.58	2.17	1.42	1.50
P3 04	3.42	2.33	1.50	2.08	1.17
P3 05	2.83	1.92	1.83	0.92	2.17
P3 06	2.83	1.83	2.33	0.75	1.92
P3 07	2.17	2.08	2.67	1.92	2.08
P3 08	1.08	2.00	2.92	2.08	1.92
P3 09	3.00	2.33	3.50	2.00	1.67
P3 10	3.00	3.33	3.42	3.08	2.92
P3 11	2.17	2.83	2.92	1.13	1.58
P3 12	3.25	2.33	2.58	3.08	2.50
P3 13	3.08	2.25	1.00	2.08	3.25
P3 14	2.58	3.17	3.75	3.33	3.00
P3 15	3.67	1.83	2.00	2.50	1.25
P3 16	3.08	3.58	3.33	2.92	3.67
P3 17	2.00	2.50	2.67	2.25	2.92
P3 18	2.17	2.00	1.33	1.92	1.58
P3 19	2.75	3.17	2.67	2.25	2.75
P3 20	3.17	1.67	0.83	3.33	1.58
P3 21	2.92	1.83	2.25	2.17	2.67
P3 22	2.17	2.58	3.17	3.42	2.17
P3 23	3.25	2.83	3.33	3.33	3.83
P3 24	3.33	3.75	3.58	3.00	2.75
P3 25	3.42	3.33	3.25	3.17	3.50

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Area GL1 (Plot 4)	Isolate G8	G9	G10	G11	G12
P4 01	2.67	2.83	2.67	2.67	3.00
P4 02	2.67	2.58	2.83	2.50	2.42
P4 03	3.33	1.25	3.42	3.08	0.46
P4 04	3.17	2.75	2.75	3.08	2.92
P4 05	2.75	2.42	2.67	2.33	2.92
P4 06	3.17	2.50	2.67	2.17	2.67
P4 07	2.83	2.50	2.67	2.67	2.92
P4 08	1.50	2.83	2.33	2.42	2.50
P4 09	2.33	2.50	3.67	2.67	2.67
P4 10	3.17	3.58	2.75	2.50	3.25
P4 11	3.50	2.33	3.17	2.42	2.58
P4 12	2.92	2.67	2.75	2.50	2.67
P4 13	2.83	2.42	2.75	2.58	2.67
P4 14	2.00	3.25	2.58	2.75	2.50
P4 15	2.83	3.17	2.75	2.33	3.33
P4 16	3.00	2.75	3.00	2.83	3.67
P4 17	3.00	3.00	2.50	2.83	2.75
P4 18	2.25	3.00	2.17	0.88	2.17
P4 19	3.67	3.08	3.42	2.50	3.25
P4 20	2.58	1.42	2.50	1.33	1.50
P4 21	2.67	1.92	2.33	2.08	3.17
P4 22	2.50	2.58	1.67	0.92	2.42
P4 23	1.92	3.17	2.92	2.75	2.42
P4 24	1.50	1.83	1.67	1.83	1.58
P4 25	1.83	2.25	2.17	1.08	2.33

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Soil Sample (Plot 1)	Isolate G8	G9	G10	G11	G12
SP1 01	3.08	0.00	4.00	3.08	0.00
SP1 02	2.17	0.83	3.33	2.83	3.50
SP1 03	2.17	3.42	3.08	3.17	2.67
(Plot 2)					
SP2 01	1.92	2.67	1.92	1.83	3.00
SP2 02	3.00	1.75	2.58	2.50	3.50
SP2 03	3.33	2.75	3.67	2.75	3.67
SP2 04	2.83	2.00	3.58	2.33	2.50
SP2 05	2.67	4.00	2.42	3.17	3.42
SP2 06	2.67	1.75	3.25	2.58	2.92
SP2 07	2.58	3.33	2.17	2.92	1.50
SP2 08	3.25	2.67	2.58	2.92	2.92
SP2 09	2.25	2.08	2.25	3.00	2.50
SP2 10	1.17	1.67	2.75	2.75	2.83
SP2 11	3.50	2.33	2.67	3.50	3.00
SP2 12	2.92	3.25	3.58	2.33	2.25
SP2 13	2.75	1.58	2.42	2.25	2.75
SP2 14	2.92	2.50	3.33	2.67	3.67
SP2 15	3.33	3.33	3.58	1.83	3.33
SP2 16	4.00	0.13	4.00	3.33	0.13
SP2 17	3.83	2.33	2.67	1.58	3.08
SP2 18	4.00	4.00	3.25	2.58	2.92
SP2 19	2.17	1.92	3.50	3.17	2.33
SP2 20	3.42	3.50	3.83	3.08	3.42
SP2 21	2.42	3.33	2.50	3.25	3.08
SP2 22	2.75	2.75	3.83	3.83	3.08
SP2 23	3.00	3.17	3.25	2.25	3.25
SP2 24	3.42	3.42	3.08	2.58	2.83
SP2 25	1.42	1.33	3.75	2.58	2.92
(Plot 3)					
SP3 01	1.25	2.50	2.25	2.00	2.50
SP3 02	2.75	1.75	2.67	3.25	2.75
SP3 03	2.50	2.42	2.83	1.83	2.50
SP3 04	3.50	3.08	4.00	2.75	3.67
SP3 05	2.92	3.00	3.83	3.25	3.25
SP3 06	3.83	3.75	3.83	2.75	4.00

APPENDIX Table 7.4 continued (Glasgow plants, NVRS isolates not tested)

Plant No Soil Sample (Plot 4)	Isolate G8	G9	G10	G11	G12
SP4 01	3.75	3.25	3.67	2.75	2.92
SP4 02	3.33	2.67	2.67	3.33	3.00
SP4 03	2.00	2.42	2.83	1.17	2.42
SP4 04	2.08	2.42	4.00	3.08	2.83
SP4 05	2.58	1.83	3.67	2.00	2.75
SP4 06	3.17	2.67	3.75	3.17	3.00
SP4 07	3.00	1.75	2.33	2.33	2.58
(Private Garden)					
PG 01	3.08	3.25	3.83	3.67	3.17
PG 02	2.92	3.33	4.00	3.75	3.75
PG 03	3.67	2.25	2.75	3.17	3.17
PG 04	3.75	2.50	3.75	3.67	3.83
PG 05	3.58	3.58	4.00	3.75	4.00
PG 06	3.17	2.75	1.50	4.00	4.00
PG 07	3.33	3.67	3.42	3.50	3.25
PG 08	3.50	3.58	4.00	3.75	4.00

APPENDIX Table 7.5 Mean infection scores obtained for the NVRS plants

Plant No. Area N3 1984	Glasgow Isolates G8	G9	G10	G11	G12	NVRS Isolates N1	N4	N5	N7	N11
84 N3 01	2.83	3.33	1.83	4.00	4.00	3.08	2.50	3.50	2.25	3.33
84 N3 02	3.00	2.08	1.00	3.00	2.33	2.67	2.25	3.58	3.67	1.75
84 N3 03	3.83	2.08	2.33	3.58	4.00	4.00	0.67	3.67	3.17	2.50
84 N3 04	3.67	2.67	3.33	3.83	3.42	2.42	2.42	3.58	1.83	3.33
84 N3 05	0.00	0.00	0.00	4.00	3.83	2.25	3.83	3.25	2.92	2.08
84 N3 06	2.08	0.00	2.17	0.75	1.25	1.21	0.83	1.42	0.00	0.56
84 N3 07	2.25	2.50	2.42	3.42	3.33	3.00	1.50	2.83	2.08	2.50
84 N3 08	3.00	2.92	3.83	2.83	3.08	3.00	0.88	1.58	1.67	3.00
84 N3 09	2.17	1.50	1.50	1.33	1.42	2.00	0.46	2.00	2.92	1.50
84 N3 10	0.83	1.75	1.25	0.83	1.33	2.42	1.50	2.33	2.17	1.58
84 N3 11	2.83	2.75	2.50	0.83	3.08	2.67	3.42	4.00	2.92	3.00
84 N3 12	2.08	1.50	1.58	2.17	3.08	1.75	0.50	0.33	1.50	1.00
84 N3 13	3.08	3.42	3.17	2.83	3.42	2.17	2.33	2.67	1.29	2.75
84 N3 14	2.42	1.75	1.33	1.83	2.00	0.13	1.33	1.58	0.92	0.46
84 N3 15	1.50	0.50	1.92	1.58	1.50	2.58	2.54	3.08	3.83	2.42
84 N3 16	0.00	0.00	0.00	1.83	0.92	2.00	0.00	0.00	2.83	2.38
84 N3 17	2.50	1.92	3.25	2.75	3.33	2.67	2.67	3.83	2.08	1.00
84 N3 18	1.50	0.50	2.67	2.17	1.25	0.63	0.79	1.67	1.00	1.17
84 N3 19	0.92	2.67	3.67	3.25	3.00	2.25	2.92	2.83	1.75	2.75
84 N3 20	2.58	1.33	2.00	1.50	2.08	1.50	2.21	2.33	1.67	0.92
84 N3 21	2.33	0.50	3.00	2.75	3.00	2.00	1.42	2.92	1.75	1.75
84 N3 22	1.00	1.08	2.00	1.42	3.08	0.50	0.79	1.33	1.08	1.50
84 N3 23	1.92	2.58	2.17	2.42	2.25	1.83	1.42	2.83	2.17	2.50
84 N3 24	0.75	2.00	0.75	2.83	3.17	0.63	0.13	0.96	0.38	0.00
84 N3 25	3.00	2.92	2.83	3.08	3.50	2.33	1.00	2.75	2.58	2.00
84 N3 26	3.25	0.00	3.42	2.33	2.33	3.42	2.75	3.83	2.58	2.83
84 N3 27	3.42	0.88	2.42	2.92	1.75	2.92	2.00	3.17	2.50	2.25
84 N3 28	2.75	2.33	3.83	2.58	2.92	1.04	0.30	0.33	0.25	0.21
84 N3 29	2.58	2.58	3.75	2.83	1.75	2.00	2.25	2.00	1.54	2.25
84 N3 30	2.00	3.17	3.25	2.58	1.08	2.38	0.88	2.33	1.92	1.96
84 N3 31	1.67	1.50	2.50	2.17	1.00	0.25	0.25	0.46	0.58	0.25
84 N3 32	3.00	2.00	3.08	2.92	4.00	2.42	1.38	2.83	2.04	2.29
84 N3 33	2.75	2.92	2.67	1.33	3.25	1.58	1.67	2.00	1.75	1.83
84 N3 34	0.75	0.67	2.25	1.42	1.00	1.21	1.75	0.63	0.96	1.00
84 N3 35	3.83	3.00	3.00	3.25	2.08	1.92	1.13	0.08	1.58	0.75
84 N3 36	2.25	2.83	2.42	2.17	1.58	2.33	1.92	0.63	1.33	1.75
84 N3 37	3.17	3.08	3.83	2.92	3.17	3.00	0.50	2.75	0.58	0.83
84 N3 38	2.92	2.17	1.83	2.00	2.33	1.13	0.63	0.75	1.13	2.08
84 N3 39	3.25	2.17	3.58	3.42	2.17	2.67	1.46	2.00	2.67	1.25
84 N3 40	2.08	1.92	3.67	1.58	1.75	2.58	2.08	2.42	1.83	2.33
84 N3 41	4.00	2.58	3.83	3.25	2.92	1.67	1.92	1.63	2.33	2.58
84 N3 42	3.42	1.17	2.83	1.50	1.75	2.42	1.50	2.42	0.38	1.33
84 N3 43	2.08	1.17	2.17	3.33	1.00	0.75	0.67	2.17	1.13	1.04
84 N3 44	3.17	2.75	2.08	2.08	2.83	3.33	2.33	2.67	1.83	1.63
84 N3 45	3.17	2.25	1.67	2.25	3.00	3.50	2.25	3.33	2.25	2.67
84 N3 46	2.50	0.63	2.92	3.00	1.50	1.92	0.50	2.92	2.17	2.83
84 N3 47	1.83	0.75	1.67	1.33	2.50	2.92	2.50	2.92	2.17	2.17
84 N3 48	3.08	2.50	3.33	2.42	2.42	1.75	1.50	1.96	1.92	2.67
84 N3 49	1.00	1.92	0.75	1.75	2.17	1.13	1.50	1.54	1.21	1.08

