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STUDIES ON THE SENECIO VULGARIS L - ERYSIPHE FISCHERI BLUMER, PLANT PATHOSYSTEM

by

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A thesis presented for the Degree of

Doctor of Philosophy

in

The Faculty of Science

at the University of Glasgow

Department of Botany,
University of Glasgow.

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To

My Mum and Dad

Mr. and Mrs. Obu Atuboinoma Harry

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SUMMARY

This thesis is in two parts. The first part reports a study of resistance factors in Senecio vulgaris (Groundsel) and of them matching virulence factors in its parasite Erysiphe fischeri. The second part reports a study of the effects of E. fischeri infection on the growth, development and functioning of the leaf tissue of S. vulgaris.

PART I

Eight resistance factors to the mildew were identified in a number of groundsel plant lines on the basis of differential reactions of the plant lines to eight different fungal isolates. Plant lines possessing no resistance factors to any of the isolates and plant lines possessing three or more resistance factors were more common in the host population than expected while plant lines possessing one or two resistance factors only were less common than expected. The selective pressures leading to this situation may have been imposed by the parasite although other environmental factors could have played a part.

The resistance factors were found to be inherited in a simple Mendelian manner with resistance generally dominant over susceptibility. Resistance factors R_3, R_4 and R_5 were found to be in the same linkage group, R_3 and R_5 being closely linked with a recombination value of about $1.5 \pm 1.5\%$.

An analysis of the virulence factors possessed by each of the eight isolates of the mildew revealed that each possessed virulence factors

matching all but one of the eight resistance factors in the host. The isolates were thus complex races. Thus, natural selection presumably as a result of selection pressures imposed by the host population has led to the development of a series of complex races, each race possessing the ability to overcome many of the resistance factors in the host. Over half of the plants susceptible to each race were very susceptible giving an infection type 4 to that race while only about 20% gave more resistant infection types 2 or 3. Thus selection appears to favour the most virulent condition in the parasite.

At least six morphological forms of groundsel were identified on the basis of stem characters (red or green; hairy or glabrous) and inflorescence characters (radiate or non-radiate) and these were present in all populations studied. The population structure of groundsel in relation to the distribution of resistance factors and of morphological characters indicates that groundsel populations are genetically very diverse and is therefore a more complex form of a mixed variety cropping system. It is thus likely that long term usage of a mixed variety cropping system to control crop parasites may lead to the development of complex races which are able to attack most, if not all, the cultivars.

PART II

When groundsel plants have more than 30% of their leaf surface infected with mildew, both dry matter production and leaf expansion are reduced. Factors affecting this reduction in growth have been investigated.

Neither the chlorophyll content per unit area of leaf nor the unit leaf rate were reduced even with 90% total mildew cover. Thus reduction in the rate of dry matter production was not due to a reduction in the efficiency of the leaf as a photosynthesizing organ, but to a reduction in the total green leaf area.

Infection reduced the total green leaf area by enhancing leaf senescence, by reducing the number of expanding leaves on the lateral branches and by reducing the rate at which individual leaves expanded. The extent to which infection reduced the area of individual leaves depended upon the time of infection. Infection did not affect fully expanded leaves, but it drastically reduced the area of leaves which were infected while still expanding. Such leaves contained fewer and smaller lower epidermal cells than did un-infected leaves, indicating that infection inhibits both cell division and cell expansion.

The succulence of the leaves was reduced by levels of infection over 60% cover. In such leaves, the amount of water lost per unit area of leaf appeared to be related to the amount of mildew present. It is thus possible that water stress in the leaves was partly responsible for the reduced expansion of the leaves.

This investigation thus indicates that *S. vulgaris* has a high level of tolerance to *E. fischeri*.

PART 1: HOST/PARASITE INTERACTION

CHAPTER 1: GENERAL INTRODUCTION

The primary objective of breeding for disease resistance in crop plants is to reduce the mean fitness of their parasites by controlling the genetic composition of the host populations with which the parasite populations must interact. This restriction on the ability of the parasite population to grow and reproduce enables the host to continue growth longer and thus its yielding capacity may be increased. The commonest means of controlling plant diseases by resistance breeding has been through the use of major genes.

In a few cases, major gene resistance has given a long lasting protection. For example, cultivars of tomato possessing race specific resistance genes have been used effectively for the control of nailhead spot; a fruit, stem and leaf spot disease caused by *Alternaria tomato* (Weber and Ramsey, 1926; Crill, 1973). This control method has been so effective that Crill (1977) reported that the parasite might be extinct, since the disease has not been reported since this control programme was implemented.

However, in most cases, major gene resistance has been effective only in the short term. Thus cultivars of wheat and oats having race specific resistance to stem and crown must have been found to be effective only until new parasite races to which the resistance is not effective become established (Ausemus, 1943; Van der Plank, 1968).

The transient nature of major gene resistance has been in many cases due to the tremendous variability of fungal parasites.

Variation in fungal parasites

The occurrence of variation in fungal parasites of crop plants was first reported by Eriksson (1894). He (1894) demonstrated that Puccinia graminis which was originally regarded as a single species capable of infecting a wide range of cereals and grasses could be subdivided into at least six formae speciales (f. sp.), tritici, avenae, secalis, phleipratensis, agrostidis and poae, according to the principal host species which each attacks.

Thus the principal host genus for P. graminis f. sp. tritici is Triticum, but it also attacks cultivated and wild species of Hordeum and Secale, and a number of grasses including Agropyron, Bromus and Elymus. The principal host for P. graminis f. sp. avenae is Avena, but several grass-genera different from those above and including Dactylis are also attacked. P. graminis f. sp. secalis has species of Secale as its main hosts, but it can also attack Hordeum and wild grasses of the same genera as those attacked by P. graminis f. sp. tritici. Similarly, the main host genus of P. graminis f. sp. phleipratensis is Phleum, although it also attacks Avena, Secale, Hordeum and Festuca species. The main host genus of P. graminis f. sp. agrostidis is Agrostis and that of P. graminis f. sp. poae is Poa.

Johnson et al. (1932), reported that though P. graminis f. sp. tritici and P. graminis f. sp. secalis have Hordeum as a common

secondary host, they did not infect this as vigorously as each infected their principal hosts, Triticum or Secale respectively. Similarly, Johnson and Newton (1933) demonstrated that the hybrids between P. graminis f. sp. avenae and P. graminis f. sp. tritici were nearly always less pathogenic on Avena or Triticum than was either parent on its particular host. It thus would appear that the ability of these special forms to grow on their particular hosts requires a fairly precise genetic interaction between them which is readily disrupted by hybridization.

The existence of special forms has also been demonstrated in other host parasite systems. Thus, Marchal (1902) demonstrated the existence of seven special forms of Erysiphe graminis on the basis of its host specialization on cereals and grasses. Thus E. graminis f. sp. tritici infects Triticum species E. graminis f. sp. hordei infects Hordeum species; E. graminis f. sp. secalis infects Secale species; E. graminis f. sp. avenae infects Avena species and Arrhenatherum elatus; and E. graminis f. sp. poae infects Poa species. Similarly, E. graminis f. sp. agropyri infects Agropyron species while E. graminis f. sp. bromi infects Bromus species.

Salmon (1904) and Reed (1909), studied the effect of E. graminis f. sp. hordei on certain species within the genera Hordeum, Triticum, Avena and Secale, and obtained similar results to those of Marchal (1902). They, however, did not study the effect of any of the special forms on wild grasses.

Several investigators (Barus, 1911; Stakman and Piemeisel, 1917; Mains and Dietz, 1930; Waterhouse, 1930) have shown that the formae

speciales of many parasites can be further subdivided into physiologic races on the basis of their ability to parasitize some cultivars of their main host but not others.

Thus, Barrus (1911) in studies on the Phaseolus vulgaris/ Colletotrichum lindemuthianum plant pathosystem showed that the parasite population could be separated into two races, α and β on the basis of pathogenic responses they elicited on different cultivars of P. vulgaris. This approach was taken up and extended to other host/parasite systems.

Thus Stakman and Piemeisel (1917) showed that the formae speciales of Puccinia graminis were each composed of a number of 'physiologic races'. These races were identified by the characteristic responses each elicited on selected groups of cultivars of their respective hosts.

Mains and Dietz (1930) also reported the existence of physiologic races in Erysiphe graminis f. sp. hordei. They studied the reactions of forty cultivars of four Hordeum species (Hordeum vulgare, H. intermedium, H. distichon and H. deficiens) to five isolates of the fungus and found that among the cultivars of each host species there existed some which were either resistant or susceptible to one or more isolates of the parasite. They reported that sixteen cultivars were resistant to all five isolates, five cultivars were resistant to four isolates but susceptible to one, eight cultivars were resistant to three isolates but susceptible to two, two cultivars were resistant to two isolates but susceptible to three, five cultivars were resistant to one isolate but susceptible to four and four cultivars were susceptible to all five isolates. On the basis of

this work, they concluded that E. graminis f. sp. hordei consisted of at least five physiologic races.

Mains (1933), using a series of wheat cultivars also showed that E. graminis f. sp. tritici could be subdivided into at least two physiologic races. Since then much work has been done on this host parasite system. Thus Lowther (1950) inoculated forty collections of E. graminis f. sp. tritici onto six cultivars of Triticum (Axminster, C.I. 8195, Ulka C.I. 11478, Chul C.I. 2227, Hope C.I. 8178 and Normandie C.I. 12747) and was able to identify nine physiologic races of the parasite. Wolfe (1965), in similar work, using these as well as other cultivars of wheat identified thirteen physiologic races of E. graminis f. sp. tritici in the United Kingdom. Eleven of the thirteen races, however, corresponded with the races already identified in North-West Europe.

Physiologic races of powdery mildew fungi on hosts other than barley and wheat have also been studied. Jagger et al. (1938), reported a strain of Erysiphe cichoracearum in the Imperial Valley of California that was pathogenic on cultivar PMR 45 of cucumber. Since the strain of the fungus previously found in the Valley was avirulent on PMR 45, the new more pathogenic strain was designated race 2.

The genetical basis of race specific resistance

The differential reactions exhibited by host plant cultivars to specific races of their parasites were first shown by Biffen (1905, 1907) to have a genetic basis. He (1905) studied the inheritance of resistance in wheat (Triticum) to yellow rust (Puccinia glumarum now Puccinia

striiformis) and found that resistant and susceptible plants in the F_2 generation segregated in the ratio of 1 resistant plant to 3 susceptible plants. He concluded that one recessive gene conditioned the resistance reaction of the wheat cultivar to P. striiformis and thus established that disease resistance could be inherited in a simple Mendelian manner.

Since this initial discovery many workers have shown that resistance in a number of other host plants to their parasites is also inherited in a similar way, although most studies have shown that resistance is dominant to susceptibility.

Thus Hooker and Russell (1962) studied the inheritance of rust resistance in six inbred corn lines (B38, B49, K148, Cuzco, GG208R, P.1. 172332) to cultures 901 ab, 904a, 926, 921b, 930R and 927R of Puccinia sorghi. Lines B38, B49, K148, Cuzco, GG208R and P.1.172332 are resistant while B14 and M14 are susceptible to all six cultures of the pathogen.

They crossed each of the six resistant lines with each of the susceptible lines and then backcrossed to the susceptible parent. On testing the backcross progenies with culture 901ab, they segregated into resistant and susceptible plants approximately in a 1 : 1 ratio, indicating that resistance to culture 901ab was conditioned by a single dominant gene in each of the six resistant inbred lines.

They then continued the cross Cuzco x B14 through to the F_3 generation, testing each F_3 line with each of cultures 901ab and 904a to determine whether it was true breeding or segregating for resistance and

susceptibility. In this way they were able to determine the proportions of homozygous resistant, heterozygous resistant and homozygous susceptible genotypes in the F_2 generation. They found that the F_3 lines consisted of one true breeding resistant: two segregating resistant: one true breeding susceptible plant when inoculated with both cultures, and they concluded that Cuzco possessed single dominant genes conditioning resistance to the two cultures.

It has also been shown (Allard and Shands, 1954) that disease resistance in certain cultivars of wheat to some races of Erysiphe graminis f. sp. tritici or Puccinia graminis f. sp. tritici may be conditioned by dominant duplicate linked genes. Allard and Shands (1954) crossed two wheat cultivars C.1. 12632 and C.1. 12633 which were both resistant to races 17 and 19 of Puccinia graminis f. sp. tritici and E. graminis f. sp. tritici with two susceptible cultivars, Reward and Marquis. The F_1 plants were uniformly resistant to both races of P. graminis f. sp. tritici, thus showing that resistance was dominant over susceptibility. In the F_2 plants the ratio of resistant to susceptible individuals was almost double the expected ratio of 3 resistant : 1 susceptible plant expected if one dominant gene had been involved. From these results and from those obtained from calculating the map distance between the genes, they suggested that resistance in C.1. 12632 and C.1. 12633 to P. graminis f. sp. tritici was conditioned by dominant duplicate genes linked with a combination value of $14.78 \pm 1.75\%$.

From similar tests with E. graminis f. sp. tritici they showed that resistance in C.1. 12632 and C.1. 12633 was conditioned by dominant

duplicate genes different from those conditioning the resistance reaction in the cultivars to P. graminis f. sp. tritici.

Sears et al. (1957) studied the inheritance of disease resistance in cultivar Thatcher of Triticum aestivum to race 56 of Puccinia graminis f. sp. tritici. A test of an F_2 population of a cross between Thatcher (resistant parent) and Chinese (susceptible parent), using race 56 of the parasite, gave a segregation closely approaching seven resistant plants to nine susceptible plants. They reported that this 7 : 9 ratio showed that there were two recessive genes, either or both of which conditioned resistance.

Thus in these and many other studies, resistance to each physiologic race has been shown to be conditioned by one (Hooker and Russel, 1962) or more dominant genes (Moseman and Starling, 1958); one or more recessive genes (Sears et al., 1957) or by a combination of dominant and recessive genes (Wu and Ausemus, 1953).

The inheritance of pathogenicity in the parasite

Just as disease resistance in most host plants is inherited in a simple Mendelian manner so also is pathogenicity in the parasite. Several investigators (Flor 1942, 1956; Moseman, 1956; Leogering and Powers, 1962) have demonstrated that pathogenicity can be conditioned by one or several genes with avirulence being usually dominant to virulence.