APPENDIX Table 7.5 continued (NVRS plants)

Plant No. Area N3 1984	Glasgow Isolates G8	G9	G10	G11	G12	NVRS Isolates N1	N4	N5	N7	N11
84 N3 50	3.42	2.75	2.92	1.83	2.58	2.67	1.42	1.92	2.67	0.88
84 N3 51	2.42	1.83	2.58	1.08	2.17	1.58	1.13	3.00	1.50	1.25
84 N3 52	1.00	2.33	2.83	2.00	1.83	2.17	1.58	2.33	1.50	1.67
84 N3 53	2.83	1.58	1.58	1.42	2.33	0.96	0.25	0.71	0.75	0.92
84 N3 54	1.50	3.17	2.67	1.25	2.33	2.50	3.58	3.83	2.17	2.50
84 N3 55	2.75	2.58	0.50	2.50	0.92	0.63	0.46	1.13	1.92	0.38
84 N3 56	2.17	1.46	0.75	0.38	1.00	0.38	0.13	0.38	0.00	0.50
84 N3 57	1.92	2.50	3.17	1.67	1.58	0.50	0.25	0.25	0.75	1.54
84 N3 58	3.25	2.25	3.83	3.25	2.50	2.33	2.17	3.30	2.17	3.42
84 N3 59	1.17	2.58	1.42	2.25	2.08	0.67	0.38	0.67	0.63	0.63
84 N3 60	3.25	2.58	4.00	2.92	2.33	1.33	2.00	1.83	1.46	2.17

APPENDIX Table 7.5 continued (NVRS plants)

Plant No. Area N2 1984	Glasgow Isolates G8	G9	G10	G11	G12	NVRS Isolates N1	N4	N5	N7	N11
84 N2 01	1.83	3.67	3.67	3.33	1.21	1.67	1.83	3.00	2.92	2.83
84 N2 02	1.83	2.92	3.25	2.67	1.25	1.42	2.83	3.00	1.58	1.42
84 N2 03	2.67	4.00	3.67	4.00	1.33	2.50	2.75	3.42	3.25	3.25
84 N2 04	3.00	2.92	3.33	3.50	2.75	4.00	3.83	3.58	4.00	4.00
84 N2 05	2.50	2.58	3.67	3.67	3.83	3.25	2.17	3.83	2.67	3.58
84 N2 06	2.67	0.00	4.00	3.83	4.00	3.08	3.83	4.00	3.50	3.42
84 N2 07	3.00	3.42	3.00	2.25	2.83	2.00	1.58	3.08	2.17	3.00
84 N2 08	3.33	0.00	4.00	3.67	3.17	3.00	3.17	4.00	3.25	3.83
84 N2 09	1.58	3.17	2.92	2.75	1.88	2.00	1.67	3.08	3.33	2.42
84 N2 10	2.75	3.33	2.83	3.25	1.58	2.58	3.00	3.08	2.33	2.33
84 N2 11	0.25	3.08	1.00	2.00	2.25	0.13	3.17	3.58	0.00	0.00
84 N2 12	2.42	2.83	2.58	2.75	3.42	2.00	1.67	3.75	1.42	3.58
84 N2 13	1.83	2.75	3.17	1.67	3.33	2.50	2.00	2.33	2.08	2.92
84 N2 14	0.00	3.00	2.75	2.33	3.08	0.00	1.75	3.83	0.38	0.13
84 N2 15	1.83	0.50	4.00	2.75	2.83	2.83	3.67	3.08	1.50	3.42
84 N2 16	2.08	0.00	3.50	2.83	2.50	1.50	1.50	3.42	3.33	3.17
84 N2 17	2.17	2.75	3.17	0.92	1.08	3.33	1.83	3.67	2.67	3.83
84 N2 18	0.96	2.00	0.92	2.92	0.75	0.88	1.38	2.08	0.13	1.04
84 N2 19	1.00	1.33	1.67	1.75	0.83	2.00	1.08	2.33	0.33	2.08
84 N2 20	2.67	3.83	2.17	2.42	2.92	0.79	2.50	3.67	2.58	2.67
84 N2 21	3.33	0.00	4.00	2.33	3.08	2.08	2.00	3.75	2.33	3.83
84 N2 22	2.17	3.08	2.42	2.25	2.92	1.13	1.58	1.29	0.25	0.92
84 N2 23	3.08	2.67	3.83	2.67	3.17	3.42	1.17	4.00	1.50	3.17
84 N2 24	3.50	3.42	4.00	2.00	4.00	2.08	3.00	3.50	1.75	3.42
84 N2 25	2.58	0.00	3.00	3.67	3.42	2.42	2.33	2.58	2.50	3.08
84 N2 26	2.58	2.92	3.83	1.42	1.83	3.08	1.67	3.42	3.08	2.50
84 N2 27	3.33	0.00	3.67	2.33	3.25	2.92	2.67	2.50	2.33	3.50
84 N2 28	3.33	0.00	3.83	4.00	4.00	2.50	2.50	3.83	4.00	3.83
84 N2 29	1.00	2.50	2.83	1.92	2.67	2.92	2.75	3.33	2.75	2.83
84 N2 30	3.50	0.00	2.67	3.33	2.25	1.92	3.08	3.17	2.58	3.17
84 N2 31	3.00	0.00	4.00	3.08	2.33	2.17	3.42	3.75	2.33	3.33
84 N2 32	2.83	3.33	4.00	2.08	3.58	2.25	2.75	3.25	2.83	2.67
84 N2 33	2.75	3.58	3.33	2.00	1.92	2.50	1.92	3.25	2.50	3.83
84 N2 34	1.33	2.67	3.08	1.75	3.42	1.83	1.58	2.75	1.50	2.00
84 N2 35	2.83	3.25	2.67	0.92	1.75	2.67	1.00	2.83	2.50	3.83
84 N2 36	2.50	3.08	3.00	2.33	2.67	3.42	2.42	2.92	2.33	3.00
84 N2 37	2.83	1.08	4.00	3.25	2.67	3.50	2.67	3.00	2.08	3.67
84 N2 38	2.50	0.00	4.00	2.50	3.83	2.00	3.25	4.00	2.08	4.00
84 N2 39	3.58	4.00	4.00	2.92	3.67	2.75	1.17	2.50	2.92	2.83
84 N2 40	2.67	0.50	2.58	3.25	3.08	2.08	3.25	3.17	2.08	2.25
84 N2 41	3.67	0.00	3.83	2.83	3.75	3.50	2.08	2.75	3.75	3.50
84 N2 42	3.33	2.17	3.33	1.33	2.17	2.08	1.67	3.33	2.42	3.58
84 N2 43	2.92	1.58	3.67	3.17	3.00	2.75	3.75	3.83	3.50	2.58
84 N2 44	2.42	0.00	3.83	3.42	3.58	2.92	2.25	4.00	2.83	3.42

APPENDIX Table 7.5 continued (NVRS plants)

Plant No.	Glasgow Isolates					NVRS Isolates				
Area N2 1983	G8	G9	G10	G11	G12	N1	N4	N5	N7	N11
83 N2 01	3.83	2.58	2.33	3.17	3.08	4.00	2.17	3.58	3.67	2.83
83 N2 02	2.33	2.50	1.50	1.17	2.00	3.33	1.50	2.33	2.33	3.33
83 N2 03	0.75	2.58	0.67	2.17	2.58	0.13	0.96	3.08	0.00	0.00
83 N2 04	3.33	0.00	2.25	2.83	3.17	3.67	3.83	3.67	3.08	3.83
83 N2 05	3.83	0.00	2.17	3.08	3.00	3.75	2.25	3.58	2.92	3.33
83 N2 06	0.25	1.92	0.00	2.33	2.83	0.00	2.00	2.17	0.13	0.13
83 N2 07	2.00	1.67	2.08	2.92	2.50	3.33	0.92	3.25	3.00	2.50
83 N2 08	2.92	0.00	2.08	1.67	2.58	2.92	1.67	3.83	2.67	2.33
83 N2 09	2.67	0.00	1.33	2.33	2.25	3.88	3.83	4.00	3.83	2.75
83 N2 10	2.75	2.50	0.92	2.08	2.75	2.75	1.67	3.83	1.08	3.25
83 N2 11	3.08	2.92	1.17	2.67	1.83	2.42	1.92	3.50	2.50	3.67
83 N2 12	4.00	0.00	2.00	3.25	3.00	4.00	2.58	3.83	3.67	3.58
83 N2 13	3.25	0.00	1.25	2.25	2.83	2.50	3.00	4.00	2.83	4.00
83 N2 14	2.92	1.75	2.83	1.42	1.83	2.33	1.25	1.42	1.75	1.58
83 N2 15	2.50	0.00	2.50	2.33	1.25	4.00	2.08	3.83	2.67	3.83
83 N2 16	3.50	0.50	1.83	2.42	2.67	3.17	1.67	3.17	2.92	3.75
83 N2 17	2.08	1.75	2.42	1.58	2.33	3.67	1.17	3.25	2.83	2.42
83 N2 18	2.75	0.00	1.67	2.33	2.42	2.50	2.25	3.33	3.33	3.83
83 N2 19	2.08	1.58	1.92	0.75	0.50	2.67	1.50	3.17	2.00	1.33
83 N2 20	1.83	0.00	2.75	2.50	2.50	2.42	1.17	4.00	3.08	1.33
83 N2 21	1.67	2.67	0.67	1.00	1.17	3.83	2.08	3.25	2.33	2.67
83 N2 22	2.50	1.17	1.00	1.42	2.00	3.08	2.67	3.00	3.08	3.67
83 N2 23	2.08	0.25	1.75	2.00	2.00	3.33	3.17	4.00	4.00	2.08
83 N2 24	1.75	1.58	1.42	1.33	1.83	1.25	0.33	0.75	1.58	0.92
83 N2 25	2.50	0.00	2.58	3.25	2.25	3.58	3.50	4.00	3.83	3.33
83 N2 26	2.33	1.17	2.25	2.50	3.00	3.50	3.08	4.00	4.00	3.25
83 N2 27	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
83 N2 28	3.83	0.38	3.25	2.50	2.75	3.33	3.50	3.50	2.92	3.08
83 N2 29	2.42	0.00	3.00	3.25	2.83	3.67	3.25	4.00	2.92	2.17
83 N2 30	3.08	0.00	2.17	2.50	2.42	3.67	3.33	2.42	3.67	3.50
83 N2 31	2.50	2.25	0.92	1.83	1.92	3.25	1.92	3.67	3.42	3.00
83 N2 32	2.75	0.00	1.83	2.92	2.58	3.42	3.25	2.92	2.00	3.58
83 N2 33	2.67	2.25	2.33	3.08	3.00	3.08	0.25	3.00	2.67	3.17
83 N2 34	1.92	0.13	1.83	2.67	2.17	3.33	3.25	3.58	2.83	3.58
83 N2 35	3.50	2.25	1.83	3.25	2.00	2.92	1.46	3.17	2.92	2.00
83 N2 36	2.92	1.75	0.92	3.25	1.33	2.08	1.25	3.67	2.58	2.08
83 N2 37	2.58	2.42	0.75	1.83	0.75	3.17	3.08	2.75	2.25	1.88
83 N2 38	2.33	0.38	2.58	2.17	2.58	2.58	2.17	3.08	3.17	2.92
83 N2 39	0.00	0.00	0.00	0.00	0.00	3.25	3.33	3.83	2.00	3.42
83 N2 40	2.83	2.42	0.75	2.67	1.42	3.17	2.25	3.17	3.33	3.42
83 N2 41	2.83	2.58	1.25	2.92	1.50	2.83	1.58	2.83	2.25	1.79
83 N2 42	2.08	0.00	1.83	2.00	2.33	1.79	1.33	3.08	2.67	2.50
83 N2 43	2.75	0.00	2.75	2.67	0.92	2.50	1.33	3.58	3.25	2.50
83 N2 44	2.75	1.83	1.83	2.58	1.83	3.67	1.63	2.25	2.67	1.92
83 N2 45	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
83 N2 46	2.83	0.00	2.25	2.75	2.67	2.83	2.83	3.58	2.67	2.46
83 N2 47	2.67	0.00	2.08	3.25	2.83	3.58	2.58	3.33	3.83	2.75

APPENDIX Table 7.5 continued (NVRS plants, NVRS isolates not tested)

Plant No Area N3 1983	Isolates G8	G9	G10	G11	G12
83 N3 01	2.92	2.25	2.67	2.92	1.50
83 N3 02	2.83	1.83	0.50	2.92	2.50
83 N3 03	3.42	2.25	2.17	2.50	2.50
83 N3 04	3.17	2.92	2.92	2.75	1.17
83 N3 05	3.83	2.67	1.67	3.58	3.58
83 N3 06	1.42	0.50	2.00	0.67	2.08
83 N3 07	0.00	2.75	0.00	1.08	2.08
83 N3 08	2.08	2.58	2.67	2.58	2.83
83 N3 09	3.33	2.67	3.50	2.42	3.33
83 N3 10	1.83	0.67	2.58	0.92	1.33
83 N3 11	0.00	0.00	0.00	0.00	0.00
83 N3 12	3.08	2.75	2.58	2.83	1.75
83 N3 13	3.67	1.50	1.75	3.08	2.25
83 N3 14	3.42	2.67	3.75	3.42	2.75
83 N3 15	1.08	3.67	2.17	2.00	2.58
83 N3 16	3.25	2.75	1.67	2.17	2.00
83 N3 17	2.75	2.00	3.00	3.33	2.67
83 N3 18	3.33	1.83	3.83	3.42	2.25
83 N3 19	3.83	1.92	2.42	4.00	2.75
83 N3 20	0.75	0.25	1.25	0.75	1.75
83 N3 21	0.00	0.00	0.00	0.00	0.00
83 N3 22	3.83	3.50	3.17	2.67	2.92
83 N3 23	2.25	1.92	2.67	0.92	2.58
83 N3 24	3.50	2.33	2.42	2.00	2.42
83 N3 25	2.92	2.83	2.58	2.58	3.00
83 N3 26	1.33	1.08	1.50	0.50	0.67
83 N3 27	4.00	4.00	3.83	3.75	3.25
83 N3 28	0.25	0.00	0.75	2.83	3.83
83 N3 29	3.17	2.92	3.08	3.50	3.33
83 N3 30	2.00	1.42	1.25	2.17	2.08
83 N3 31	3.17	1.08	1.25	2.17	2.58
83 N3 32	3.42	2.58	2.17	2.67	2.50
83 N3 33	3.00	1.67	2.25	2.67	2.67
83 N3 34	3.25	1.92	2.25	1.92	1.75
83 N3 35	0.50	2.42	2.58	1.67	2.17
83 N3 36	3.00	3.25	1.67	1.75	2.67
83 N3 37	3.17	2.75	2.92	2.75	2.17
83 N3 38	2.83	2.42	1.67	3.08	3.25
83 N3 39	1.33	2.33	2.92	3.17	3.25
83 N3 40	2.58	1.75	2.75	1.58	1.17

APPENDIX Table 7.5 continued (NVRS plants, NVRS isolates not tested)

Plant No Area N1 1983	Isolates G8	G9	G10	G11	G12
N1 01	3.33	0.00	3.75	0.67	1.83
N1 02	2.58	2.25	2.83	3.58	3.75
N1 03	1.58	1.96	1.58	1.67	1.83
N1 04	2.92	0.00	2.58	2.50	3.00
N1 05	2.75	2.54	0.92	1.25	1.33
N1 06	4.00	0.00	3.83	4.00	4.00
N1 07	3.83	0.00	3.25	2.67	3.08
N1 08	2.83	0.00	2.33	2.75	2.83
N1 09	2.58	0.00	2.25	3.17	3.25
N1 10	3.25	0.00	2.67	3.33	2.67
N1 11	3.33	0.00	3.33	3.83	2.92
N1 12	3.50	2.33	2.75	2.92	4.00
N1 13	3.33	2.33	2.25	3.17	2.58
N1 14	3.33	0.00	3.67	2.25	2.25
N1 15	0.00	0.21	0.00	1.00	1.00
N1 16	3.50	0.00	3.58	1.92	2.17
N1 17	3.17	0.25	3.33	2.83	2.75
N1 18	3.83	0.13	3.33	2.67	2.75
N1 19	2.08	1.08	3.33	2.92	2.58
N1 20	3.50	2.83	2.42	3.83	3.67
N1 21	3.33	0.00	3.75	1.92	3.08
N1 22	2.67	2.75	3.67	3.17	2.08
N1 23	1.42	0.28	1.25	0.92	0.75
N1 24	0.50	2.33	0.75	1.58	1.00
N1 25	2.92	0.33	3.17	2.75	2.83
N1 26	2.83	0.83	3.33	2.17	1.50
N1 27	3.50	0.00	3.42	2.83	3.83
N1 28	3.00	0.00	3.83	3.83	3.25
N1 29	2.92	0.00	3.42	3.00	4.00
N1 30	3.50	0.58	3.50	2.75	1.83
N1 31	3.33	1.75	2.92	1.42	2.67
N1 32	1.83	0.00	2.33	3.17	2.92
N1 33	2.75	0.00	2.67	2.17	3.17
N1 34	3.50	0.50	3.75	2.67	3.67
N1 35	0.75	0.00	3.17	1.58	3.33
N1 36	0.75	0.46	1.92	0.83	0.83
N1 37	3.67	1.75	3.83	3.08	2.33
N1 38	2.25	0.25	2.58	1.67	2.67
N1 39	2.58	2.25	3.83	2.50	2.67
N1 40	1.92	0.63	2.92	2.83	2.33
N1 41	2.92	0.00	3.17	1.83	2.08
N1 42	3.42	0.00	1.50	3.17	1.75
N1 43	3.17	0.00	3.17	2.08	2.25
N1 44	3.08	2.33	2.67	0.50	1.33
N1 45	2.42	1.00	2.25	1.75	2.67
N1 46	2.25	0.00	2.33	1.13	1.08
N1 47	1.50	0.00	2.58	3.00	3.33
N1 48	0.00	0.00	0.83	0.00	0.00
N1 49	2.58	0.25	2.67	1.25	2.50