Thus, Leogering and Powers (1962) studied the inheritance of pathogenicity in Puccinia graminis f. sp. tritici. They used race 111-55A, which was avirulent on most wheat cultivars and race 36-55A which was

virulent on many wheat cultivars. Crosses between the two races were carried out on Berberis vulgaris and the resulting aeciospores from a single aecial cluster were inoculated back onto Triticum compactum (Little Club) seedlings. They found that the pathogenicity of the resulting uredial culture, the F_1 generation was similar to the avirulent parent race 111-55A, thus indicating that avirulence was dominant over virulence.

To obtain the F_2 generation they inoculated Berberis vulgaris with germinating teliospores obtained from the F_1 generation. From these inoculations they obtained 108 aecia from which they obtained 108 uredial cultures which they regarded as the F_2 population. The pathogenicity of each of these 108 F_2 cultures was determined on twenty cultivars of wheat.

On the cultivar Chinese Spring which has a single resistant gene, 81 of the 108 cultures were avirulent while 27 were virulent, giving a ratio of 3 avirulent : 1 virulent. This indicates the segregation of a single gene (P_5) pair with avirulence dominant to virulence.

On the cultivar Marquis, cultures 111-55A and the F_1 were found to be avirulent while culture 36-55A was virulent. This also indicates that dominance of avirulence over virulence. The 108 F_2 cultures segregated on this cultivar into 80 avirulent, 21 intermediate and 7 virulent cultures. This fits a 12 : 3 : 1 ratio indicating the segregation of two independent genes for pathogenicity.

After testing all the twenty cultivars, they discovered that at least eight independent genes conditioned pathogenicity.

The gene-for-gene hypothesis

Resistance and susceptibility are clearly expressions of specific incompatible or compatible interactions between the hosts and pathogens metabolisms and thus ultimately between their genotypes. The specificity of these interactions has been studied by several workers (Flor, 1942, 1955; Moseman, 1957; Powers and Sando, 1957).

Thus, Flor (1942,1955) in his studies on the relationship between the host and parasite genetic systems of *Linum usitatissimum* and *Melampsora lini* showed that each pathogen race carried genes for avirulence or virulence corresponding to the genes for resistance or susceptibility present in its host. He reported that on cultivars of *L. usitatissimum* that have one gene for resistance to the avirulent parent race of the pathogen, the F_2 cultures of *M. lini* segregated giving monofactorial ratios and on cultivars having two genes for resistance to the avirulent parent race, the F_2 cultures of *M. lini* segregated giving bifactorial ratios. From these results, he concluded that for each gene that conditions a reaction in the host there is a corresponding gene in the pathogen that conditions pathogenicity. This is the basis of his gene-for-gene hypothesis. He (1971) further reported that the gene-for-gene hypothesis is applicable only to host-parasite systems in which resistance in the host is conditioned by major genes.

Since this discovery, similar gene-for-gene systems have been shown to operate in several host parasite systems. Flor (1971) lists a number of them.

Moseman (1957) showed that a gene-for-gene system operates in the relationship between barley (*Hordeum vulgare*) and *Erysiphe graminis f. sp. hordei* genetic systems. He studied the interaction of the genes conditioning resistance to *E. graminis f. sp. hordei* in barley cultivars Kwan and Goldfoil and the genes conditioning pathogenicity on these cultivars in isolate 12A1 of race 12. He also studied the relationship of genes conditioning resistance to isolate CR3.1 of race 3 and isolate 21.1-3 of race 8 in the same two cultivars. From his results, he concluded that cultivars Kwan and Goldfoil each has one gene conditioning resistance to isolates CR3.1 and 21.1-3 and these genes are inherited independently. These genes were designated Ml_k and Ml_g respectively. By studying the progeny of a cross between isolates 12A1 and 21.1-3 he determined the inheritance factors for pathogenicity on Kwan and Goldfoil and showed that isolate 12A1 had one gene for pathogenicity on each of the two cultivars. These two genes A_k and A_g respectively appeared also to be inherited independently. He thus showed that two independently inherited genes Ml_k and Ml_g in Kwan and Goldfoil conditioned resistance and two corresponding independently inherited genes A_k and A_g conditioned pathogenicity in the fungal isolate 12A1, (one for each cultivar).

Moseman (1966) stated that a high infection type (3-4) resulted when the pathogen gene corresponding to the resistance host gene was virulent, and when the host gene was for non-resistance or susceptibility. A low infection type (0-2), he said, resulted only when the pathogen gene corresponding to the host resistance gene was avirulent. These interactions can in general terms be represented as shown in Table 1.

TABLE 1

Infection types resulting from the interactions of incompletely dominant host genes and the corresponding pathogen genes.

HOST GENES	PATHOGEN GENES A	V
Ml/Ml	0	4
Ml/ml	2	4
ml/ml	4	4

Where:

Ml/Ml	=	homozygous dominant genes conditioning resistance
Ml/ml	=	heterozygous incompletely dominant genes conditioning moderate resistance
ml/ml	=	homozygous recessive genes conditioning susceptibility
A	=	corresponding avirulence gene
V	=	corresponding virulence gene

The use of major gene resistance for disease control

Plant breeders have commonly incorporated major genes singly into commercially acceptable cultivars using the backcross technique which is a relatively simple breeding technique. The presence of single resistance genes has imposed strong selection pressure on the parasite population leading eventually, sometimes fairly rapidly to the development of races virulent on plants containing the resistance gene. Plant breeders have responded to this frequent breakdown by incorporating different race specific resistance genes into new cultivars which are then widely grown.

In the case of bread wheat and black stem rust, genes for resistance were extracted from Triticum durum (durum wheat) and Triticum dicoccum (emmer wheat) and incorporated into bread wheat types. Durum and emmer wheat resistances effectively controlled the stem rust through the 1940's (Ausemus, 1943), but race 15B of Puccinia graminis f. sp. tritici appeared in 1953 and forced their retirement, (Stakman and Rodenhiser, 1958). 'Selkirk' then became the leading spring wheat. Sources of resistance from Kenya wheats and 'Khapli' emmer have also been used to control wheat stem rust (Frey and Browning, 1971).

Sources of disease resistance genes

The basic requirements for breeding disease-resistant plants are sources of resistance and the methodology for combining this resistance with commercially acceptable plant types. In order to continue resistance breeding, there must be a continual search for germ

plasm with disease resistance traits that meet the changing needs of plant breeders.

Bolley (1901), Orton (1909), Vavilov (1949-1950, 1957), Zhilkovsky (1959, 1961, 1964) and others have shown that a wealth of natural resistance genes occur among several cultivated crops and their wild progenitors, particularly in their centres of origin. In these centres both host and parasite have long been associated as reciprocal selective factors in evolution and whenever serious diseases occurred in nature the highest degree of resistance has been found among the surviving wild species and cultivars as a result of natural selection. In addition to these sources, disease resistance genes can also be obtained from mutagen treated populations by means of selection. Selection is the primary means of isolating sources of disease resistance genes from cultivated crops, related species or mutagen treated populations. The resistant survivors of naturally or artificially induced epiphytotics are selected and used directly for disease control or for further breeding (Hayes et al., 1955; Cherewick, 1946; Kirally et al., 1970).

The practical problem now faced by plant breeders is how best to use the resistance genes to obtain maximum benefit, since as was stated earlier, introducing them singly into cultivars and sowing uniform cultivars in a given area has generally given only short term protection (Hartly, 1939; Stevens, 1942, 1948; Stakman and Rodenhiser, 1958). Hartly (1939) advocated planting mixtures of clones rather than single clones. The resulting heterogeneous population being expected to control pathogen spread and development in the population, by avoiding the genetic

uniformity that leads to pest epidemics (Ullstrup, 1972; National Academy of Sciences, 1972). The yield of the mixtures in the presence of infection would thus be expected to be higher than that of either of the component lines in pure stands. Mixtures of remotely related plants; interspecific and intraspecific mixtures of plants are some of the tactics suggested. It is intended here to discuss these tactics in broad terms limiting the discussion to a few examples that are thought to be of relevance in the context of this thesis.

Mixtures of remotely related pure stands

Wheat-oat mixtures have been reported to reduce rust penetration through the plot (Suneson, 1960) and to yield more than either component in a pure stand under rust attack (Atkinson, 1900).

In the stem rust epiphytotics of 1935, mixtures of resistant oats and susceptible barley yielded more than the mean of the pure stands (Klages, 1936). Oat-barley mixtures have also been suggested (Vacke, 1967) for the control of oat sterile-dwarf virus disease.

Interspecific mixtures

The cotton crop of a part of Central India (Malwa Plateau) is a complex mixture of two cotton species. The mixture called Malwa, is composed of several strains of the indigeneous diploid, 'Desi' (*Gossypium arboreum*) plus a variable proportion of the introduced American tetraploid 'Upland' (*G. birsutum*). The Upland in pure stand performs very poorly and suffers much from disease, especially red leaf and leaf roll, but in the mixture grows better because it is less attacked by the disease.

Upland is resistant to cotton wilt to which Desi is susceptible and appears to check the spread of this disease in the Desi component. The integrity of the component species is preserved because introgression has not been shown to occur (Simmonds, 1962). The yields and quality are higher than the means of the components (Aiyer, 1949).

Pfahler worked with mixtures of cultivated oat species for grain (1964) and grain and forage yield (1965). He found that the fitness of the composites exceeded that of the components for both grain and forage yield.

Intraspecific Mixtures or Mixed varieties

Browning (1957) reported that mixtures of two oat cultivars. Clintland and Mo 0-205, yielded 13% more than the mean of the pure stands when under rust attack. This blend also produced a 5. 5% advantage when no rust was present, indicating that other factors besides disease control lead to increased yield in such mixtures.

Although mixtures of cultivars of several crops produce higher yields than the components of the mixtures in pure stands, there are a few drawbacks to their use. The components of a mixture may germinate at different times and subsequently may have different rates of growth leading to different times of ripening of the grains. Secondly, mixed grains in most cases can only be used for animal feed as most industrial processes demand a uniform crop sample. For example, the Australian barley marketing board demands a uniform crop so that maltsters will have less trouble in adjusting their methods to their raw materials

(Day, 1973). To overcome the problem of lack of uniformity Borlaug (1959,1965) has suggested the use of multilines.

Multilines are precisely determined mixture of backcross derived lines. Each line contains a different resistance gene, but is as far as possible isogenic in all other respects. Borlaug (1959, 1965) suggests that race surveys should be carried out so that the components of the mixture can be varied depending on the prevalent races. The proportion of susceptible components in the mixture can then be kept low. A high proportion of resistant plants in the mixture would be expected to delay the development of the parasite on susceptible components enabling them to mature with light damage.

The first wheat multiline cultivar in commercial production was Miramar 63, which was released for use in the high, cool savannahs of the Andes where stripe rust was serious (Rockefeller Foundation Programme, 1963). To develop Miramar 63, the Brazilian wheat frocor was crossed with some 600 varieties and lines. Over 1, 200 lines, similar phenotypically to frocor, but with resistance genes from the 600 non-recurrent parents were produced. Miramar 63 is a mechanical mixture of equal parts of ten of the best lines giving resistance to stripe and stem rust. It was widely accepted and its yield more than doubled those of older varieties in some areas (Rockefeller Foundation Programme, 1964). Within two years of its release, stem rust was parasitizing two component lines, but total losses were always less than expected. The two stem rust susceptible lines and two others were dropped from the composite variety and four new ones from the reserve of over 600 lines were added

to form a new multiline cultivar, Miramar 65. This thus illustrates the plasticity that can be inherent in multiline cultivars.

Pyramiding race specific resistance genes

It has also been suggested that if several major genes are incorporated into a single genetic background, then population shifts of the parasite will be curbed. The argument being that if the mutation rates of each avirulence gene to virulence in the parasite is low, say, one in a million (1×10^{-6}) then the probability of mutation to virulence in the two or more avirulence genes that would be required to enable the parasite to attack a cultivar with the two or more complementary resistance genes would be obtained by multiplying the mutation rates of the individual avirulence genes, (1×10^{-n}). So the more resistance genes in a cultivar, the longer it will take for the appropriate mutations in the parasite to occur. Thus the cultivar is likely to remain resistant that much longer. This method is strongly advocated by Nelson (1973). Pyramiding of resistance genes has been found to be effective in *Zea mays* for the control of *Trichometasphaera turcica* (Nelson *et al.*, 1970).

Changes in the parasite population in response to the use of multilines or mixed varieties

Whilst there is a lot of evidence to show how parasite populations change in response to resistance genes used singly in monoculture, multilines have been used for too limited a period to show how parasite populations will respond to their continued use.

Simmonds (1962) has warned that long term usage of multiline cultivars on a continental scale could result in highly heterogeneous

parasite populations containing races which are virulent on all components of the multiline.

Hooker (1972) points out that there seems to be no reason why races virulent to specific resistance should not develop on multilines as well as on pure stands. What has been accomplished in decades, he says, when the fungus is confronted with one gene at a time, may be accomplished in a few years when the fungus is confronted with all the genes present singly or in groups in different varieties of isogenic lines.

It is clearly not known whether multilines or mixed varieties with combinations of resistance genes will have lasting disease resistance or not. A clue may be obtained from studies on how resistance is deployed in wild plants and how their parasites have evolved in response to this. In wild plants evolution and natural selection have almost certainly led to the development of systems where resistance factors in the host and virulence factors in the parasite are distributed in their respective populations to provide the maximum and most lasting protection possible.

Thus studies of wild plant pathosystems would indicate if race specific resistance is an important component of the host plant and if so, how the genes are deployed within the population. If they are deployed to provide multiline or mixed variety systems, then studies of virulence in the parasite population would indicate the likely long term responses or evolutionary changes that multilines or mixed varieties are likely to bring about. It should also provide indications of how the virulence of parasites on crop plants may evolve with the continued use of multilines

or mixed varieties. Knowing how parasite populations are likely to evolve may suggest ways in which resistance factors may be utilized in crop plants to minimize evolutionary tendencies in the parasite. The study reported in this thesis was undertaken to investigate one such relationship.