APPENDIX Table 7.5 continued (NVRS plants, NVRS isolates not tested)

Plant No Area N1 1983	Isolates G8	G9	G10	G11	G12
N1 50	3.00	2.50	3.08	2.83	3.33
N1 51	3.00	0.50	2.83	3.00	3.50
N1 52	2.67	0.00	2.83	2.50	2.38
N1 53	2.58	0.33	2.75	0.92	3.08
N1 54	2.67	0.33	1.83	1.00	2.25
N1 55	4.00	3.00	3.50	4.00	1.92
N1 56	1.83	0.00	2.75	0.50	2.17
N1 57	3.08	0.00	3.38	1.75	1.33
N1 58	1.83	0.33	2.33	1.25	0.25
N1 59	1.75	0.00	2.50	2.17	2.67
N1 60	2.67	0.00	3.17	2.67	0.83
N1 61	3.42	0.00	2.50	3.08	3.08
N1 62	0.67	1.08	0.92	1.25	0.00
N1 63	0.83	1.67	1.92	1.25	1.75
N1 64	1.33	2.83	1.17	1.17	2.25
N1 65	2.00	2.08	3.00	1.08	2.08
N1 66	0.00	1.58	0.00	0.67	1.33
N1 67	1.25	0.75	0.75	0.42	2.33
N1 68	2.42	1.33	0.83	1.17	1.25
N1 69	1.50	3.50	1.67	3.33	1.83
N1 70	3.08	0.00	3.50	1.67	2.75
N1 71	2.83	0.00	3.33	3..17	2.50
N1 72	2.75	0.00	2.92	2.83	3.08
N1 73	3.67	3.58	4.00	2.08	4.00
N1 74	3.00	0.00	3.00	1.83	2.33
N1 75	2.33	0.00	2.58	1.00	1.75

APPENDIX Table 8.1 Maximum, Minimum temperatures and rainfall

Date 1983	Maximum Temperature ^{o}C	Minimum Temperature ^{o}C	Rainfall mm
10.5	10.1	03.3	02.7
11.5	11.4	06.4	02.6
12.5	13.5	06.8	03.4
13.5	11.2	07.1	11.9
14.5	13.2	07.5	00.8
15.5	14.5	07.8	TRACE
16.5	15.0	08.0	02.8
17.5	11.2	07.3	00.1
18.5	13.2	07.7	00.1
19.5	14.3	06.3	TRACE
20.5	15.8	06.0	07.3
21.5	13.8	07.1	01.8
22.5	12.7	04.8	00.5
23.5	16.2	-0.1	00.0
24.5	14.7	04.3	01.3
MEAN	13.4	06.0	02.4
Se	00.46	00.55	00.85
25.5	16.0	08.2	00.0
26.5	17.9	05.7	00.0
27.5	10.5	00.6	01.3
28.5	09.8	07.2	TRACE
29.5	11.0	07.7	TRACE
30.5	14.8	06.7	02.4
31.5	14.7	08.1	00.2
01.6	10.9	09.3	17.2
02.6	10.0	08.1	01.2
03.6	11.3	07.0	12.4
04.6	18.4	07.5	TRACE
05.6	14.3	08.7	00.0
06.6	17.0	02.5	00.0
07.6	19.1	06.6	00.2
MEAN	14.0	06.7	02.5
Se	00.90	06.64	01.4

APPENDIX Table 8.1 continued

Date 1983	Maximum Temperature °C	Minimum Temperature °C	Rainfall mm
08.6	19.2	12.0	02.5
09.6	14.8	11.0	TRACE
10.6	15.4	08.0	03.5
11.6	16.2	11.3	TRACE
12.6	15.7	08.4	00.4
13.6	14.9	06.1	08.1
14.6	13.8	08.5	01.0
15.6	15.8	06.3	02.5
16.6	13.9	06.1	01.1
17.6	17.3	09.4	00.0
18.6	21.0	08.9	00.0
19.6	24.7	07.4	00.0
20.6	22.2	12.5	TRACE
21.6	18.3	10.1	00.0
MEAN	17.4	09.0	01.4
Se	00.89	00.58	00.60
22.6	20.70	08.7	TRACE
23.6	13.60	11.8	00.0
24.6	18.70	09.7	00.0
25.6	19.8	07.1	TRACE
26.6	17.4	11.4	TRACE
27.6	17.5	09.2	00.1
28.6	14.6	08.9	02.8
29.6	16.3	09.4	00.0
30.6	19.7	09.7	TRACE
01.7	15.1	06.9	09.9
02.7	14.9	10.4	01.0
03.7	14.7	10.7	TRACE
04.7	16.5	10.8	00.9
05.7	22.7	13.9	TRACE
MEAN	17.3	09.9	01.1
Se	00.72	00.49	00.71

APPENDIX Table 8 continued

Date 1983	Maximum Temperature °C	Minimum Temperature °C	Rainfall mm
06.7	26.1	10.0	TRACE
07.7	23.4	14.5	TRACE
08.7	26.9	12.4	00.0
09.7	27.2	13.6	00.0
10.7	21.5	12.9	00.0
11.7	27.9	13.7	00.0
12.7	30.1	14.1	00.0
13.7	26.7	13.7	00.0
14.7	21.9	13.4	TRACE
15.7	18.8	14.3	00.2
16.7	19.8	05.5	00.0
17.7	21.9	10.8	TRACE
18.7	18.5	12.0	TRACE
19.7	18.9	09.2	00.0
20.7	18.9	11.0	00.0
21.7	24.6	09.6	00.0
22.7	27.8	07.9	TRACE
23.7	24.7	14.4	01.2
24.7	22.4	14.4	02.8
25.7	21.6	15.0	03.8
26.7	20.2	15.0	TRACE
27.7	23.1	12.1	00.2
28.7	20.7	12.3	TRACE
29.7	20.8	14.2	TRACE
30.7	19.4	09.8	01.2
31.7	17.9	13.5	00.0
01.8	17.9	06.4	00.1
02.8	18.7	04.5	TRACE
03.8	17.7	08.1	03.7
04.8	18.9	11.7	00.0
MEAN	22.2	11.7	00.5
Se	00.66	00.53	00.19

For calculation of mean
Trace = 0.1

RERERENCES

ALLARD, R.W. (1970). 'Population structure and sampling methods'. In "Genetic resources in plants". Ed. FRANKEL, O.H. and BENNET, E. IPB Book 11. Blackwell Scientific Publications, Oxford and Edinburgh. p 97-107.

ANDREWS, J.H., BERBEE, F.M. and NORDHEIM, E.V. (1983). 'Microbial antagonism to the imperfect stage of the apple scab pathogen Venturia inaequalis.' PHYTOPATHOLOGY ,73, 228-234.

ASHER, M.J.C. and THOMAS, C.E. (1983). 'The expreesion of partial resistance to Erysiphe graminis in spring barley. PLANT PATHOLOGY ,32, 79-89.

BARRETT, J. (1985). 'The gene-for-gene hypothesis: parable or paradigm.' In "Ecology and genetics of host-parasite interactions." Eds ROLLINSON, D. and ANDERSON, R.M. Academic Press.

BEN-KALIO, V.D. (1976). 'Effects of powdery mildew (Erysiphe cichoracearum D.C. Ex Merat) on the growth and development of groundsel (Senecio vulgaris L.).' Ph.D Thesis. Department of Botany, University of Glasgow.

BEN-KALIO, V.D. and CLARKE, D.D. (1979). 'Studies on tolerance in wild plants: Effects of Erysiphe fischeri on the growth and development of Senecio vulgaris.' PHYSIOLOGICAL PLANT PATHOLOGY ,14, 203-211.

BENNETT, F.G.A. (1981a). 'The expression of resistance to powdery mildew infection in winter wheat cultivars. I. Seedling resistance.' ANNALS OF APPLIED BIOLOGY ,98, 295-303.

BENNETT, F.G.A. (1981b). 'The expression of resistance to powdery mildew infection in winter wheat cultivars. II. Adult plant resistance.' ANNALS OF APPLIED BIOLOGY ,98, 305-317.

BENNETT, F.G.A. and VAN KINTS, T.M.C. (1983). 'Powdery mildew of wheat.' ANNUAL REPORT OF THE PLANT BREEDING INSTITUTE FOR 1982. p90-92.

BERGER, R.D. (1975). 'Disease incidence and infection rates of Cercospora apii in plant spacing plots.' PHYTOPATHOLOGY ,65, 485-487.

BETTENCOURT, A.J. and NORONHA-WAGNER, M. (1967). 'Genetic study of the resistance of Coffea spp to leaf rust.I. Identification and behaviour of four factors conditioning disease reaction in Coffea arabica to twelve physiologic races of Hemileia vastatrix.' CANADIAN JOURNAL OF BOTANY ,45, 2021-2031.

BIFFEN, R.H. (1905). 'Mendel's laws of inheritance and wheat breeding.' JOURNAL OF AGRICULTURAL SCIENCE ,1, 4-48.

BIFFEN, R.H. (1912). 'Studies on the inheritance of disease resistance. II.' JOURNAL OF AGRICULTURAL SCIENCE ,4, 421-429.

BLAKEMAN, J.P. and FOKKEMMA, N.J. (1982). 'Potential for biological control of plant diseases on the phylloplane.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,20, 167-192.

BLOWES, W.M., HEATHER, W.A., MALAJCZUK,N. and SHEA, S.R. (1982). 'The distribution of Phytophthora cinnamoni Rands at two sites in south western Australia and at Durras in south-eastern New South Wales.' AUSTRALIAN JOURNAL OF BOTANY ,30, 139-145.

BORLAUG, N.E. (1965). 'Wheat rust and people.' PHYTOPATHOLOGY ,55, 1088-1098.

BOUGHEY, A.S. (1949). 'The ecology of fungi which cause economic plant diseases.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,32, 179-189.

BRIGGLE, L.W and SCHAREN, A.L. (1961). 'Resistance in Triticum vulgare to infection by Erysiphe graminis f.sp. tritici as influenced by the stage of development of the host plant.' PLANT DISEASE REPORTER ,45, 846-850.

BROWDER, L,E. (1966). 'A rapid method of assaying pathogenic potential of populations of Puccinia graminis tritici.' PLANT DISEASE REPORTER ,50, 673-676.

BROWNING, J.A. (1974). 'Relevance of knowledge about natural ecosystems to development of pest management programs for agro-ecosystems.' PROCEEDINGS OF THE AMERICAN PHTOPATHOLOGICAL SOCIETY ,1, 191-199.

BURDON, J.J. (1978). 'Mechanisms of disease control in heterogeneous plant populations - an ecologists view.' In "Plant Disease Epidemiology". Ed. SCOTT, P.R. and BAINBRIDGE, A. Blackwell Scientific publications, Oxford and Edinburgh. p193-200.

BURDON, J.J. (1980a). 'Intra-specific diversity in a natural population of Trifolium repens.' JOURNAL OF ECOLOGY ,68, 717-735.

BURDON, J.J. (1980b). 'Variation in disease resistance within a population of Trifolium repens.' JOURNAL OF ECOLOGY ,68, 737-744.

BURDON, J.J. (1982a). 'The effect of fungal pathogens on plant communities.' In "The Plant Community as a Working Mechanism." Ed. NEWMAN, E.I. Special publication number 1 of the British Ecological Society. Blackwell Scientific Publications, Oxford. p99-112.

BURDON, J.J. (1982b). Host density as a factor in plant disease ecology.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,20, 143-166.

BURDON, J.J. and CHILVERS, G.A. (1974). 'Fungal and insect parasites contributing to niche differentiation in mixed species stands of eucalypt saplings.' AUSTRALIAN JOURNAL OF BOTANY ,22, 103-114.

BURDON, J.J. and CHILVERS, G.A. (1975). 'Epidemiology of damping-off disease (Pythium irregulare) in relation to density of Lepidium sativum seedlings.' ANNALS OF APPLIED BIOLOGY ,81, 135-143.

BURDON, J.J. and CHILVERS, G.A. (1976). 'Controlled environment experiments on epidemics of barley mildew in different density host stands.' OECOLOGIA (Berlin) ,26, 61-72.

BURDON, J.J. and CHILVERS, G.A. (1982). 'Host density as a factor in plant disease ecology.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,20, 143-166.

BURDON, J.J. and MARSHALL, D.R. (1981). 'Inter and Intra-specific diversity in the disease of Glycine species to the leaf rust fungus Phakopsora pachyrhizi.' JOURNAL OF ECOLOGY ,69, 381-390.

BUTT, D.J. (1978). 'Epidemiology of powdery mildews.' In "The Powdery Mildews." Ed. SPENCER, D.M. Academic Press. London, New York, San Francisco.

BUZACKI, S.T. and OCKENDON, J.G. (1979). 'Preliminary observations on variation in susceptibility to clubroot among collections of some wild crucifers. ANNALS OF APPLIED BIOLOGY ,92, 113-118.

CAMPELL, J.M. and ABBOTT, R.J. (1976). 'Variability of outcrossing frequency in Senecio vulgaris L.' HEREDITY ,36(2), 267-274.

CARVER, T.L.W. and BUSHNELL, W.R. (1983). 'The probable role of primary germ tubes in water uptake before infection by Erysiphe graminis.' PHYSIOLOGICAL PLANT PATHOLOGY ,23, 229-240.

CARVER, T.L.W. and CARR, A.J.H. (1977). 'Race non-specific resistance of oats to primary infection by mildew.' ANNALS OF APPLIED BIOLOGY ,86, 29-36.

CARVER, T.L.W. and CARR, A.J.H. (1978). 'Effects of host resistance on the development of haustoria and colonies of oat mildew.' ANNALS OF APPLIED BIOLOGY ,88, 171-178.

CHEREWICK, W.J. (1944). 'Studies on the biology of Erysiphe graminis D.C.' CANADIAN JOURNAL OF RESEARCH.C. ,22, 52-86.

CHIN, K.M. and WOLFE, M.S. (1984a). 'The spread of Erysiphe graminis f.sp. hordei in mixtures of barley varieties.' PLANT PATHOLOGY ,33, 89-100.

CHIN, K.M. and WOLFE, M.S. (1984b). 'Selection on Erysiphe graminis in pure and mixed stands of barley.' PLANT PATHOLOGY ,33, 535-546.

CHIPPINDALE, H.G. and MILTON, W.E.J. (1934). 'On the variable seeds present in the soil beneath pasture.' JOURNAL OF ECOLOGY ,22, 508-531.

CLAPHAM, A.R., TUTIN, T.G. and WARBURG, E.F. (1962) "Flora of the British Isles." 2nd Edition. Cambridge University Press.

CLARKE, D.D. (1986). 'Tolerance of parasites and disease in plants and its significance in host-parasite interactions.' ADVANCES IN PLANT PATHOLOGY ,5, (In Press).

CLIFFORD, B.C. (1974). 'Relation between compatible and incompatible infection sites of Puccinia hordei on barley.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,63, 215-220.

CLIFFORD, B.C. and CLOTHIER, R.B. (1974). 'Physiologic specialization of Puccinia hordei on barley hosts with non hypersensitive resistance.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,63, 421-430.

CRUTE, I.R. and JOHNSON, A.G. (1976). 'The genetic relationship between races of Bremia lactucae and cultivars of Lactuca sativa.'

ANNALS OF APPLIED BIOLOGY ,83, 125-137.

CRUTE, I.R. and NORWOOD, J.M. (1978). 'Incomplete specific resistance to Bremia lactucae in lettuce.' ANNALS OF APPLIED BIOLOGY ,89, 467-474.

CRUTE, I.R., NORWOOD, J.M. and GORDON, P.L. (1980). NATIONAL VEGETABLE RESEARCH STATION ANNUAL REPORT. p77-79.

CRUTE, I.R., PHELPS, K., BARNES, A., BUCZACKI, S.T. and CRISP, P. 'The relationship between genotypes of three Brassica species and collections of Plasmodiophora brassicae.' PLANT PATHOLOGY ,32, 405-420.

CRUTE, I.R., DE WIT, P.J.G.M. and WADE, W. (1985). 'Mechanisms by which genetically controlled resistance and virulence influence host colonization by fungal and bacterial parasites.' In "Mechanisms of resistance to plant diseases." Ed FRASER, R.S.S. Martinus Nijhoff/ Dr W.Junk Publishers, Dordrecht. p197-309.

CRISP, P.C. and JONES, B.M.G. (1970). 'Senecio squalidus L., S.vulgaris L. and S.cambrevis Rosser.' WATSONIA ,8, 47-48.

DINOOR, A. (1970). 'Sources of oat crown rust resistance in hexaploid and tetraploid wild oats in Israel.' CANADIAN JOURNAL OF BOTANY ,48, 153-161.

DINOOR, A. (1977). 'Oat crown rust resistance in Israel'. ANNALS OF THE NEW YORK ACADEMY OF SCIENCES ,287, 357-366.

DINOOR, A. and ESHED, N. (1984). 'The role and importance of pathogens in natural plant communities.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,22, 443-466.

DRANDAREVSKI, C.A. (1978). 'Powdery mildews of beet crops'. In "The Powdery Mildews." Ed. SPENCER, D.M. Academic Press, London. p323-345.

DYCK, P.L. and JOHNSON, R. (1983). 'Temperature sensitivity of genes for resistance in wheat to Puccinia recondita.' CANADIAN JOURNAL OF PLANT PATHOLOGY ,5, 229-234.

EENINK, A.H. and DIELEMAN, F.L. (1980). 'Development of Myzus persicae on a partially resistant and on a susceptible genotype of lettuce (Lactuca sativa) in relation to plant age.' NETHERLANDS JOURNAL OF PLANT PATHOLOGY ,86, 111-116.

EENINK, A.H. and DIELEMAN, F.L. (1982). 'Resistance of Lactuca accessions to leaf aphids: components of resistance and exploitation of wild Lactuca species as sources of resistance.' In "Proceedings of the 5th International Symposium on insect-plant relationships." Eds. VISSER, J.H. and MINKS, A.K. Wageningen Centre for Agricultural Publishing and Documentation. p349-355.