The Senecio vulgaris L - Erysiphe fischeri Blumer plant pathosystem was chosen for several reasons: The host plant S. vulgaris is a very common annual weed in the United Kingdom and is frequently infected by its powdery mildew E. fischeri. Although supporting heavy infections of the parasite, the host is still an extremely common plant.

Although groundsel (S. vulgaris) is inbreeding producing numerous 'seeds' (achenes-single seeded fruits), cross pollination also occurs contributing to the wide range of morphological and physiological variation which occurs within natural populations.

It has a relatively short life cycle of about 12 -14 weeks, which allows the production of several generations of plants per year. Thus providing ideal material for genetic investigations.

The parasite E. fischeri is very common, and can be made to infect the plants at all times of the year in the growth cabinet. Infection can be obtained over a wide range of environmental conditions.

CHAPTER 2: AN INVESTIGATION OF A PROBABLE GENE-FOR-GENE SYSTEM IN THE S. VULGARIS/ E. FISCHERI PATHOSYSTEM

GENERAL METHODS: THE HOST

Collection of host plant materials

Seeds from 6 - 19 individual plants were collected from different locations per site and a total of 25 sites were sampled, Figure 1.

Production of plant materials

All plants were raised from seeds sown in 11.4 cm plastic pots in Levington potting compost. Plants were transplanted about two weeks after germination into 11.4 cm pots containing the same compost; one plant per pot. Plants were either grown in growth room maintained at a constant temperature of about 19°C with a 16 hour photoperiod or in a greenhouse, depending upon the requirements of the experiment. There was no temperature control in the greenhouse and the temperature usually ranged from 15 - 28°C. Additional light was not supplied; the plants were allowed to grow under natural day and night regimes. Plants in the growth room were placed in 58.4 x 27.9 cm trays; 8 plants per tray and were allowed to grow without protection until capitula appeared.

Production of inbred lines

In order to determine the resistance factors present in each plant line, it was essential to study populations of lines which were as genetically uniform as possible. In an attempt to achieve this, each plant line was inbred for three generations prior to further analysis, using the following method.

FIGURE 1: Locations of sites from which host plant collections were made.

KEY

1	Glasgow	14	Dumfries
2	Crail	15	Aberdeen
3	Ayr	16	Pitlochry
4	Dublin	17	Kingussie
5	Far Sawrey	18	Inverness
6	Ulverston	19	Lairg
7	Coniston	20	Portree
8	Wellesbourne	21	Mallaig
9	Perth	22	Fort William
10	Stranraer	23	Crianlarich
11	Abington	24	Oban
12	Peebles	25	Stirling
13	Hawick		

FIG. 1

At the appearance of the capitula, each plant was covered with an 11.4 - 12.7 cm transparent plastic propagating cover (Stewart Plastic, Plate 1) to protect the florets from cross pollination. The pots were watered from below by pouring water into the trays. This avoided any need for uncovering the plants and exposing them to chance cross pollination. Ripe seeds were collected from one plant of each plant line, only if all the plants of that line were morphologically uniform, i.e. were not segregating for any obvious characteristic, and were stored in transparent cellophane packets. Progenies from each seed lot were then raised and selfed in the same way for two further generations, seeds only being collected at each stage from non-segregating lines.

Production of mildew free plants

In order to determine which resistance factors were present in each inbred line, it was essential to obtain mildew free plants for inoculation. For this reason, plants of each of the inbred lines were raised as required in the growth room. Three plants of each plant line were covered at germination with propagating covers, to protect them from any chance infection.

The Parasite - isolation of isolates of Erysiphe fischeri

Further plants of each inbred plant line were grown in the greenhouse and allowed to become naturally infected with E. fischeri. During this period, the greenhouse ventilators were left open to ensure maximum exposure of the plants to inoculum. The date when infection was first observed on each plant line, and the infection types (see page 30).

PLATE 1: The host plant grown under clear plastic covers to keep them free from mildew and to protect them from cross pollination.

occurring on the leaves and stems of each plant line, were recorded.

Observations

Infection among the susceptible varieties of the host exposed to natural infection in the greenhouse, was at different times, the most susceptible varieties being the first to become infected. The reaction of the plants to infection can be grouped into three types:-

(1) very susceptible (about 100% mildew cover),

(2) moderately resistant (about 50% mildew cover),

(3) totally resistant (about 0% mildew cover).

In very susceptible plant lines, the growth and development of the fungus was rapid and by the third week, the fungus had completely colonized the entire surface of the plants. Neither necrosis nor chlorosis was observed.

The moderately resistant plant lines showed either necrotic or chlorotic reaction at the points of infection on the leaf surface. These reactions were probably a defence mechanism whereby the plants were able to remove the pathogen at the points of infection. This left dark brown or yellow patches on the leaf surface at the points of earlier infection, while the pathogen moved on to colonize fresh areas. The stems of some of these moderately resistant plant lines were found to be very susceptible without necrosis or chlorosis. Thus different parts of the plants exhibited a difference in the degree of susceptibility. Furthermore, the stems of plant lines 9a, 9b, 9j were resistant to infection while their leaves were moderately resistant. In a few plants, the stems were

resistant while the leaves were very susceptible (Appendix, Table 1).

Plant lines 7b, 8h and 10j did not show any visible reaction to Erysiphe fischeri infection. They remained resistant to the pathogen even after they had been grown for $2\frac{1}{2}$ years. They were, however, observed to be highly susceptible to Puccinia lagenophora.

The use of benzimidazole in detached leaf culture

E. fischeri is an obligate parasite and can only be maintained in culture on living host tissues.

Earlier methods for culturing obligate parasites had utilised detached leaves and had simply involved flotation of leaf segments on water in petri dishes, but the early death of such leaves had presented major problems (Yarwood, 1946).

Since the discovery that detached leaves of wheat and barley floated on a solution 27 - 60 ppm benzimidazole remained green and turgid longer than detached leaves floated on water (Person et al., 1957), these solutions have been successfully used by several investigators (Caldwell, 1960; Wolfe, 1965; Hermansen, 1966).

In order to provide the most suitable conditions for growth and development of E. fischeri on detached leaves of its host, it was considered necessary that the optimum concentration of benzimidazole be known.

A series of solutions containing 7.8, 10.0, 15.6, 20.0, 30.0, 40.0, 62.5, 125 and 250 ppm benzimidazole were prepared and 15 ml. of

each dilution was poured into three 9 cm glass petri dishes; distilled water was used in control dishes. A 9 cm disc of filter paper (Whatman No. 1) was placed into one of the three dishes of each dilution, to act as a support for the leaf segments. Mature fully expanded leaves were detached from healthy susceptible plants and each was cut across into segments of approximately 1 - 2 cm in length. Thirty segments were placed in each of two groups. One group of leaf segments was inoculated by blowing conidia from an infected plant onto them. With the conidia bearing surface uppermost, one leaf segment was placed into each petri dish. The un-inoculated leaf segments from the second group were then placed singly into each of the thirty dishes, so that each dish contained two leaf segments, one inoculated and the other un-inoculated. The dishes were then incubated for seven days in the laboratory at a temperature of about 23 - 25°C. An identical experiment, set up at the same time was incubated for seven days in the growth room.

To compare the levels of infection produced on leaves floating on each concentration of benzimidazole, with those on the leaves of whole plants, four plants from the same plant line as those used for the detached leaf experiments were inoculated by blowing conidia of the parasite from an infected plant over them. Two of these plants were then incubated in the laboratory for seven days and the other two were incubated in the growth room for the same length of time. This experiment was necessary since this is the natural condition in which the host and the parasite co-exist.

Results

Leaves floated on distilled water or a solution containing 30 ppm benzimidazole, gave levels of infection comparable with those on the leaves of whole plants, while concentrations between 7.8 and 20 ppm supported higher levels of infection. Concentrations above 30 ppm led to a gradual reduction of infection and above 125 ppm no infection occurred at all. Levels of infection at all concentrations where infection occurred were slightly higher in the growth room than on the laboratory bench. This was probably due to the higher light intensity and lower temperature maintained in the growth room. Leaves floating on distilled water showed signs of chlorosis at the end of the seven day period, but leaves were still green on solutions containing between 7.8 and 30 ppm benzimidazole. Above 30 ppm, the leaves developed increasing amounts of necrotic tissue. One of the features of leaf segments floating freely on the surface of the solutions, was the occasional presence of droplets of solutions on their surface. Where these droplets occurred, mildew growth was suppressed. Leaves supported on filter paper were less affected by water splash than those floating freely on the solution, because as the filter paper absorbed the solution, it puckered forming a support for the leaf segments.

Since a solution of 30 ppm benzimidazole supported growth of the parasite similar to that on whole plant, and since the leaf segments remained green, this concentration was used in most further experiments, with the leaf segments being supported on filter paper. In some of the later work, the leaf segments were supported on 30 ppm benzimidazole

solution solidified with 0.5% agar.

Isolation of single conidial isolates

Detached leaves were cut into lengths of about 1 - 2 cm and inoculated by blowing conidia of the parasite from an infected plant onto them. These were then placed in petri dishes on filter paper moistened with 30 ppm benzimidazole and incubated in the growth room for seven days. At the end of this period, a single chain of conidia from a single conidiophore was picked from one colony, under the disecting microscope using a steel needle. This was lightly touched on the surface of a fresh leaf segment and incubated. After incubation, one of the few colonies which developed was chosen, and a chain of conidia was again transferred onto a fresh leaf segment as described above. This procedure was repeated twice more and the resulting mildew colonies were then regarded to be pure lines.

Maintenance of single conidial isolates on detached leaves

Single conidial isolates were maintained on detached leaves as described. The isolates were transferred onto fresh leaf segments every three weeks.

Detached leaf segments of some lines produced adventitious roots in culture about 7 - 21 days after inoculation. These adventitious roots helped to prolong the life span of the leaves, possibly by increasing the rate at which the detached leaves absorbed the aqueous solution on which they were placed.

Maintenance of single conidial isolates on whole plants

Single conidial isolates were also maintained on whole plants which were covered with plastic propagating covers to prevent contamination by other isolates. By this method, isolates were maintained for periods of at least two months.

Assessment of infection and infection types

Several investigators (Moseman, 1956; Wolfe, 1965) have shown that isolates of powdery mildew fungi differ in virulence on different host varieties and this was found to be the case for E. fischeri on S. vulgaris. For this reason, it was considered necessary to quantify the infection types, E. fischeri produced on leaves of S. vulgaris. A common method for assessing the degree of susceptibility in the host has been the use of the mean infection type (Moseman, 1956) and this method was adopted (for use) in this work. The different infection types which occur on S. vulgaris have been grouped into essentially the same five types (0 - 4) as those described by Moseman (1956) and they are as follows:-

Infection type 0 = No spore 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 to be resistant reactions since they never led to the production of conidia, while infection types 2, 3 and 4 were considered to be susceptible since they allowed conidial production. Infection types 0, 2, 3 and 4 are illustrated in Plate 2.

Comparison of conidial production of infection types 2 and 4

In order to quantify more exactly the differences between the two extremes of susceptible infection types, 2 and 4, conidial production of isolate 1 (see page35) per unit area of infected leaf, was measured on plant line 24e, on which it gave a type 2 reaction and on plant line 24g on which it gave a type 4 reaction.

Ten leaf segments of each plant line were inoculated and incubated in five petri dishes (two leaf segments per dish) for ten days. At the end of this incubation period, discs were removed from each leaf segment using an 8 mm diameter cork borer. Each leaf disc was then shaken with 1 ml. distilled water containing a drop of 1% lactophenol in cotton blue in a 10 ml. test tube. The number of conidia in each 1 ml. of distilled water was then counted using a haemocytometer. An average of 10 - 15 counts per ml. was obtained and the number of conidia per mm^2 of infected leaf area was calculated. The results are given in Table 2.

These results show clearly marked differences in conidial production by isolate 1 on the two plant lines. Subjective observations suggest that similar differences in conidial production occur between all infection type 2 and infection type 4 reactions.

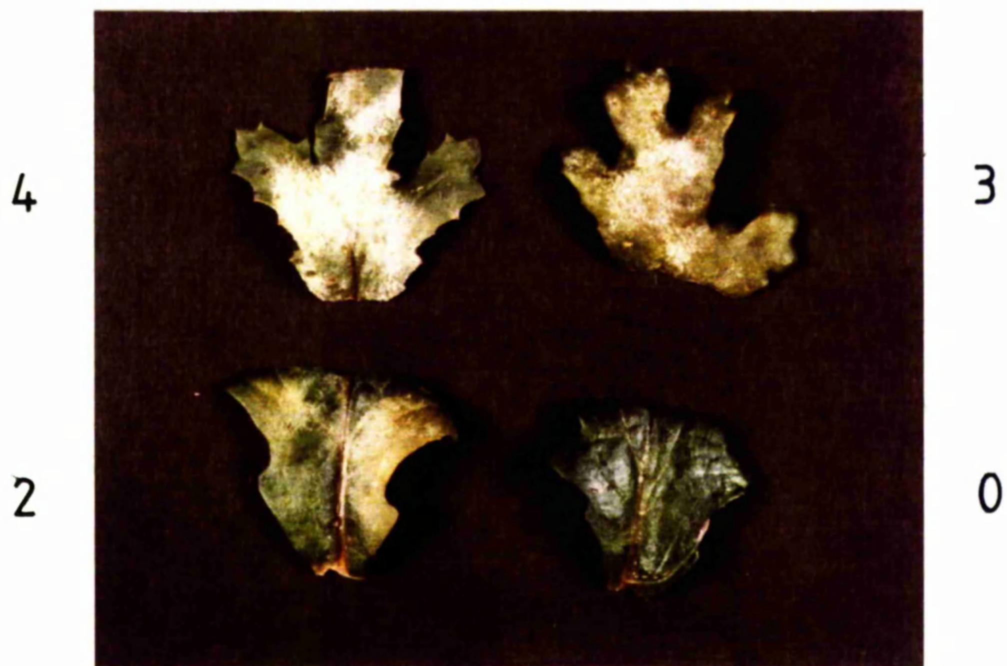


PLATE 2: Detached leaf segments showing infection types 0, 2, 3 and 4

TABLE 2

Comparison between infection types 2 and 4 caused (incited) by Isolate 1.