ELLINGBOE, A.H. (1972). 'Genetics and physiology of primary infection by Erysiphe graminis.' PHYTOPATHOLOGY ,62, 401-406.

ELLINGBOE, A.H. (1982). 'Genetic aspects of active defence.' In "Active defence mechanisms in plants." Ed. WOOD, R.K.S. Plenum Press, New York, London. p179-192.

ESKES, A.B. (1983). 'Incomplete resistance to coffee leaf rust.' In "Durable resistance in crops." Ed LAMBERTI, F., WALLER, J.M. and VAN DER GRAFF, N.A. Plenum Press, New York and London.

EYAL, Z., YURMAN, R., MOSEMAN, J.G. and WAHL, I. (1973). 'Use of mobile nurseries in pathogenicity studies of Erysiphe graminis hordei on Hordeum spontaneum.' PHYTOPATHOLOGY ,63, 1330-1334.

FLOR, H.H. (1935). 'Physiologic specialization of Melampsora lini on Linum usitatissimum.' JOURNAL OF AGRICULTURAL RESEARCH ,51, 819-837.

FLOR, H.H. (1942). 'Inheritance of pathogenicity in a cross between physiologic races 22 and 24 of Melampsora lini.' PHYTOPATHOLOGY ,32, 5. (Abstract).

FLOR, H.H. (1946). 'Genetics of pathogenicity in Melampsora lini.' JOURNAL OF AGRICULTURAL RESEARCH ,73, 335-357.

FLOR, H.H. (1947). 'Inheritance of reaction to rust in flax.' JOURNAL OF AGRICULTURAL RESEARCH , 74, 241-262.

FLOR, H.H. (1954). 'Identification of race of flax rust by lines with single rust-conditioning genes.' U.S. DEPARTMENT OF AGRICULTURE TECHNICAL BULLETIN. 1087, 1-25.

FLOR, H.H. (1956). 'The complementary genetic system in flax and flax rust.' ADVANCES IN GENETICS ,8, 29-54

FUTRELL, M.C. and DICKSON, J.G. (1954). 'The influence of temperature on the development of powdery mildew on spring wheats.' PHYTOPATHOLOGY ,44, 247-251.

FRASER, R.S.S. (1985). 'Some basic concepts and definitions in resistance studies.' In "Mechanisms of resistance to plant diseases." Ed FRASER R.S.S. Martinus Nijhoff/ Dr W. Junk Publishers. Dordrecht. p1-12.

FRIED, P.M., MacKENZIE and NELSON, R.R. (1979). 'Disease progress curves of Erysiphe graminis f.sp. tritici on Chancellor wheat and four multi-lines.' PHYTOPATHOLOGISCHE ZEITSCHRIFT ,95, 151-166.

FRITSCH, F.E. and SALISBURY, E.J. (1938). Plant form and function. G. Bell and Sons Ltd, London.

GREGORY, P.H. (1973). "The microbiology of the atmosphere." Aylesbury: Leonard Hill. 2nd Edition. pp377.

HALSHALL, D.M. (1982). A forest soil suppressive to Phytophthora cinnamomi and conducive to Phytophthora cryptogea I. Survival, germination and infectivity of mycellium and chlamydospores.' AUSTRALIAN JOURNAL OF BOTANY ,30, 11-25.

HARLAN, J.R. (1976). 'Diseases as a factor in plant evolution.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,14, 31-51.

HARPER, J.L. (1977). 'Pathogens.' In "Population biology of plants." Ed. HARPER, J.L. Academic press. London, New York. p483-496.

HARRY, I.B. (1980). 'Studies on the Senecio vulgaris L /Erysiphe

fischeri Blumer, plant pathosystem.' Ph.D. Thesis. Department of Botany. University of Glasgow.

HARRY, I.B. and CLARKE, D.D. (1986). 'Race specific resistance in groundsel (Senecio vulgaris) to the powdery mildew Erysiphe fischeri.' NEW PHYTOLOGIST, 103, (In Press).

HASKELL, G. (1953). 'Adaptation and the breeding system in groundsel.' GENETICA ,26,468-484.

HEATHER, W.A. and CHANDRASHEKAR, M. (1982a). 'Evolutionary, epidemiological and ecological implications of forms of resistance in Populus spp to Melamspora leaf rust.' AUSTRALIAN FOREST RESEARCH ,12, 231-244.

HEATHER, W.A. and CHANDRASHEKA, M. (1982b). 'Stability of disease resistance in natural and agroecosystems.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,78, 381-383.

HERMANSEN, J.E. and WIBERG, A. (1972). 'On the appearance of Erysiphe graminis f.sp hordei and Puccinia hordei in the Faeroes and the possible primary sources of inoculum.' FRIESIA ,10, 30-34.

HIRST, J.M., STEDMAN, O.J. and HURST, G.W. (1967). 'Long distance spore transport: vertical sections of spore clouds over the sea.' JOURNAL OF GENERAL MICROBIOLOGY ,48, 357-377.

HOLMES, F.W. (1962). 'Recorded dutch elm disease distribution in north America as of 1961.' PLANT DISEASE REPORTER ,46, 715-718.

HULL, P. (1974a). 'Self fertilization and the distribution of the radiate form of Senecio vulgaris l. in central Scotland.' WATSONIA ,10, 69-75.

HYDE, P.M. (1982). 'Temperature-sensitive resistance of the wheat cultivar Maris Fundin to Puccinia recondita.' PLANT PATHOLOGY ,31, 25-30.

INGOLD, C.T. (1978). 'Dispersal of micro-organisms.' In "Plant disease epidemiology." Ed SCOTT, P.R. and BAINBRIDGE, A. Blackwell Scientific publications, Oxford.

INGRAM, R. (1977). 'Synthesis of the hybrid Senecio squalidus L. x S.vulgaris l.f. radicitus Hegi.' HEREDITY ,39, 171-173.

INGRAM, R., WIER, J. and ABBOT, R.J. (1980). 'New evidence concerning the origin of inland radiate groundsel Senecio vulgaris L. var Hibernicus Syme.' NEW PHYTOLOGIST ,84, 543-546.

JAIN, S.K. (1975). 'Population structure and the effects of breeding system.' In "Crop genetic resources for today and tommorrow." Eds. FRANKEL, O.H. and HAWKES, J.G. Cambridge University Press, Cambrige. p15-36. pp492

JEGER, M.J. and GROTH, J.V. (1985). 'Resistance and pathogenicity : Epidemiological and ecological mechanisms.' In "Mechanisms of resistance to plant disease." Ed. FRASER, R.S.S. Martinus Nijoff/Dr W.Junk Publishers, Dordrecht, Boston, Lancaster. p310-372.

JENKYN, J.F., HURST, J.M. and KING, G. (1973). 'An apparatus for the isolated propagation of foliar pathogens and their hosts.' ANNALS OF APPLIED BIOLOGY ,73, 9-13.

JEPSON, P.C. (1982). 'The movements of apterous Myzus persicae on sugar beet.' In "Proceedings of the 5th International Symposium on insect-plant relationships." Eds VISSER, J.H. and MINKS, A.K. Vageningen Centre for Agricultural Publishing and Documentation. p415-416.

JOHNSON,R. (1979). 'The concept of durable resistance.' PHYTOPATHOLOGY ,69, 198-199. Letter to the editor.

JOHNSON, R. (1983). 'Genetic background of durable resistance.' In "Durable resistance in crops." Ed LAMBERTI, F., WALLER, J.M. and VAN DER GRAFF, M.A. Plenum Press, London and New york. p5-26.

JOHNSON, R. and LAW, C.N. (1967). 'A genetic study of leaf rust resistance in wheat.' CANADIAN JOURNAL OF GENETICS AND CYTOLOGY ,9, 805-822.

JOHNSON, L.E.B., BUSHNELL, W.R. and ZEYEN, R.J. (1979). Binary pathways for analysis of primary infection and host responses in populations of powdery mildew fungi.' CANADIAN JOURNAL OF BOTANY ,57, 497-511.

JOHNSON, R. and TAYLOR, A.J. (1972). 'Isolates of Puccinia striiformis collected in England from the wheat varieties Maris Beacon and Joss Cambier.' NATURE, LOND, 238, 105-106.

JOHNSON, L.E.B., BUSHNELL, W.R. and ZEYEN, R.J. (1982). 'Defence patterns in nonhost higher plant species against two powdery mildew fungi.I. Monocotyledonous species.' CANADIAN JOURNAL OF BOTANY ,60, 1068-1083.

JONES, I.T. (1975). 'The preconditioning effect of daylength and light intensity and adult plant resistance to powdery mildew in oats.' ANNALS OF APPLIED BIOLOGY ,80, 301-309.

JONES,I.T. (1978). 'Components of adult plant resistance to powdery mildew (Erysiphe graminis f.sp. avenae) in oats.' ANNALS OF APPLIED BIOLOGY ,90, 233-239.

KRAMMER, Th., GILDEMACHER, B.H., VAN DER STER, M. and PARLEVLIET, J.E. (1980). 'Tolerance of spring barley cultivars to leaf rust, Puccinia hordei.' EUPHYTICA ,29, 209-216.

KRANZ, J. (1968a). 'Eine Analayze von annuellen Epidemien pilzlicher Parasiten. I. Die Befallskurven und ihre Abhängigkeit von eingen Umweltfaktoren.' PHYTOPATHOLOGISCHE ZEITSCHRIFT ,61, 59-86.

KRANZ, J. (1968b). 'Eine Analayze von annuellen Epidemien pilzlicher Parasiten. II. Qualitative und Quantitative Merkmale von Befallskurven.' PHYTOPATHOGISCHE ZEITSCHRIFT ,61, 171-190.