Replicates	No. of conidia/mm^2 on leaves of plant line 24e (infection type 2)	No. of conidia/mm^2 on leaves of plant line 24g (infection type 4)
1	57.5	944.5
2	49.7	1000.4
3	50.8	900.3
4	46.1	1000.0
5	51.2	835.2
6	49.3	946.9
7	50.0	721.1
8	42.6	1000.8
9	45.0	901.5
10	43.4	807.1
	Mean 48.6 ± 4.2	Mean 905.8 ± 88.8

Inoculation procedure of detached leaf segments

In studies on the interaction between hosts and parasites it is important that the isolates be inoculated onto the host free from contamination with other isolates. A method for inoculating detached leaf segments of several plant lines with inoculum of one isolate only is described below.

Four detached mature leaf segments (1 - 2 cm in length) were cut from each protected plant line. Two of the segments of each plant line were cut at right angles to the main vein while the other two segments were cut at an acute angle to the main vein. The leaf segments cut at an acute angle to the main vein were placed with the upper epidermis uppermost into a petri dish. The isolate to be used as the inoculum was then lightly touched with another piece of detached leaf segment and the adhering conidia were lightly touched on the surface of the two leaf segments in the petri dish. The two leaf segments cut at right angles to the main vein were used as un-inoculated controls and placed in the petri dish alongside the inoculated segments. The differences in the angle of the cut made it easy to distinguish un-inoculated from inoculated leaf segments when parasitic growth was insufficient to be seen by the naked eye or when infection did not occur. All inoculations with one isolate were completed and the inoculated leaf segments removed to the growth room before inoculations were attempted with other isolates. The dishes were incubated in the growth room for 7 - 10 days. At the end of the incubation period, the infection types on the inoculated leaf segments were assessed.

In order to be certain that host reactions were a true reflection of the plant's response and not due to the physiological condition of the conidia of the isolates used, or of the particular leaf segments of the plant line being tested, replicated lines from different sites were tested with different isolates at the same time. Leaf segments from highly susceptible clones were also always included as controls. All inoculations giving rise to a resistance reaction were repeated.

The inoculation of host plant seedlings

Seedlings of several plant lines were inoculated, covered with 11.4 - 12.7 cm transparent plastic propagating covers and then incubated for 7 - 10 days. This experiment was designed to investigate whether seedling reactions were similar to the reactions of adult plants and to the reactions of detached leaves. After establishing that seedling resistance was similar to adult plant resistance (Appendix, Table 2), seedlings were considered as a possible substitute for leaf segments in test inoculations. However, the method using seedlings was abandoned because it was found to be wasteful of plant material and occupied more space in the growth room than did detached leaf segments.

The interaction between single conidial isolates and the host plant lines

The first single conidial isolate obtained from the leaves of plant line 1b, was tested for virulence on each of the 250 plant lines under controlled conditions and the results are given in Table 3, column 3. As can be seen, some plants were resistant while others were susceptible to varying degrees (infection types 2, 3 and 4). Most of the plants which were shown to be resistant to isolate 1, in the controlled experiments,

became, however, heavily infected with mildew in the green house when they were exposed to natural infection. This showed that other isolates of *E. fischeri* existed and isolate 2 was obtained from one such plant, plant 2c. The virulence of this isolate was also tested under controlled conditions on all plant lines and the results are given in Table 3, column 4. A further four isolates were obtained in a similar manner. Isolate 3 was obtained from plant 4b (4b was resistant to isolates 1 and 2), isolate 4 from plant 6h (6h was resistant to isolates 1, 2 and 3), isolate 5 from plant 1o (1o was resistant to isolates 2, 3 and 4) and isolate 6 from plant 4h (4h was resistant to isolates 1, 2, 3, 4 and 5); isolate 6 was however tested on only 193 plant lines. The results of controlled inoculations with these isolates are given in Table 3, columns 5, 6, 7 and 8 respectively.

Three more isolates were later selected at random, isolate 7 was obtained from the leaves of plant 15g and isolate 8 was obtained from the stems of the same plant. Isolate 9 was obtained from the stem of plant 7e. The results of test inoculating 51 plant lines with these isolates are given in Table 3, columns 9, 10 and 11 respectively.

As can be seen, isolate 9 produces infection types on the plant lines on which it was tested, similar to those produced by isolate 5. It is thus probable that isolate 9 was similar to isolate 5.

The reactions of a selection of whole plants to inoculation with isolates 1 - 6, were found to give similar results to those obtained with detached leaves.

RESULTS

The results given in Table 3 and illustrated in Plate 3 show clear differences between each of the isolates in terms of the infection types produced on each plant line. The relationship shown between the plant lines and the isolates is very similar to that which would be predicted if the interaction were based on a gene-for-gene system (Person, 1959). By applying this system, it is possible to assign resistance factors to each of the test plants with respect to their reactions to each of the 8 isolates of the parasite.

Thus each plant line resistant to isolate 1, for example, lines 1r, 2c, 2f, 2i, 2j, etc. contains a resistance factor which can be designated R_1 and plant lines resistant to isolate 2, for example, lines 1a, 1c, 1e, 2g, 2h, etc. contain a resistance factor which can be designated R_2. In a similar way, resistance factors R_3, R_4, R_5, R_6, R_7 and R_8 can be assigned to plant lines. The full assignments in terms of resistance factors are given in Table 3, column 12.

The percentage numbers of plant lines possessing each resistance factor is as follows: R_1 = 20.4%, R_2 = 22.8%, R_3 = 17.6%, R_4 = 29.6%, R_5 = 23.6%, R_6 = 31.09%, R_7 = 31.4%, and R_8 = 13.7%. As can be seen R_7 is the commonest and R_8 the least common resistance factor.

Virulence factors

Having assigned resistance factors to each of the 250 plant lines, it is possible to identify the matching virulence factors present in the 8 fungal isolates. Thus, as can be seen from Table 3, isolate 1 is virulent

on plants containing all resistance factors except the R_1 factor. It is therefore a complex race containing virulence factors matching all but one resistance factor. In the same way all the other isolates can be shown to be complex races having virulence factors matching all but one of the resistance factors. The virulence and avirulence factors possessed by each of the 8 isolates are listed in Table 4.

TABLE 3

The interactions between isolates 1 - 9 of E. fischeri and plant lines of S. vulgaris, giving infection types 0, 1, 2, 3 and 4.

Column 1	2	3	4	5	6	7	8	9	10	11	12
Plant line	Collection area	Isolates									Resistance factors
		1	2	3	4	5	6	7	8	9	
1a	Glasgow	4	0	4	4	3	4				R_2
b		4	4	4	4	4	4	4	4	4	
c		4	0	4	4	4	4	0	4	4	R_2R_7
d		4	4	3	4	4	4				
e		4	0	2	4	4	4				R_2
f		4	4	4	4	3	3				
g		4	4	2	4	4	0				R_6
h		4	4	3	4	1	4				R_5
i		4	4	4	4	3	4				
j		4	4	4	4	3	4				
k		4	4	2	0	2	4				R_4
l		3	4	3	3	4	0				R_6
m		3	0	3	0	3	3	0	2	4	$R_2R_4R_7$
n		3	0	3	4	2	0				R_2R_6
o		3	0	0	0	4	0				$R_2R_3R_4R_6$
p		4	4	4	4	4	4				
q		4	4	3	3	2	4				
r		0	4	4	0	4	4				R_1R_4
s		3	0	4	4	4	0				R_2R_6
2a	Crail	4	1	4	4	4	3				R_2
b		4	3	4	3	0	2				R_5
c		0	3	4	0	0	0	4	4	0	$R_1R_4R_5R_6$
d		4	4	3	2	2	4	4	4	3	
e		4	4	4	2	3	2	2	4	4	
f		0	4	4	0	0	3				$R_1R_4R_5$
g		4	0	3	3	3	2				R_2
h		4	1	4	3	4	4				R_2
i		0	3	2	0	0	0				$R_1R_4R_5R_6$
j		0	4	4	0	0	0				$R_1R_4R_5R_6$
3a	Ayr	4	4	4	3	3	4				
b		4	4	4	3	2	3				
c		4	4	4	4	2	3				
d		4	4	4	3	3	4				
e		4	4	4	4	2	4				
f		4	4	3	0	2	3	2	3	2	R_4
g		4	4	4	3	2	3	2	3	3	
h		4	4	4	4	4	4				
i		4	3	4	4	3	4	0	1	4	R_7R_8
j		4	0	4	4	4	4				R_2

Column 1	2	3	4	5	6	7	8	9	10	11	12
Plant line	Collection area	Isolates									Resistance factors
		1	2	3	4	5	6	7	8	9	
4a	Dublin	4	2	2	4	0	4	3	3	0	R_5
b		0	2	3	3	0	4				R_1R_5
c		3	2	2	0	0	2				R_4R_5
d		3	3	4	2	1	4				R_5
e		4	3	1	0	2	2				R_3R_4
f		4	3	3	4	4	3				
g		4	2	0	4	4	2				R_3
h		0	0	0	0	0	4	0	0	0	$R_1R_2R_3R_4R_5$ R_7R_8
i		2	4	2	4	4	4				
j		0	3	4	3	4	4	4	4	4	R_1
5a	Far Sawrey	2	3	4	3	3	4	2	3	2	
b		3	3	3	4	4	4				
c		3	3	4	4	4	4				
d		2	2	1	4	4	4	2	4	4	R_3
e		3	1	3	4	4	3				R_2
f		4	2	1	4	4	4				R_3
g		4	3	3	2	1	0				R_5R_6
h		4	4	3	3	4	4				
i		4	4	2	4	4	1				R_6
j		2	1	2	0	2	4	2	4	3	R_2R_4
6a	Ulverston	3	3	4	4	2	3	0	4	2	R_7
b		0	0	0	0	0	2	0	0	0	$R_1R_2R_3R_4R_5R_7R_8$
c		4	0	0	0	3	4				$R_2R_3R_4$
d		0	0	4	0	3	0	0	4	3	$R_1R_2R_4R_6R_7$
e		0	4	4	4	4	4				R_1
f		4	0	3	4	4	0				R_2R_6
g		4	4	3	4	4	4				
h		0	0	0	4	0	0				$R_1R_2R_3R_5R_6$
i	Seeds not viable										
j		4	4	0	4	3	0				R_3R_6
7a	Coniston	4	3	4	3	4	3	0	2	4	R_7
b		0	0	0	0	0	0	0	0	0	$R_1R_2R_3R_4R_5$ $R_6R_7R_8$
c		4	4	4	4	4	4	3	2	4	
d		0	0	4	0	0	0				$R_1R_2R_4R_5R_6$
e		3	4	4	4	4	4				
f		4	4	0	3	3	3				R_3
g		4	4	4	4	4	4				
h		4	4	3	4	4	4				
i		4	4	4	4	4	4	4	4	4	
j		4	4	4	3	2	4				

Column 1	2	3	4	5	6	7	8	9	10	11	12
Plant line	Collection area	Isolates 1	2	3	4	5	6	7	8	9	Resistance factors
8a	Wellesbourne	4	4	4	4	4	2	4	4	4	
b		4	4	4	2	2	4				
c		4	4	4	0	4	4				R_4
d		4	4	3	4	3	4				
e		4	3	2	4	4	4				
f		3	2	4	4	4	4				
g		3	3	4	4	3	3	4	4	4	
h		0	0	0	0	0	0				$R_1R_2R_3R_4R_5R_6$
i		1	4	2	2	4	3				R_1
j		2	4	2	4	4	3				
k		4	3	4	4	4	4				
9a	Perth	0	1	0	0	0	0	4	4	0	$R_1R_2R_3R_4R_5R_6$
b		0	4	4	0	0	2	3	4	0	$R_1R_4R_5$
c		0	4	3	0	0	0				$R_1R_4R_5R_6$
d		0	4	4	0	0	3				$R_1R_4R_5$
e		0	2	3	2	0	0				$R_1R_5R_6$
f		0	4	4	0	0	0				$R_1R_4R_5R_6$
g		2	1	0	0	0	1	2	0	0	$R_2R_3R_4R_5R_6R_8$
h		0	2	1	4	3	3				R_1R_3
i		0	1	2	0	0	0				$R_1R_2R_4R_5R_6$
j		0	2	3	0	0	0				$R_1R_4R_5R_6$
10a	Stranraer	1	1	4	4	2	4				R_1R_2
b		3	2	3	2	2	3	3	2	3	
c		4	4	2	4	4	3				
d		4	2	4	3	3	3				
e		3	3	4	4	4	2				
f		3	4	4	3	4	2	4	4	3	
g		4	0	2	0	4	0				$R_2R_4R_6$
h		2	1	2	0	3	0				$R_2R_4R_6$
i		2	2	4	1	4	2				R_4
j		0	0	0	0	0	0	0	0	0	$R_1R_2R_3R_4R_5R_6$ R_7R_8
11a	Abington	1	2	1	4	4	4				R_1R_3
b		2	2	4	4	2	4				
c		2	4	2	4	4	4				
d		2	2	4	2	2	3	4	4	3	
e		4	0	3	4	3	4	0	2	2	R_2R_7
f		2	2	4	4	3	2				
g		0	4	4	0	0	1				$R_1R_4R_5R_6$
h		0	4	2	0	0	2				$R_1R_4R_5$
i		0	4	4	0	0	0	4	4	0	$R_1R_4R_5R_6$
j		0	4	4	0	0	0				$R_1R_4R_5R_6$