KULKARNI, R.N. and CHOPRA, V.L. (1982). 'Environment as the cause of differential interaction between host cultivars and pathogenic races.' PHYTOPATHOLOGY ,72, 1384-1386. Letter to the editor.

KUNOH, H. and ISHIZAKI, H. (1976). 'Accumulation of chemical elements around the penetration sites of Erysiphe graminis hordei on barley leaf epidermis : (111) micromanipulation and X-ray microanalysis.' PHYSIOLOGICAL PLANT PATHOLOGY ,8, 91-96.

KUNOH, H., ISHIZAKI, H. and NAKAYA, K. (1977). 'Cytological studies of early stages of powdery mildew in barley and wheat leaves :II. Significance of the primary germ tube of Erysiphe graminis on barley leaves.' PHYSIOLOGICAL PLANT PATHOLOGY ,10, 191-199.

LAST, F.T. (1954). 'The effect of time of application of nitrogenous fertilizer on powdery mildew of winter wheat.' ANNALS OF APPLIED BIOLOGY ,41, 381-392.

LAST, F.T. (1962). 'Analysis of the effects of Erysiphe graminis on the growth of barley.' ANNALS OF BOTANY ,26, 279-289.

LAST, F.T. (1963). 'Effect of temperature on cereal powdery mildews.' PLANT PATHOLOGY ,12, 132-133.

LAW, C.N. and JOHNSON, R. (1967). 'A genetic study of leaf rust resistance in wheat.' CANADIAN JOURNAL OF GENETICS AND CYTOLOGY ,9, 805-822.

LEBEDA, A. (1984). 'Screening of wild Cucumis species for resistance to cucumber powdery mildew (Erysiphe cichoracearum and Sphaerotheca fuliginea).' SCIENTIA HORTICULTURAE ,24, 241-249.

LEWELLEN, R.T., SHARP, E.L. and HEHN, E.R. (1967). 'Major and minor genes in wheat for resistance to Puccinia striiformis and their responses to temperature changes.' CANADIAN JOURNAL OF BOTANY ,45, 2155-2172.

LEWIS, T. and TAYLOR, L.R. (1967). 'Introduction to experimental ecology.' Academic press Inc (London) Ltd. p93-107.

LIN, M.R. and EDWARDS, H.H. (1974). 'Primary penetration process in powdery mildewed barley related to host cell age, cell type and occurrence of basic staining material.' NEW PHYTOLOGIST, 73, 131-137.

LOVELESS, M.D. and HAMRICK, J.L. (1984). 'Ecological determinants of genetic structure in plant populations.' ANNUAL REVIEW OF ECOLOGY AND SYSTEMATICS ,15, 65-95.

LUKE, H.H., CHAPMAN, W.H. and BARNET, R.D. (1972). 'Horizontal resistance of red rustproof oats to crown rust.' PHYTOPATHOLOGY ,62, 414-417.

MARSHALL, D.F. Written communication. Department of Genetics, The University of Birmingham, P.O. Box 363, Birmingham.

MARSHALL, D.F. and ABBOTT, R.J. (1980). 'On the frequency of introgression of the radiate (Tr) allele from Senecio squalidus L. into Senecio vulgaris L.' HEREDITY ,45, 133-135.

MARSHALL, D.R. (1977). 'The advantages and hazards of genetic homogeneity.' ANNALS OF THE NEW YORK ACADEMY OF SCIENCES ,287, 1-19.

MARSHALL, D.R. and BROWN, A.H.D. (1975). 'Optimum sampling strategies in genetic conservation.' In "Crop genetic resources for today and tomorrow." Eds. FRANKEL, O.H. and HAWKES, A.H.D. Cambridge University Press, Cambridge. p53-80.

MARSHALL, I.D. and FENNER, F. (1958). 'Studies on the epidemiology of infectious Myxomatosis in rabbits. V. Changes in the innate resistance of Australian wild rabbits exposed to Myxomatosis.' JOURNAL OF HYGIENE ,56, 288-302.

MARTIN, S.B., ABAWI, G.S. and HOCH, H.C. (1985) 'Biological control of soil bourne pathogens with antagonists.' In "Biological control in agricultural IPM systems." Ed HOY, M.A. and HERZOG, D.C. Academic Press Inc (London) Ltd. p433-454.

MASRI, S.S. and ELLINGBOE, A.H. (1966). 'Primary infection of wheat and barley by Erysiphe graminis.' PHYTOPATHOLOGY ,56, 389-395.

MATTA, A and KERLING, L.C.P. (1964). Verticillium albo-atrum as a parasite of Senecio vulgaris.' NETHERLANDS JOURNAL OF PLANT PATHOLOGY ,70, 27-32.

MAY, R.M. and ANDERSON, R.M. (1983). 'Epidemiology and genetics in the coevolution of parasites and hosts.' PROCEEDINGS OF THE ROYAL SOCIETY OF LONDON ,B 219, 281-313.

MONAGHAN, J.L. and HULL, P. (1975). 'Difference in vegetative characteristics amoung four populations of Senecio vulgaris L. possibly due to interspecific hybridization.' ANNALS OF BOTANY ,40, 125-128.

MOSEMAN, J.G. (1956). 'Physiologic races of Erysiphe graminis f.sp. hordei in North America.' PHYTOPATHOLOGY ,46, 318-322.

MOSEMAN, J.G. (1957). 'Host-parasite interactions between culture 12A1 of the powdery mildew fungus and the M/k and M/g genes in barley.' PHYTOPATHOLOGY ,47, 453. (Abstract).

McCOY, M.S. and ELLINGBOE, A.H. (1966). 'Major genes for resistance and the formation of secondary hyphae by Erysiphe graminis f.sp. hordei.' PHYTOPATHOLOGY ,56, 683-686.

McKENZIE, E.H.C. and HUDSON, H.J. (1976). 'Mycoflora of rust infected and non-infected plant material during decay.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,66, 223-238.

NAIR, K.R.S. and ELLINGBOE, A.H. (1962). 'A method of controlled inoculations with conidiospores of Erysiphe graminis var. tritici.' PHYTOPATHOLOGY ,52, 714.

NELSON, R.R. (1978). 'Genetics of horizontal resistance to plant diseases.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,16, 359-378.

NIKS, R.E. (1983). 'Haustorium formation by Puccinia hordei in leaves of hypersensitive, partially resistant, and non-host plant genotypes.' PHYTOPATHOLOGY ,73, 64-66.

NIKS, R.E and KUIP , H.J. (1983). 'Histology of the relation between minor and major genes for resistance of barley to leaf rust.' PHYTOPATHOLOGY ,73, 55-59.

NORWOOD, J.M. and CRUTE, I.R. (1984). 'The genetic control and expression of specificity in Bremia lactucae (lettuce downy mildew).' PLANT PATHOLOGY , 33, 385-400.

PADMANABHAN, S.Y. (1973). 'The great Bengal famine.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,11, 11-26.

PALMBLAD, I.G. (1965). Chromosome numbers in Senecio (Compositae). I. CANADIAN JOURNAL OF BOTANY ,43, 715-721.

PAPAVIZAS, G.C. and LUMSDEN, R.D. (1980). 'Biological control of soil borne fungal propagules.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,18, 389-413.

PARKER, R.E. (1973). 'Introductory statistics for biology.' 2nd Edition. Edward Arnold (Publishers) Ltd. London.

PARLEVLIET, J.E. (1977). 'Plant pathosystems: An attempt to elucidate horizontal resistance.' EUPHYTICA, 553-556.

PARLEVLIET, J.E. (1979). 'Components of resistance that reduce the rate of epidemic development.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,17, 203-222.

PARLEVLIET, J.E. (1981). 'Race-non-specific disease resistance.' In "Strategies for the control of cereal diseases." Ed JENKYN, J.F. and PLUMB, R.T. Blackwell Scientific Publications, Oxford. p47-54.

PARLEVLIET, J.E. and ZADOKS , J.C. (1977). 'The integrated concept of disease resistance; a new view including horizontal and vertical resistance in plants.' EUPHYTICA ,26, 5-21.

PAUL, N.D. and AYRES, P.G. (1984). 'Effects of rust and post-infection drought on photosynthesis growth and weather relations in groundsel.' PLANT PATHOLOGY ,33, 561-569.

PERSON, C.O. (1959). 'Gene-for-gene relationships in host-parasite systems.' CANADIAN JOURNAL OF BOTANY ,37, 1101-1130.

POPAY, A.I. and ROBERTS, E.H. (1970). 'Factors involved in the

dormancy and germination of Capsella bursa-pastoris (L.) Medik and Senecio vulgaris L.' JOURNAL OF ECOLOGY ,58, 103-122.

PRATT, B.H. and HEATHER, W.A. (1973). 'The origin and distribution of Phytophthora cinnamomi Rands in Australian native plant communities and the significance of its association with particular plant species.' AUSTRALIAN JOURNAL OF BIOLOGICAL SCIENCE ,26, 559-573.

PRICE, P.W. (1980). "Evolutionary biology of parasites." Princeton University Press, New Jersey. pp237.

PRIESTLEY, R.H. (1978), 'Detection of increased virulence in populations of wheat yellow rust.' In "Plant disease epidemiology." Eds. SCOTT, P.R. and BAINBRIDGE, A. Blackwell Scientific Publications, Oxford. p63-70.

QUALSET, C.O. (1975). 'Sampling germplasm in a centre of diversity : an example of disease resistance in Ethiopian barley.' In "Crop genetic resources for today and tomorrow." Eds. FRANKEL, O.H. and HAWKES, J.G. Cambridge University Press, Cambridge. p81-96.

RAJARAM, S., LUIG, N.H. and WATSON, I.A. (1971). 'The inheritance of leaf rust resistance in four varieties of common wheat.' EUPHYTICA ,20, 574-585.