1	2	3	4	5	6	7	8	9	10	11	12
Plant line	Collection area	Isolates									Resistance factors
		1	2	3	4	5	6	7	8	9	
12a	Peebles	4	4	4	4	4	4				
b		4	4	4	4	4	4				
c		4	3	4	2	2	3				
d		4	4	4	4	4	4				
e		0	0	4	0	2	1	4	3	3	$R_1R_2R_4R_6$
f		4	4	4	0	4	4				R_4
g		2	3	0	0	3	0				$R_3R_4R_6$
h		4	4	4	4	4	4				
i		0	1	0	0	1	0				$R_1R_2R_3R_4R_5R_6$
j		4	3	0	0	0	3	4	2	0	$R_3R_4R_5$
13a	Hawick	4	4	4	0	0	0	4	4	0	$R_4R_5R_6$
b		4	0	3	4	4	4				R_2
c		4	2	4	2	4	0				R_6
d		4	4	4	0	4	4				R_4
e		4	2	4	4	4	4	3	0	4	R_8
f		4	4	4	4	4	0				R_6
g		4	4	0	4	4	4				R_3
h		3	3	4	3	4	0				R_6
i		4	4	4	4	4	0				R_6
j		4	4	4	4	3	0	0	2	4	R_6R_7
14a	Dumfries	4	3	4	4	3	4				
b		4	3	0	0	0	0				$R_3R_4R_5R_6$
c		4	0	4	0	4	0	0	2	3	$R_2R_4R_6R_7$
d		4	0	4	3	4	0				R_2R_6
e		4	4	4	4	4	4	4	4	4	
f		4	2	4	0	4	0				R_4R_6
g		4	4	4	4	4	4				
h		4	1	4	0	3	0				$R_2R_4R_6$
i		2	4	3	4	0	0				R_5R_6
j		0	4	2	2	4	0				R_1R_6
15a	Aberdeen	4	4	4	0	0	4	0	4	0	$R_4R_5R_7$
b		4	4	4	4	3	4				
c		4	4	4	4	3	4				
d		3	0	0	0	0	0				$R_2R_3R_4R_5R_6$
e		4	4	3	4	4	4	4	3	4	
f		4	3	4	0	4	4				R_4
g		2	4	0	0	0	0				$R_3R_4R_5R_6$
h		2	4	4	2	4	4				
i		2	2	0	0	0	4				$R_3R_4R_5$
j		4	4	4	4	4	4				

1	2	3	4	5	6	7	8	9	10	11	12
Plant line	Collection area	Isolates 1	2	3	4	5	6	7	8	9	Resistance factors
16a	Pitlochry	1	2	0	0	0	0				$R_1R_3R_4R_5R_6$
b		4	2	4	4	4	3				
c		2	2	0	0	0	0				$R_3R_4R_5R_6$
d		4	3	4	4	4	0				R_6
e		2	4	4	0	4	1				R_4R_6
f		2	0	2	0	0	0	2	0	0	$R_2R_4R_5R_6R_8$
g		3	0	4	3	1	3	0	2	1	$R_2R_5R_7$
h		2	4	4	4	2	0				R_6
i		0	0	3	3	0	0				$R_1R_2R_5R_6$
j		3	0	4	0	4	0	3	4	3	$R_2R_4R_6$
17a	Kingussie	4	3	4	4	4	4				
b		0	0	0	0	0	3				$R_1R_2R_3R_4R_5$
c		4	4	0	0	4	4				R_3R_4
d		4	4	4	4	4	4				
e		4	3	4	4	4	4				
f		2	1	4	4	1	4				R_2R_5
g		4	4	4	4	4	4	4	4	4	
h		4	2	0	0	4	0				$R_3R_4R_6$
i		4	2	4	4	4	3	4	4	4	
j		2	2	4	2	3	1				R_6
18a	Inverness	4	4	4	4	4	4				
b		4	0	0	0	4	4				$R_2R_3R_4$
c		4	4	3	0	4	4				R_4
d		4	4	4	4	4	4	4	3	4	
e		4	4	4	4	4	4				
f		4	4	4	2	4	0	4	4	4	R_6
g		4	4	3	4	2	4				
h		1	0	4	4	3	4				R_1R_2
i		4	1	4	2	4	0				R_2R_6
j		2	2	3	4	2	3				
19a	Lairg	Seeds not viable									
b		4	3	4	4	1	4	4	4	0	R_5
c		Seeds not viable									
d		4	4	4	4	4	4				
e		2	3	2	4	2	3				
f		3	4	4	4	4	4	4	4	4	
20a	Portree	4	4	4	4	2					
b		3	4	4	4	3					
c		2	2	2	1	4					R_4
d		2	2	2	1	3					R_4
e		1	1	2	2	2					R_1R_2
f		2	4	4	2	4					
g		2	2	2	2	2					
h		0	4	4	1	0					$R_1R_4R_5$
i		0	2	4	4	2					R_1
j		3	3	3	3	4					

column 1	2	3	4	5	6	7	12
Plant line	Collection area	Isolates					Resistance factors
		1	2	3	4	5	
21a	Mallaig	4	4	4	3	4	
b		3	4	0	4	2	R_3
c	Seeds not viable						
d		4	3	4	4	2	
e		4	4	3	4	4	
f		2	3	2	4	3	
g		2	4	4	4	2	
h		4	4	2	3	4	
i		2	2	2	2	2	
j		4	4	1	4	4	R_3
22a	Fort William	4	2	4	4	4	
b		2	0	3	3	1	R_2R_5
c		4	1	2	0	0	$R_2R_4R_5$
d		4	2	2	4	4	
e		2	2	2	3	4	
f		2	4	2	2	2	
g		4	2	4	4	2	
h		2	2	2	2	4	
i		3	0	2	1	2	R_2R_4
j		4	4	4	4	4	
23a	Crianlarich	2	4	1	3	2	R_3
b		0	0	0	0	0	$R_1R_2R_3R_4R_5$
c		2	3	4	3	4	
d		4	4	4	4	4	
e		2	2	0	3	0	R_3R_5
f		1	3	1	3	0	$R_1R_3R_5$
g		3	2	0	3	0	R_3R_5
h		0	0	0	1	0	$R_1R_2R_3R_4R_5$
i		0	0	3	0	3	$R_1R_2R_4$
j	Seeds not viable						
24a	Oban	4	2	2	2	2	
b	Seeds not viable						
c		1	4	2	3	4	R_1
d		0	2	2	2	0	R_1R_5
e		2	4	0	2	2	R_3
f		1	0	1	0	2	$R_1R_2R_3R_4$
g		4	0	1	0	2	$R_2R_3R_4$
h		2	2	2	2	2	
i		4	0	4	4	4	R_2
j		3	4	4	4	4	

column 1	2	3	4	5	6	7	12
Plant line	Collection area	Isolates 1	2	3	4	5	Resistance factors
25a	Stirling	0	4	0	0	0	$R_1R_3R_4R_5$
b		4	4	4	4	4	
c		3	4	4	4	2	
d		2	2	2	2	2	
e		4	3	4	2	2	
f		3	3	3	3	2	
g		4	2	3	4	4	
h		4	4	2	4	4	
i		4	2	3	4	4	
j		0	0	3	0	0	$R_1R_2R_4R_5$

PLATE 3

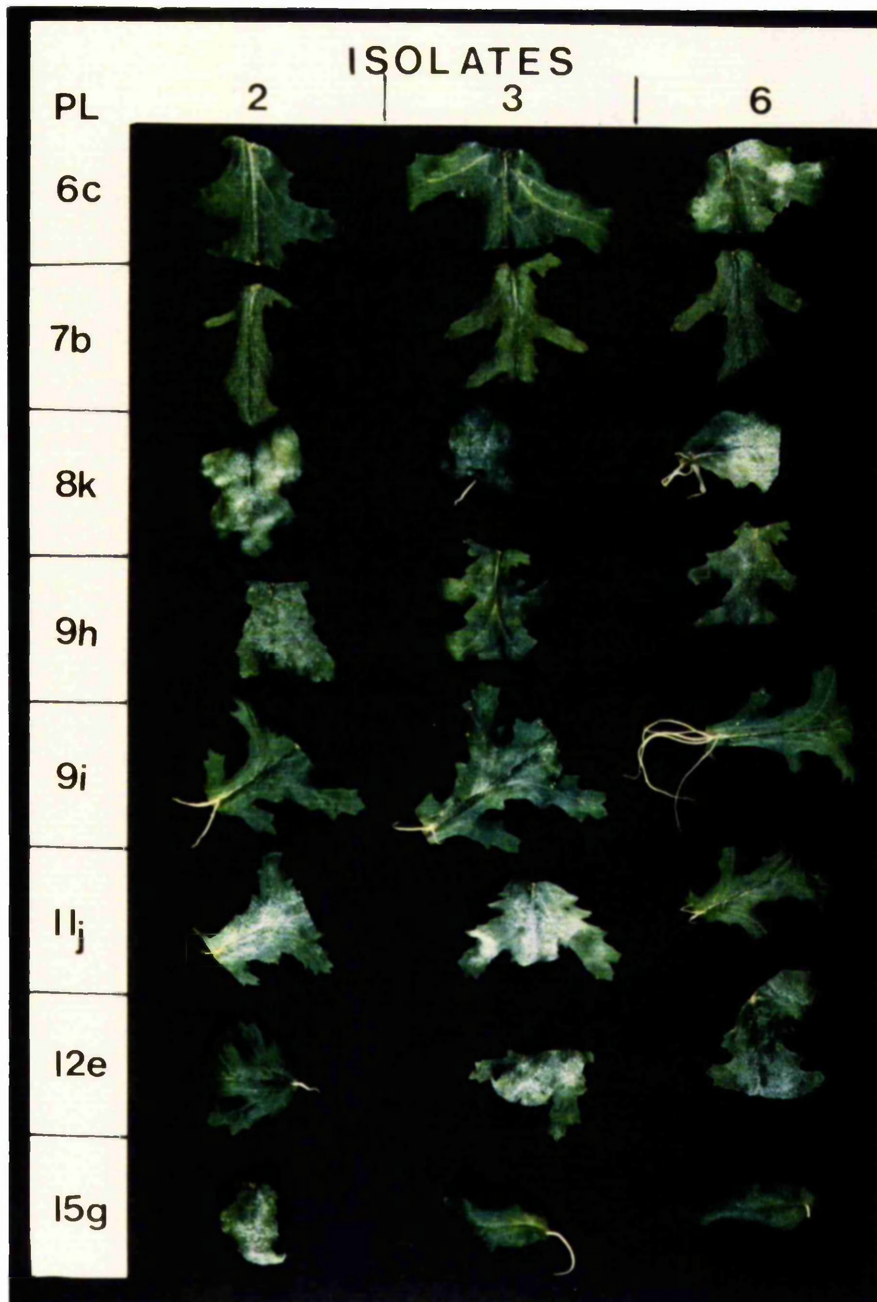


PLATE 3: Differential reactions between three mildew isolates and eight plant lines.

TABLE 4

Virulence and avirulence factors in isolates 1 - 8

Isolate 1	=	A_1	V_2	V_3	V_4	V_5	V_6	V_7	V_8
Isolate 2	=	V_1	A_2	V_3	V_4	V_5	V_6	V_7	V_8
Isolate 3	=	V_1	V_2	A_3	V_4	V_5	V_6	V_7	V_8
Isolate 4	=	V_1	V_2	V_3	A_4	V_5	V_6	V_7	V_8
Isolate 5	=	V_1	V_2	V_3	V_4	A_5	V_6	V_7	V_8
Isolate 6	=	V_1	V_2	V_3	V_4	V_5	A_6	V_7	V_8
Isolate 7	=	V_1	V_2	V_3	V_4	V_5	V_6	A_7	V_8
Isolate 8	=	V_1	V_2	V_3	V_4	V_5	V_6	V_7	A_8

Where, A = avirulence factor

V = virulence factor

Following the system of nomenclature for *Phytophthora infestans* (Black, 1952), each isolate can be named after the resistance factors it is able to overcome.

Thus,
Isolate 1 is race 2345678
Isolate 2 is race 1345678
Isolate 3 is race 1245678
Isolate 4 is race 1235678
Isolate 5 is race 1234678
Isolate 6 is race 1234578
Isolate 7 is race 1234568
Isolate 8 is race 1234567

The isolates are thus complex races, each possessing one avirulent factor only. No isolates having virulence factors for all resistance factors have so far been found.

Comparative studies on the aggressiveness of isolates 1 - 6

Table 5 shows the percentage number of plant lines which are susceptible to each isolate, this being expressed by production of infection types 2, 3 or 4. An analysis of variance showed that there was no significant difference in aggressiveness between isolates. However, there was a significant difference for all isolates, in the numbers of plants expressing each infection type with infection type four being the commonest for all. Thus, when infection occurs, selection appears to favour the most virulent, condition of the parasite.

TABLE 5

Percentage number of plants expressing infection types 2, 3 or 4 when inoculated with isolates 1 - 6.

Isolates	Infection Types			Total No. of Plants
	2	3	4	
1	21.11	15.07	63.82	199
2	23.83	19.17	56.99	193
3	19.42	18.93	61.65	206
4	15.91	19.32	64.77	176
5	23.56	17.80	58.64	191
6	10.53	22.56	66.92	133

The distribution of the resistance factors

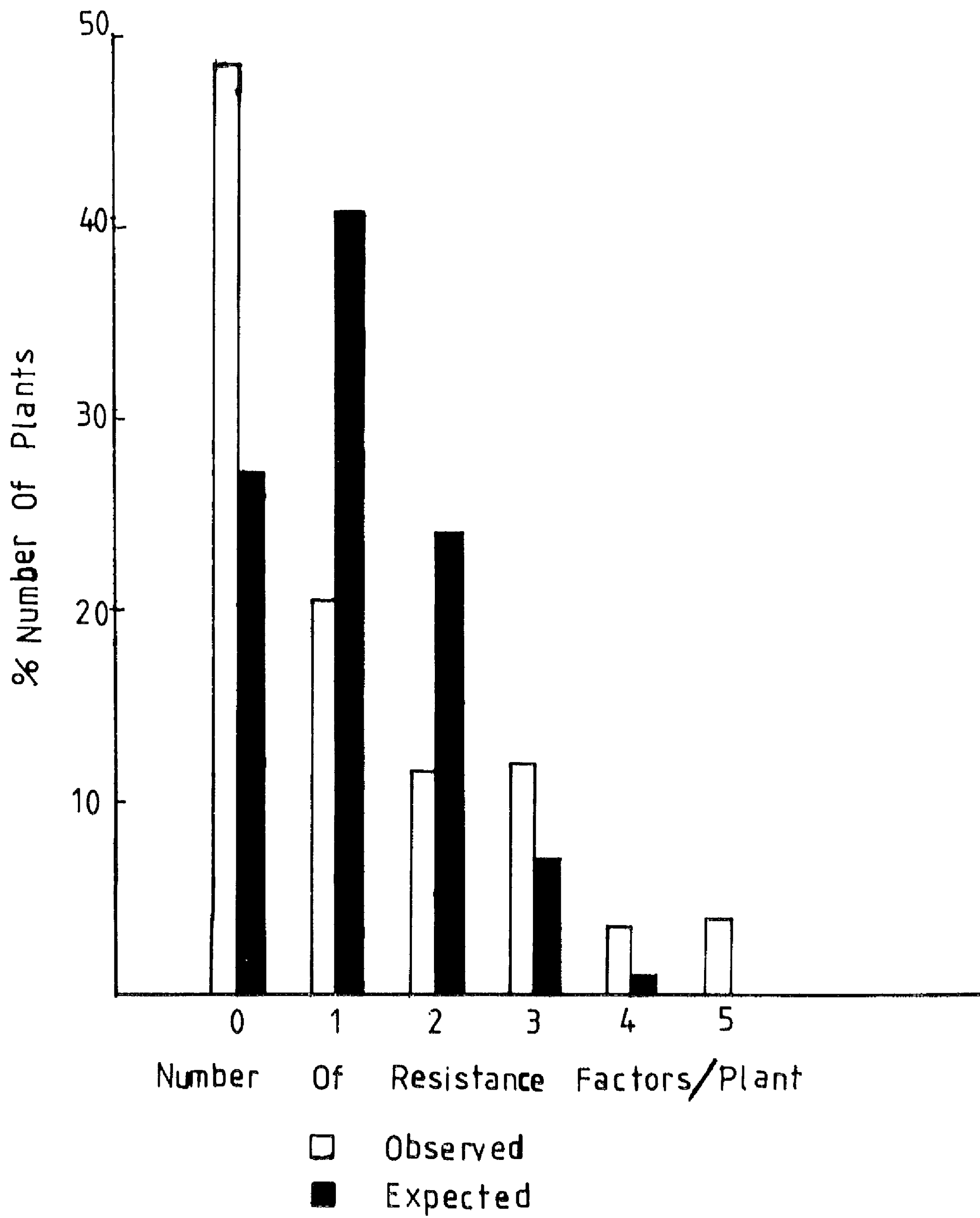
Of the 8 isolates, only isolates 1 - 5 have been tested on all 250 plant lines and so an analysis of the distribution of resistance factors within the host population has been considered for these isolates. The data on which this analysis is based, is given in the Appendix, Table 3a.

Knowing the frequency with which the individual resistance factors occur in the population, it is possible to predict the frequency with which plants possessing none, one only or combinations of the different resistance factors would occur, if selection favours no particular combination. Any significant deviation from the expected frequency would indicate the operation of selection pressures favouring or not favouring particular combinations (Sinnott et al., 1958). The observed and expected numbers of the different combinations are given in Table 6a and are expressed as a percentage of the total number of plants in Figure 2.

As can be seen, plant lines without resistance factors and those possessing 3 or more resistance factors are more common than expected, while those possessing one or two resistance factors are less common than expected. These differences are highly significant ($P < 0.001$). It would thus appear that selection pressures, presumably imposed by the parasite population, favour the survival of plant lines possessing none or combinations of 3 or more resistance factors.

In order to determine if the selection pressures favouring the survival of different combinations of resistance factors operated at different geographical sites, the total population (Appendix, Table 3a)

FIG. 2.



The percentage number of observed and expected plant lines containing 0, 1, 2, 3, 4 or 5 resistance factors.

TABLE 6a

The relative numbers of plant lines possessing resistance factors 0; R_1 - R_5 and their different combinations in the total host population.

Resistance factors	Observed No. of plants	Expected* No. of plants	X^2 Contribution
0	121	68.0869	41.1142
R_1	6	17.4494	7.5130
R_2	16	20.1086	0.8400
R_3	10	14.5428	1.4176
R_4	12	28.6274	9.6595
R_5	7	21.0321	9.3619
R_1R_2	3	5.1534	
R_1R_3	2	3.7271	
R_1R_4	1	7.3361	
R_1R_5	3	5.3901	
R_2R_3	0	4.2950	
R_2R_4	8	8.4547	
R_2R_5	3	6.2115	
R_3R_4	4	6.1146	
R_3R_5	2	4.4923	
R_4R_5	3	8.8430	
	29	60.0184	16.0308
$R_1R_2R_3$	0	1.1007	
$R_1R_2R_4$	3	2.1668	
$R_1R_2R_5$	1	1.5919	
$R_1R_3R_4$	0	1.5671	
$R_1R_3R_5$	1	1.1513	
$R_1R_4R_5$	14	2.2663	
$R_2R_3R_4$	4	1.8059	
$R_2R_3R_5$	0	1.3267	
$R_2R_4R_5$	2	2.6117	
$R_3R_4R_5$	5	1.8888	
	30	17.4772)	
$R_1R_2R_3R_4$	1	0.4628)	
$R_1R_2R_3R_5$	1	0.3400)	
$R_1R_2R_4R_5$	3	0.6693)	41.3840
$R_1R_3R_4R_5$	2	0.4841)	
$R_2R_3R_4R_5$	2	0.5578)	
	9	2.5140)	
$R_1R_2R_3R_4R_5$	10	0.1430)	
TOTAL	250	249.9995	$X^2_{(2)}$ = 127.3190

* The expected number of plants was calculated from the observed frequencies of the resistance factors: R_1 = 0.2040; R_2 = 0.2280; R_3 = 0.1760; R_4 = 0.2960; R_5 = 0.2360; 0 = 0.2723.

TABLES 6b and 6c

The relative numbers of plants possessing resistance factors 0; R_1 - R_5 and their different combinations in the Coastal (Table 6b) and Inland (Table 6c) areas of sample collection.

Resistance factors	Coastal Area			Inland Area		
	Observed No. of plants	Expected* No. of plants	X^2 Contribution	Observed No. of plants	Expected** No. of plants	X^2 Contribution
0	61	40.8999	9.8781	60	27.8571	37.0881
R_1	4	8.1819	2.1374	2	8.8888	5.3388
R_2	12	13.9257	0.2663	4	7.0341	1.3087
R_3	5	6.8190	0.4852	5	7.3917	0.7739
R_4	7	14.5125	3.8889	5	13.7579	5.5750
R_5	4	8.6517	2.5010	3	11.8480	6.6076
	32	52.0908		19	48.9205	
R_1R_2	3	2.7858		0	2.2445	
R_1R_3	0	1.3641		2	2.3586	
R_1R_4	1	2.9032		0	4.3900	
R_1R_5	2	1.7308		1	3.7805	
R_2R_3	0	2.3218		0	1.8664	
R_2R_4	4	4.9412		4	3.4739	
R_2R_5	1	2.9457		2	2.9917	
R_3R_4	1	2.4196		3	3.6506	
R_3R_5	0	1.4425		2	3.1438	
R_4R_5	2	3.0699		1	5.8514	
	14	25.9246	5.4850	15	33.7514	10.4178

Resistance factors	Coastal Area			Inland Area		
	Observed No. of plants	Expected* No. of plants	X^2 Contribution	Observed No. of plants	Expected** No. of plants	X^2 Contribution
$R_1R_2R_3$	0	0.4645		0	0.5956	
$R_1R_2R_4$	1	0.9885		2	1.1085	
$R_1R_2R_5$	0	0.5893		1	0.9546	
$R_1R_3R_4$	0	0.4840		0	1.1648	
$R_1R_3R_5$	0	0.2886		1	1.0031	
$R_1R_4R_5$	5	0.6141		9	1.8671	
$R_2R_3R_4$	4	0.8238		0	0.9218	
$R_2R_3R_5$	0	0.4911		0	0.7938	
$R_2R_4R_5$	1	1.0452		1	1.4775	
$R_3R_4R_5$	2	0.5118		3	1.5526	
	13	6.3009)		17	11.4394)	
$R_1R_2R_3R_4$	1	0.1648)		0	0.2941)	
$R_1R_2R_3R_5$	1	0.0982)		0	0.2533)	
$R_1R_2R_4R_5$	0	0.2091)	20.040	3	0.4715)	20.2832
$R_1R_3R_4R_5$	0	0.1024)		2	0.4954)	
$R_2R_3R_4R_5$	1	0.1743)		1	0.3920)	
	3	0.7488)		6	1.9060)	
$R_1R_2R_3R_4R_5$	3	0.0349)		7	0.1251)	
TOTAL	126	125.9979	$X^2_{(2)} = 44.6822$	124	123.9998	$X^2_{(2)} = 87.3931$

* Expected number of plants was calculated from the observed frequencies of the resistance factors: $R_1 = 0.1667$; $R_2 = 0.2540$; $R_3 = 0.1429$; $R_4 = 0.2619$; $R_5 = 0.1746$; $0 = 0.3298$.

** Expected number of plants calculated from the observed frequencies of the resistance factors: $R_1 = 0.2419$; $R_2 = 0.2016$; $R_3 = 0.2097$; $R_4 = 0.3306$; $R_5 = 0.2984$; $0 = 0.2246$.

TABLES 6d and 6e

The relative numbers of plants possessing resistance factors 0; R_1 - R_5 and their different combinations in the Northern (Table 6d) and Southern (Table 6e) areas of sample collection.

Resistance factors	Northern Area			Southern Area		
	Observed No. of plants	Expected No. of plants	X^2 Contribution	Observed No. of plants	Expected** No. of plants	X^2 Contribution
0	49	23.28	28.4157	62	39.4731	12.8559
R_1	2	7.48	7.0148	2	7.5747	4.1028
R_2	5	7.12	0.6312	11	12.8646	0.2703
R_3	4	6.08	0.7116	5	6.6457	0.4753
R_4	5	11.64	3.7878	6	14.6887	5.1396
R_5	2	10.72	7.0931	3	7.1028	2.3699
1R	18	43.04		27	48.8766	
R_1R_2	2	2.2881		1	2.4687	
R_1R_3	1	1.9553		1	1.2753	
R_1R_4	0	3.7402		1	2.8187	
R_1R_5	2	3.4452		0	1.3630	
R_2R_3	0	1.8608		0	2.1659	
R_2R_4	2	3.5595		6	4.7872	
R_2R_5	3	3.2788		0	2.3149	
R_3R_4	2	3.0419		1	2.4730	
R_3R_5	2	2.8020		0	1.1958	
R_4R_5	1	5.3597		1	2.6431	
	15	31.3316	8.5128	11	23.5056	6.6533

Resistance factors	Northern Area			Southern Area		
	Observed No. of plants	Expected No. of plants	X^2 Contribution	Observed No. of plants	Expected No. of plants	X^2 Contribution
$R_1R_2R_3$	0	0.5980		0	0.4156	
$R_1R_2R_4$	1	1.1439		2	0.9186	
$R_1R_2R_5$	1	1.0536		0	0.4442	
$R_1R_3R_4$	0	0.9775		0	0.4746	
$R_1R_3R_5$	1	0.9004		0	0.2295	
$R_1R_4R_5$	10	1.7224		4	0.5072	
$R_2R_3R_4$	2	0.9303		2	0.8060	
$R_2R_3R_5$	0	0.8569		0	0.3897	
$R_2R_4R_5$	2	1.6391		0	0.8614	
$R_3R_4R_5$	3	1.4008		2	0.4450	
	20	11.2229)		10	5.4918)	
$R_1R_2R_3R_4$	1	0.2989)		0	0.1547)	
$R_1R_2R_3R_5$	0	0.2754)		1	0.0748)	
$R_1R_2R_4R_5$	1	0.5267)	18.3827	2	0.1653)	22.8723
$R_1R_3R_4R_5$	1	0.4501)		1	0.0854)	
$R_2R_3R_4R_5$	2	0.4284)		0	0.1450)	
	5	1.9796)		4	0.6252)	
$R_1R_2R_3R_4R_5$	4	0.1377)		4	0.0278)	
TOTAL	111	110.9918	$X^2_{(2)} = 71.5497$	118	118.0001	$X^2_{(2)} = 54.6715$

* Expected number of plants was calculated from the observed frequencies of the resistance factors: $R_1 = 0.2432$; $R_2 = 0.2342$; $R_3 = 0.2072$; $R_4 = 0.3333$; $R_5 = 0.3153$; $0 = 0.2097$.

** Expected number of plants was calculated from the observed frequencies of the resistance factors: $R_1 = 0.1610$; $R_2 = 0.2458$; $R_3 = 0.1441$; $R_4 = 0.2712$; $R_5 = 0.1525$; $0 = 0.3345$.

was arbitrarily divided into two and the two halves compared. The first division was into coastal and inland collection sites (Appendix, Tables 3b and 3c) and the second was into collection sites North and South of Stirling (Appendix, Tables 3d and 3e). The samples collected from Dublin and Wellesbourne were excluded from the North versus South analysis because it was considered that they were too far away from the rest of the collection sites.

The results of both analyses are summarised in Tables 6b - 6e. The mean numbers of plants possessing 0 or R_1, R_2, R_3, R_4 or R_5 resistance factors were compared between sub-populations, using a T test. None of the differences were found to be significant. The distribution of two or more resistance factors was also found to be similar in these areas.

However, in all four sub-populations plant lines possessing no resistance factors, or 3 or more resistance factors were more common than we expected while those possessing one only or two resistance factors occurred less frequently than expected. Thus the population structure of these sub-populations mimics that of the total plant population.

Host Differentials

The advantages of host differentials is that they can be used to obtain all of the isolates used here for any further work that may be necessary.

Thus, isolate 1 may be obtained from plant line 15d because

plant line 15d possesses resistance factors against isolates 2, 3, 4, 5 or 6. Similarly, isolates 2, 3, 4 or 5 may be obtained from plant lines 16a, 9i, 6h or 17b respectively, as each plant line is only susceptible to its respective isolate.

However, isolate 1 or 5 may be obtained from plant line 1o, since plant line 1o possesses resistance factors against isolates 2, 3, 4 and 6. These isolates may be distinguished by testing the isolate thus obtained on plant line 15d. A resistance factor in plant line 15d would indicate that the isolate is probably 5, whereas susceptibility would indicate isolate 1.

Isolates 1, 2, 3, 4 or 5 may be tested on plant line 9a to distinguish them from isolates 7 or 8. Since plant lines 15d, 1o, 16a, 9i and 6h were not tested with isolate 7 or 8, it is possible that some or all of these plant lines may be susceptible to one or both isolates. Resistance in plant 9a to isolates 1, 2, 3, 4 or 5 would confirm their presence, but susceptibility would indicate isolate 7 or 8.