ROBERTS, J.J. and CALDWELL, R.M. (1970). 'General resistance (slow mildewing) to Erysiphe graminis f.sp. tritici in Knox wheat.' PHYTOPATHOLOGY ,60, 1310 (Abstract).

ROBINSON, R.A. (1976). "Plant pathosystems." Springer-Verlag, Berlin, Heidelberg, New York. pp184.

ROBINSON, R.A. (1982a). 'Theoretical resistance models.' In "Durable resistance in crops." Eds. LAMBERTI, F., WALLER, J.M. and VAN DER GRAAFF, N.A. Series A: Life Sciences, Vol 55. NATO Scientific affairs division. Plenum Press, New York, London. p45-55.

ROBINSON, R.A. (1982b). 'Pathosystem management.' In "Durable resistance in crops." Eds LAMBERTI, F., WALLER, J.M. and VAN DER GRAFF, N.A. Plenum Press, New York, London.

ROELFS, A.P. and GROTH, J.V. (1980). 'A comparison of virulence phenotypes in wheat stem rust populations reproducing sexually and asexually.' PHYTOPATHOLOGY ,70, 855-862.

ROYLE, D.J. (1978). 'Powdery mildew of hop.' In "The powdery mildews." Ed. SPENCER, D.M. Academic Press. London. p381-409.

SALISBURY, E.J. (1961). "Weeds and aliens." Collins, St James Place London p182 pp384.

SALISBURY, E.J. (1943). 'The flora of bombed areas.' NATURE ,151, 462-466.

SCHAFFER, J.F. (1971). 'Tolerance to plant disease.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,9, 235-252.

SCHNATHORST, W.C. (1959). 'Resistance in lettuce to powdery mildew, related to osmotic value.' PHYTOPATHOLOGY ,49, 562-571.

SCHWARZBACH, E. (1979). 'A high throughput jet trap for collecting mildew spores on living leaves.' PHYTOPATHOLOGISCHE ZEITSCHRIFT ,94, 165-171.

SCOTT, P.R. and HOLLINS, T.W. (1977). Interactions between cultivars of wheat and isolates of Cercosporella herpotrichoides.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,69, 397-403.

SCOTT, P.R., JOHNSON, R., WOLFE, M.S., LOWE, H.J.B. and BENNETT, F.G.A. 'Host specificity in cereal parasites in relation to their control.' APPLIED BIOLOGY ,5, 349-393.

SEGAL, A., MANISTERSKI, J., FISHBECK, G. and WAHL, I. (1980). 'How plant populations defend themselves in natural ecosystems.' In "Plant disease: An advanced treatise." V. Eds. HORSFALL, J.G. and COWLING, E.B. Academic Press, London, New York.

SEWELL, G.W.F. (1981). 'Soil-borne fungal pathogens in natural vegetation and weeds of cultivation.' In "Pests, pathogens and vegetation." Ed THRESH, J.M. Pitman, London. p175-190.

SHANER, G. (1973a). 'Evaluation of slow-mildewing resistance in Knox wheat in the field.' PHYTOPATHOLOGY ,63, 867-872.

SHANER,G. (1973b). 'Reduced infectability and inoculum production as factors of slow mildewing in Knox wheat.' PHYTOPATHOLOGY ,63, 1307-1311.

SIMONS, M.D. (1954). 'The relationship of temperature and stage of growth to the crown rust reaction of certain varieties of oat.' PHYTOPATHOLOGY ,44, 221-223.

SITTERLY, W.R. (1978). 'Powdery mildews of cucurbits.' In "The powdery mildews." Ed. SPENCER, D.M. Academic Press, London. p359-379.

SLESINSKI, R.S. and ELLINGBOE, A.H. (1969). 'The genetic control of primary infection of wheat by Erysiphe graminis f.sp. tritici.' PHYTOPATHOLOGY ,59, 1833-1837.

SLESINSKI, R.S. and ELLINGBOE, A.H. (1970). 'Gene-for-gene interactions during primary infection of wheat by Erysiphe graminis f.sp. tritici.' PHYTOPATHOLOGY ,60, 1068-1070.

SLESINSKI, R.S. and ELLINGBOE, A.H. (1971). 'Transfer of 35S from wheat into the powdery mildew fungus with compatible and incompatible parasite/host genotypes.' CANADIAN JOURNAL OF BOTANY ,49, 303-310.

SPENCER, D.M. (1980). 'Parasitism of carnation rust (Uromyces dianthi) by Verticillium lecanii.' TRANSACTIONS OF THE BRITISH MYCOLOGICAL SOCIETY ,74, 191-194.

STACE, C.A. (1977). 'The origin of radiate Senecio vulgaris L.' HEREDITY ,39, 383-388.

STAKMAN, E.C., PIEMEISEL, F.J. and LEVINE, M.N. (1918). 'Plasticity of biologic forms of Puccinia graminis.' JOURNAL OF AGRICULTURAL RESEARCH ,15, 221-250.

STAUB, T., DAHMEN, H. and SCHWINN, F.J. (1974). 'Light and scanning electron microscopy of cucumber and barley powdery mildew on host and non-host plants.' PHYTOPATHOLOGY ,64, 364-372.

STAVELY, J.R. and HANSON, E.W. (1966). 'Some effects of temperature and relative humidity on development of Erysiphe polygoni on Trifolium pratense.' PHYTOPATHOLOGY ,56, 940-943.

STUCKEY, R.E. and ELLINGBOE, A.H. (1974). 'Elongation of secondary hyphae of Erysiphe gramins f.sp. tritici on wheat with compatible and incompatible parasite/host genotypes.' PHYTOPATHOLOGY ,64, 530-533.

SUTHERLAND, R.A. (1986). 'A method for inferring the minimum number of genes controlling the reactions of cultivars of a host plant species to isolates of a pathogen under a gene-for-gene model.' BIOMETRICS ,42, (In Press).

TAPKE, V.J. (1953). 'Further studies on barley powdery mildew as influenced by environment.' PHYTOPATHOLOGY ,43, 162-166.

TARR, S.A.J. (1972). "The principles of plant pathology." MacMillan, London.

TRONSMO, A. and DENNIS, C. (1977). 'The use of Trichoderma species to control strawberry fruit rots.' NETHERLANDS JOURNAL OF PLANT PATHOLOGY ,83(Suppl 1), 449-455.

TROW, A.H. (1913). 'On the inheritance of certain characters in the common groundsel - Senecio vulgaris Linn - and its segregates.' JOURNAL OF GENETICS ,2, 239-276.

TROW, A.H. (1916). 'On the number of nodes and their distribution along the main axis in Senecio vulgaris and its segregates.' JOURNAL OF GENETICS ,6, 1-65.

VAN ALFEN, N.K. (1982). 'Biology and potential for disease control of hypovirulence of Endothia parasitica.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,20, 349-362.

VANDERPLANK, J.E. (1963). "Plant diseases : Epidemics and control." Academic Press. London.

VANDERPLANK, J.E. (1968). "Disease resistance in plants." Academic Press, New York. pp206.

VANDERPLANK, J.E. (1984). "Disease resistance in plants." 2nd Edition. Academic Press, New York. pp194.

VAN EMDEN, H.F., EASTOP, V.F. HUGHES, R.D. and WAY, M.J. (1969). 'The ecology of Myzus persicae.' ANNUAL REVIEW OF ENTOMOLOGY ,14, 197-270.

WAHL, I. (1970). 'Prevalence and geographic distribution of resistance to crown rust in Avena sterilis.' PHYTOPATHOLOGY ,60, 746-749.

WALKER, J.C. (1969). "Plant pathology." 3rd Edition. McGraw-hill Book Company, New York. p632, pp819.

WHITE, E.M. (1982). 'The effects of mixing barley cultivars on incidence of powdery mildew (Erysiphe graminis) and on yield in Northern Ireland.' ANNALS OF APPLIED BIOLOGY ,101(3), 539-546.

WHITE, N.H. and BAKER, E.P. (1954). 'Host pathogen relations in powdery mildews of barley. I. Histology of tissue reactions.' PHYTOPATHOLOGY ,44, 657-662.

WOLFE, M.S. and MINCHIN, P.N. (1979). 'Effects of variety mixtures.' REPORT OF THE PLANT BREEDING INSTITUTE CAMBRIDGE, 1978. p148-150.

WOLFE, M.S. and SCHWARZBACH, E. (1978a). 'The recent history of the evolution of barley powdery mildew in Europe.' In "The powdery mildews." Ed. SPENCER, D.M. Academic Press, London , New York.

WOLFE, M.S. and SCHWARZBACH, E. (1978b). 'Patterns of race changes in powdery mildews.' ANNUAL REVIEW OF PHYTOPATHOLOGY ,16, 159-180.

WOLFE, M.S., BARRETT, J.A. and JENKINS, J.E.E. (1981). 'The use of cultivar mixtures for disease control.' In "Strategies for control of cereal disease." Eds. JENKYN, J.F. and PLUMB, R.T. Blackwell Scientific Publications, Oxford. p73-80.

WOOLACOTT, B. and ARCHER, S.A. (1984). 'The influence of the primary germ tube on infection of barley by Erysiphe graminis f.sp. hordei.' PLANT PATHOLOGY ,33, 225-231.

WRIGHT, A.J. and HEALE, J.B. (1984). 'Adult plant resistance to powdery mildew (Erysiphe graminis) in three barley cultivars.' PLANT PATHOLOGY ,33, 493-502.

YARWOOD, C.E. (1939). 'Control of powdery mildew with a water spray.' PHYTOPATHOLOGY ,29, 288-290.

YARWOOD, C.E. (1957). 'Powdery mildews.' BOTANICAL REVIEW ,23, 235-301.

YATES, F. (1981). "Sampling methods for censuses and surveys." 4th Edition. Charles Griffin and Company Ltd. London and High Wycombe. p99-104.