Isolate 1 or 7 could be obtained from plant line 9g, but the two isolates may be distinguished by testing the isolate thus obtained on plant line 9a. A resistance factor in plant 9a would indicate that the isolate is probably 1, but susceptibility may indicate isolate 7.

Finally, the isolate obtained from plant line 9a could be isolate 7 or 8 since isolates 1 - 6 are avirulent on plant line 9a. To distinguish between isolates 7 and 8, the isolate thus obtained should be tested on plant line 9g. Susceptibility to this isolate may indicate isolate 7, but a resistance factor against it would indicate isolate 8.

Any of these isolates could then be tested on plant lines 7b, 8h or 10j because these plant lines possess resistance factors against isolates 1 - 8. Susceptibility to any of the eight isolates may indicate an entirely new race, but resistance factors against them may confirm their presence. All the host differentials and their reaction type to these isolates are listed in Table 7.

TABLE 7

Host differentials and the reaction types obtained by their inoculation with isolates 1 - 8.

Host Differentials	Isolates 1	2	3	4	5	6	7	8
15d	S	R	R	R	R	R	-	-
16a	R	S	R	R	R	R	-	-
9i	R	R	S	R	R	R	-	-
6h	R	R	R	S	R	R	-	-
1o	S	R	R	R	S	R	-	-
17b	R	R	R	R	R	S	R	R
9g	S	R	R	R	R	R	S	R
9a	R	R	R	R	R	R	S	S
8h or 7b or 10j	R	R	R	R	R	R	R	R

Where, S = susceptibility

R = resistance

\- = not tested

DISCUSSION

The differential reactions between 250 plant lines of S. vulgaris and 8 isolates of E. fischeri demonstrate the existence of resistance factors in the host which are matched by virulence factors in the parasite. The interactions between the host plant line and particular isolates of the parasite are similar to those which would be predicted with a gene-for-gene system (Person, 1959).

The reaction of seedlings to infection was basically the same as that of the adult plant, indicating that resistance in the seedling and in the adult plant is probably conditioned by the same resistance factors. The only noticeable difference was that infection in the seedling stage reduced subsequent growth and development to a greater extent than was the case in the adult plant. In this respect, the reactions of groundsel to infection were similar to those of many crop plants. For example, genes Ml_t, Ml_u, Ml_c and Ml_a in Triticum vulgare, have been shown to confer resistance to Erysiphe graminis f. sp. tritici in both the seedling and adult plant stages, (Pugsley and Carter, 1954).

All the 250 plant lines were tested with 5 out of the 8 isolates of the parasite and out of the 32 different combinations of resistance factors expected, 28 were found. The missing 4 can be attributed to the smallness of the sample size.

The most numerous phenotype was that without resistance factors to any of the five isolates and they made up 48.4% of the 250 plants tested. Those possessing only one resistance factor accounted for 20.4% and

those possessing 2, 3, 4 or all 5 resistance factors accounted for 11.6, 12.0, 3.6 or 4.0% respectively. The population structures of the plants sampled from the sub-areas (Coastal and Inland or North and South, Appendix, Tables 3b - 3e) are similar to that exhibited by the total plant sample (Appendix, Table 3a).

The population structure of groundsel in terms of resistance factors, thus resembles a multiline. However, the great morphological variation shown between the plant lines (See Chapter 4), clearly indicates that they are not isogenic lines and so the population is more directly equivalent to a mixed variety cropping system.

An analysis of the resistance factors in the total host population, indicates that the plant lines without resistance factors and those possessing 3 or more resistance factors were much more common than expected, while those possessing one or two resistance factors were less common than expected. It thus appears that plant lines possessing no resistance factors, detectable by the 5 isolates, and those possessing 3 or more resistance factors, have a selective advantage over those possessing ~~3 or more resistance factors, have a selective advantage over those possessing~~ one or two resistance factors. The selection pressure leading to this structure was most likely imposed by the parasite although other environmental factors probably contributed to it.

Since nothing is known about the total mildew population in terms of its race structure and the frequencies of the individual races, one can only speculate about the selection pressures that may be involved. There

are two possible explanations for the selective advantage of plants without resistance factors to the five isolates of the parasite. Firstly, these plant lines may possess resistance factors against several other races about which nothing is known. If this is true, then these plant lines are not likely to be selected against. Secondly, if such plant lines have high levels of tolerance to infection, then selection pressures exerted by the parasite may not be very great, in which case plants without resistance factors would not be at a great disadvantage compared to those with resistance factors. The prevalence of plant lines possessing 3 or more resistance factors, however, could be due to the fact that each plant line would be attacked by fewer races of the parasite and so their survival rate might be higher.

A total of 9 isolates of E. fischeri were obtained and an analysis of the virulence factors possessed by each, revealed 8 different races, each possessing virulence factors matching all but one of the 8 resistance factors. Thus, the 8 races are complex races with avirulence for one resistance factor only. The total number of different races in the Glasgow population of the mildew is not known, but clearly it is greater than 8 and since out of 9 isolates obtained only one was similar to any of the others, it may be fairly extensive.

Over half of the plants susceptible to each race gave a reaction type 4 to that race, with only about 20% falling into each of the other two categories (infection types 2 and 3). Thus selection appears to favour the most virulent condition in the parasite, presumably because the host plant is tolerant of high levels of infection as has been shown by Ben-Kalio

(1976) and confirmed in Part II, Chapter 2 of this thesis. It would appear that low levels of infection, i.e. infection types 2 or 3 are selected against.

Analysis of variance revealed that there was no significant difference in aggressiveness between the 5 races with which the detailed study was carried out. It has been suggested that the accumulation of genes for virulence in single races of a pathogen might result in decreased fitness or aggressiveness of these races (Van der Plank, 1963; Leonard, 1969). This investigation, however, provides no support for this view.

Thus natural selection in the parasite, presumably as the result of selection pressures exerted by the host population, has led to the development of a number of complex races, each of which can infect a large proportion of the host population within a given area. Similarly, natural selection in the host as the result of selection pressures exerted by the parasite population, has led to the development of populations containing plant lines possessing complex resistance factors (3 or more), and also to the development of high levels of tolerance.

These results thus indicate that multilines or mixed varieties when used to control plant diseases, are likely to give only a short term protection against their various pathogens. In the long term, selection in the parasite population is likely to give rise to highly complex races which are able to attack most, if not all, the cultivars of the multiline or mixed variety.

CHAPTER 3 THE INTERACTIONS BETWEEN FIVE ISOLATES OF ERYSIPHE FISCHERI AND OTHER SENECIO SPECIES

MATERIALS AND METHODS

Since seed of a range of *Senecio* species became available during the course of this study it was decided that their susceptibility to *E. fischeri* be determined.

Sources of plant material

The seed of *S. squalidis* was collected from Crianlarich (Scotland); that of *S. alpinus*, *S. incanus* and *S. viscosus* was kindly donated by Dr. I. Crute of National Vegetable Research Station, Wellesbourne; and that of *S. appendiculata*, *S. buncheli* and *S. vernalis* was obtained from the Royal Botanic Gradens, Kew.

Production of plant material

All plants were raised as described in the General Methods (p. 21).

S. squalidis, *S. viscosus* and *S. vernalis* were inbred for three generations as described in the General Methods (p. 21). *S. incanus* flowered but did not set viable seed while *S. alpinus*, *S. buncheli* and *S. appendiculata* did not flower at all throughout the period of this investigation.

Inoculation procedure

Inoculations with isolates 1, 2, 3, 4 and 5 were carried out under controlled conditions as described in the General Methods, (p.34).

RESULTS

When plants of all species were exposed to natural infection in the greenhouse, only *S. incanus* and *S. vernalis* became infected.

The results of controlled inoculations with isolates 1 to 5 are given in Table 8.

As can be seen,

TABLE 8

The interaction between some *Senecio* species and isolates 1 - 5 of *Erysiphe fischeri*

Host plants	Isolates 1	2	3	4	5
	Infection Types				
Senecio squalidis	0	0	0	0	0
Senecio vernalis	4	4	4	0	4
Senecio alpinus	0	0	0	0	0
Senecio appendiculata	0	0	0	0	0
Senecio buncheli	0	0	0	0	0
Senecio incanus	4	3	2	4	3
Senecio viscosus	0	0	0	0	0

Where, Infection type 0 = resistant

Infection types 2 - 4 = susceptible

only the two species, *S. vernalis* and *S. incanus* that became infected in the greenhouse were susceptible. *S. vernalis* was susceptible to all

isolates except 4 while S. incanus was susceptible to all five isolates. Thus, S. squalidis, S. alpinus, S. appendiculata, S. buncheli and S. viscosus were resistant to each of the five isolates and probably to all other isolates which were present in the Glasgow mildew population during the period of this investigation.

DISCUSSION

In this study none of the five isolates of E. fischeri was able to infect S. alpinus, S. squalidis, S. buncheli, S. viscosus or S. appendiculata. These were also not infected when exposed to natural infection by the Glasgow mildew population over a period of about two and a half years. It may be that isolates of the parasite which could infect any of these species were not present in the mildew population during the time of the experiments. Contrary to these findings, Blumer (1967) has reported that S. alpinus and S. viscosus are host plants of Erysiphe fischeri.

Senecio vernalis was found to be susceptible to four of the five isolates of E. fischeri tested. This species was listed (Blumer, 1967) only as a probable host plant of E. fischeri. This result now confirms that it is a host plant of the groundsel powdery mildew.

Senecio incanus was found to be susceptible to all five isolates of the parasite although it was not listed as a host plant by Blumer (1967). Thus Erysiphe fischeri has a wider host range within the genus Senecio than that listed by Blumer (1967).

CHAPTER 4: CORRELATION BETWEEN MORPHOLOGICAL CHARACTERISTICS AND RESISTANCE FACTORS IN THE HOST PLANT

PREVIOUS WORK

Senecio vulgaris is a species with very varied morphological forms. Plants may be profusely branched or may have a single unbranched or little branched axis. They usually have purple (often described as red in the literature, Trow, 1912), glabrous stems, but forms with green glabrous stems also occur.

Koch (1902), recognised two forms (*Sordidus* and *Radiatus*) on the basis of floral morphology. He described *Sordidus* as one in which all florets of the inflorescence are, non-radiate and the other *Radiatus* in which the outer layer of the inflorescence has ligulate ray florets.

Trow (1912, 1916), basing his identification on the stem, leaf and floral characteristics recognised twelve forms in culture experiments, but he studied only seven of them in detail.

Erectus, *latifolius* and *multicaulis* are non-radiate forms with green glabrous stems. *Multicaulis* is different from *erectus* and *latifolius* in its branching habit. It generally produces many branches from the axils of a basal rosette of leaves; the basal rosette probably resulting from the shortening of the basal internodes. *Erectus* and *latifolius* are little branched and the internodes are of appreciable length. All three forms produce about 17 - 24 leaves on the main axis, but those of *erectus* are broad, flat and yellowish green in colour while those of *multicaulis*

are comparatively smaller, but dark green in colour. Latifolius produces broad, incurved, dark green leaves which are shiny on the upper epidermis.

Praecox and genevensis are non-radiate forms with red glabrous stems. Praecox is different from genevensis in that its stem is little branched producing secondary branches only, while that of genevensis is profusely branched with the secondary axis producing tertiary axes as well. They both have internodes of appreciable length, but praecox produces only about 8 - 15 narrow, dark green leaves while genevensis produces 11 - 18.

Lanuginosus and angustifolius possess red hairy stems. Angustifolius is little branched and possesses long internodes with about 14 - 20 narrow, dark green leaves. It is a non-radiate form and each flower is long styled while lanuginosus is a radiate form with short styled flowers. It is profusely branched with branches arising from a close rosette of basal leaves. Similar crowding of leaves also occurs at the apex. It produces about 28 - 32 broad dark green leaves.

Trow (1916) discovered that all forms were cross compatible and he was able to produce radiate forms of the non-radiate forms by hybridization.

Although, the majority of Botanists follow Koch (1902) in recognising two forms only: (a) the type form Senecio vulgaris L which is the non-radiate form, and (b) a variety of this with ligulate ray florets, Senecio vulgaris L var radiatus Koch, it is clear that a considerable degree of

morphological variation exists within the species.

The aim of this section of study was therefore to examine the morphological variation between the different plant lines to see how closely it conforms to those reported by Trow (1912, 1916) and to determine if there are any associations between particular morphological forms and mildew resistance factors.

RESULTS

Morphological variability between the inbred lines of groundsel

The plant lines used for this study are all the same as those used for the tests on host/parasite interaction in Chapter 2. The stem, leaves and floral characteristics of each of the inbred lines were examined and the main features are briefly described below. Detailed observations are given in the Appendix, Table 4.

The Stem

A few plant lines had green stems, but the majority had purplish red stems. The intensity of redness was in part dependent upon light conditions. Some green stemmed plants produced reddish pigments, particularly at the nodes and the lowermost internodes when exposed to light, while red stemmed plants lost some of their red colour in shade.

Epidermal hairs

Most of the plants were glabrous, although hairs were often present in the leaf axils, on the leaf buds and on the youngest internodes.

A few of the plants were, however, distinctly hairy. Environmental conditions influenced hair production and in most plants, hairs were less well developed as the plants grew older.

Branching habit

Various branching habits were observed. Some plants were unbranched or little branched while others were profusely branched. Among the profusely branched forms were one producing tertiary branches. Plants producing tertiary branches are rare as the majority of them produce only secondary branches. In some plants branches arose from basal nodes which had very short internodes so that a rosette of basal branches developed. In some others, the basal internodes are of appreciable length (Plate 4).

The leaves

The leaves are very variable in shape, size and colour. Some of them are incurved, a few are upturned, but the commonest types are flat. The size varied from small and narrow to large and broad. Most leaves are dark green in colour, but a few are yellow green while the rest are intermediate. The lower epidermis of the leaves of some red stemmed plants are red in colour while others are green. The red colour gradually decreased in intensity as the leaves grew older.

The inflorescence

The inflorescence consists of two types: one type is composed of disc florets only (non-radiate inflorescence) and the other is composed

PLATE 4: Photomicrographs illustrating differences in branching habit of two groundsel plants.

of ligulate ray florets as well as disc florets (radiate inflorescence). The majority of the plants possess non-radiate inflorescences with only about 6.4% possessing radiate inflorescences. The capitulum of most plants possessing the radiate type of inflorescence consists of about 8 - 14 bright yellow ray florets, but one plant line (1d) had about 8 - 13 creamy yellow ray florets.

For the purpose of this investigation, these plant lines have been classified on the basis of stem colour (red or green), presence or absence of epidermal hairs (hairy or glabrous) and the inflorescence type (non-radiate or radiate). Of the eight possible combinations of these morphological characters, only six were found and these are listed in Table 9.

Analysis of the deviation of the observed frequency of each phenotype from the expected frequency was carried out and the results are given in Table 9.

TABLE 9

The possible combinations of phenotypic characters and their frequency within the sampled population.

Morphological characters of the host plant lines	Observed No. of plant lines	Expected* No. of plant lines	X^2 Contribution
Red, glabrous non-radiate	163	154.10	0.5140
Green, glabrous non-radiate	41	42.46	0.0502
Red, hairy non-radiate	17	29.35	5.1967
Green, hairy non-radiate	13	8.09)	16.4762
Red, hairy radiate	10	2.01)	
Red, glabrous radiate	6	10.54)	
Green, hairy radiate	0	0.55)	4.5633
Green, glabrous radiate	0	2.90)	
TOTAL	250	250.00	$X^2_{(1)} = 26.8004$
			$P = < .001$

* Expected number of plant lines was calculated from the observed

* frequencies of each morphological character:

red $= \frac{196}{250} = 0.784$; green $= 1 - .784 = 0.216$

glabrous $= \frac{210}{250} = 0.840$; hairy $= 1 - .84 = 0.160$

non-radiate $= \frac{234}{250} = 0.936$; radiate $= 1 - .936 = 0.064$

The results show a non-random combination of morphological characters. There appears to be a deficiency of red, hairy and non-radiate plants and an excess of red, hairy and radiate plants. A possible explanation for this deviation is that hairy stem and radiate inflorescence characters are in some way associated and an analysis in Table 10 confirms this.

TABLE 10

Test of association between hairy and radiate; and glabrous and non-radiate characters.

Stem character	Inflorescence character		Total
	Non-radiate	Radiate	
Glabrous	204	6	210
Hairy	30	10	40
	234	16	250

$X^2_{(1)} = 23.93$, $(P < .001)$

The high X^2 value thus indicates that hairy stem and radiate inflorescence characters are associated and also that glabrous stem and non-radiate inflorescence characters are associated.

The association between morphological characteristics and mildew resistance factors

The morphological characteristics and resistance factors

contained by each of the 250 plant lines are listed in the Appendix, Table 4. An attempt was made to determine if there was any association between particular resistance factors and particular morphological characters but because of the limited number of plants tested with isolates 6, 7 and 8, only isolates 1 - 5, which were tested on all lines, are considered in the detailed analysis. The number of plant lines within each morphological character possessing no resistance factors, or possessing each of the resistance factors R_1 to R_5 are given in Table 11.

TABLE 11

The numbers of plant lines within each host morphological character possessing resistance factors, 0, R_1, R_2, R_3, R_4 or R_5.

Host morphology	Individual Resistance Factors						Total No. of Plants
	0	R_1	R_2	R_3	R_4	R_5	
Red, glabrous non-radiate	80	31	33	26	47	39	163
Green, glabrous non-radiate	18	11	10	6	14	12	41
Red, hairy non-radiate	10	4	4	5	5	5	17
Green, hairy non-radiate	9	1	0	4	1	1	13
Red, hairy radiate	2	3	8	3	6	1	10
Red, glabrous radiate	2	1	2	0	1	1	6
TOTAL	121	51	57	44	74	59	250

It should be noted that some plant lines possessed several resistance factors and they are thus included more than once in the totals. This is why the overall total number of plants is greater than 250.

The number of plants in most categories was small and so it was impossible to analyse the data given in Table 11 as it stands. For this reason, the plant lines were classified simply on the basis of stem characters or inflorescence type as shown in Table 12. The number of plant lines possessing none or any of the five resistance factors were compared between each complementary pair of characters and the

TABLE 12

An analysis of the association of resistance factors 0_1, R_1, R_2, R_3, R_4 and R_5 with particular host morphologies.

Host Morphology	Resistance Factors						Total No. of Plants
	0	R_1	R_2	R_3	R_4	R_5	
Red Stem	94	39	47	34	59	46	195
	(48.0)*	(19.9)	(24.0)	(17.3)	(30.1)	(23.5)	
Green Stem	27	12	10	10	15	13	54
	(50.0)	(22.2)	(18.5)	(18.5)	(27.8)	(24.1)	
X^2	0.1760**	0.3203	0.4406	0.1616	0.0265	0.0749	
P	NS	NS	NS	NS	NS	NS	
Hairy Stem	21	8	12	12	12	7	40
	(52.5)	(20.0)	(30.0)	(30.0)	(30.0)	(17.5)	
Glabrous Stem	100	43	45	32	62	52	210
	(47.6)	(20.5)	(21.4)	(15.2)	(29.5)	(24.8)	
X^2	0.1549	0.0798	0.9578	4.0822	0.0165	1.4268	
P	NS	NS	NS	< 0.05	NS	NS	
Radiate	4	4	10	3	7	2	16
	(25.0)	(25.0)	(62.5)	(18.7)	(43.75	(12.5)	
Non-radiate	117	47	47	41	67	57	234
	(50.0)	(20.1)	(20.1)	(17.5)	(28.6)	(24.4)	
X^2	4.8157	0.0229	12.9915	0.0460	0.9971	1.9184	
P	< 0.05	NS	< 0.001	NS	NS	NS	

Where, NS = Not Significant at $P = 0.05$.

* Number of plant lines expressed as a percentage of the total number of plants in each row

** Test of association of each pair of morphological characters with resistance factors as calculated using the two x two contingency table method; (Bishop, 1978), for example (see over) -

Stem Colour	Resistance Factors		Total No. of plants
	Absent	Present	
Red	94	102	196
Green	27	27	54
TOTAL	121	129	250

$$X^2_{(1)} = 0.1760$$

Results show that there is no specific association between resistance factors R_1, R_4 or R_5 and any host morphology. However, an association was apparent between resistance factor R_2 and radiate inflorescence character and R_3 and hairy stem character. A greater proportion of plants with non-radiate inflorescences contained no resistance factors to isolates 1 - 5 than did plants with radiate inflorescences.

DISCUSSION

In this study, three pairs of characters (red stem vs. green stem; hairy vs. glabrous and radiate inflorescence vs. non-radiate inflorescence) were considered. Of these pairs, red stem was more common than green stem; glabrous stem was more common than hairy stem and non-radiate inflorescence was more common than radiate inflorescence character. It is not clear whether selection pressure imposed by the parasite or by some other environmental factors determine the frequencies of these characters or whether some other factor is involved.

Of the eight possible combinations of these characters, only six

were found and their phenotypes closely resembling those described by Trow (1912, 1916). The absence of green glabrous plants with radiate inflorescences and green hairy plants with radiate inflorescences was probably a reflection of the smallness of the sample size. Plant lines with red, hairy stems and non-radiate inflorescence were fewer than expected; there is thus a negative association between hairy stem character and non-radiate inflorescence character. Hairy stem character was, however, positively associated with the radiate inflorescence character and glabrous stem character was also positively associated with non-radiate inflorescence character. No evidence was, however, obtained to indicate the nature of these associations. These results, however, confirm Trow's (1912) findings.

An analysis of the association between the resistance factors R_1 - R_5 and the different morphological characters of the host showed that R_1, R_4 and R_5 were present in more or less equal proportions in all the morphological characters. However, a positive association was found between the R_2 factor and the radiate inflorescence character and between the R_3 factor and the stem epidermal hair character. Also, a greater proportion of radiate plants than non-radiate plants possessed resistance factors.

CHAPTER 5: THE INHERITANCE OF DISEASE RESISTANCE FACTORS IN GROUNDSEL

INTRODUCTION

Very few studies have been carried out on the genetics of groundsel. One such study was that reported by Trow (1912, 1916). He (1912) studied the inheritance of morphological characters in groundsel and found that in a cross between a red stemmed plant and a green stemmed plant, the F_1 hybrids had an intermediate stem colour with the F_2 plants segregating in a ratio of 1 dark red stemmed plant : 2 pale red stemmed plants : 1 green stemmed plant. He further reported that genes determining the presence or absence of epidermal hairs or the development of radiate or non-radiate inflorescences segregated in a similar way in the F_2 generations. He concluded that red stem, the presence of epidermal hairs on the stem and the radiate inflorescence characters were incompletely dominant over green stem, glabrous stem and non-radiate inflorescence respectively. Yellow ray colour was, however, completely dominant over cream ray colour. Thus, the inheritance of the morphological characters in groundsel occurs in a simple Mendelian manner.

The inheritance of disease resistance in groundsel has, however, not been reported to my knowledge.

The studies on the interactions between groundsel and mildew reported in Chapter 2, indicated the operation of a gene-for-gene system between host and parasite. This section reports a study on the inheritance of disease resistance factors in the host which was carried out to

determine if it might be under the control of a major gene system of the same type that has been demonstrated for race specific resistance in crop plants.

MATERIALS AND METHODS

Production of plant materials

Inbred lines of non-radiate and radiate plants exhibiting different reactions to *E. fischeri* were raised in the growth room as described in Chapter 2.

Production of hybrids

Several methods are available for cross pollinating plants under controlled conditions. When the flowers are large and have a simple structure, the flower to be used as the female parent can be emasculated to prevent self pollination. Pollen can then be transferred from the anther of the plant intended to be the male parent onto the stigma of the female parent. This method would be very difficult and tedious to carry out on groundsel because the individual flowers are too small to be emasculated.

However, crosses in groundsel can be made by rubbing the capitulum of one parent plant with the capitulum of the other parent plant. In this way cross pollination can be effected in both parents plants. But since groundsel is self compatible, most of the fruit produced in any attempt at cross pollination without emasculation is likely to be due to self pollination. The few resulting from cross pollination can be distinguished if segregation of marker genes can be followed.

The evaluation of the marker genes

The possible markers are stem characters, i. e. stem colour (red or green), presence (hairy) or absence (glabrous) of epidermal hairs; and the inflorescence type, i. e. whether it has ray florets as well as disc florets (radiate) or whether it has disc florets only (non-radiate), and the colour of the corolla of the ray florets (yellow or cream). A number of these markers were investigated and the best markers proved to be the radiate or non-radiate inflorescence characters. Radiate plants can easily be distinguished from non-radiate plants and the hybrids are clearly recognisable because the form of the inflorescence is intermediate between the two parental types, Plate 5.

In this study most of the crosses were made between radiate and non-radiate plants and out of 21 attempted crosses 15 were successful.

The colour of the corolla of the ray floret is not a very suitable marker because the colour difference in the disc florets of the non-radiate plants was not distinct enough to be classified into bright yellow or creamy yellow.

Due to environmental effects on the expression of stem colour and epidermal hair characters, as described on page 68, these markers were not found suitable for routine use, but had to be used in crosses in which both parents had non-radiate inflorescences. In these cases, stem colour was used as the marker and out of three attempted crosses one was successful.

A B C

PLATE 5: Photomicrograph illustrating a hybrid plant (B) between two parental plants - A - the non-radiate parent, C - the radiate parent.

Procedure for cross-pollinating the experimental plants

All crosses were made in the growth room. At the first visible appearance of capitula a string was tied from the capitulum of one parent plant to the other in such a way that the capitula of the parental plants were in close proximity, with the buds touching end to end as far as was practicable. Each pair of parental plants was then covered with a perforated polythene bag to prevent cross pollination by other plants. The string was adjusted as often as necessary, without removing the bags so that the flower heads remained in close contact during further growth. As soon as the flowers of the capitula opened they were rubbed against each other daily to ensure transfer of pollen grains. Seeds from each of the cross pollinated capitula of the two plants were later collected.

The Production of the F_2 generation

Seeds from these cross pollinated plants were sown and after germination the seedlings were protected from infection by covering each pot with a propagating cover. At two weeks they were transplanted as described previously in Chapter 2. The hybrid plants were later identified by the marker gene used in the cross and the selfed plants eliminated. The hybrids were then allowed to self pollinate and their seeds collected and sown to produce the F_2 generation. At germination the F_2 plants were covered with propagating covers to protect them against mildew infection and to prevent cross pollination. When the plants flowered a record of the segregation of the marker character was made.

Testing for resistance factors in the F_1 and F_2 generations

The resistance of each plant was tested by inoculating detached

leaves with the appropriate mildew isolates as required. The parental types were included as controls.

Experimental results

Fifteen successful crosses were made between non-radiate and radiate plants. Out of these, hybrids were obtained from the radiate parent only in ten crosses, from the non-radiate parent only in four crosses and from both parents in one cross. In a cross between a red stemmed non-radiate plant and a green stemmed non-radiate plant, hybrids were obtained from both parents.

The inheritance of resistance factors in the F_1 hybrids and segregation of the resistance factors in the F_2 generation

Only isolates 3, 4 and 5 of the original isolates could be used in this study. Isolates 1 and 2 and 1, 500 F_2 plants were lost when the growth room in which they were being grown over heated, leaving only 314 plants from five crosses. A further two isolates X and Y that had been found to be avirulent on some of the parental plants used in the crosses, and virulent on others, were also used. They were partially characterised by testing for virulence on the parent plants. Isolate X infected plants containing resistant factors one to seven inclusive, and thus following the system used in Chapter 2, this isolate was designated $V_1V_2V_3V_4V_5V_6V_7$. Since some plant lines were resistant to it, it clearly possessed at least one avirulent factor, but it was not possible to determine whether this factor was the same or different from factor A_8 and thus whether the isolate was similar or different from isolate 8 described in Chapter 2, (Table 4). Similarly, isolate Y was characterised as far as possible

using the parental plant lines and shown to possess virulence factors $V_1V_2V_4V_5$. Since some of the plant lines were resistant to it, it also clearly possessed avirulent factors, but it was not possible to determine whether or not they were identical with any of the known ones such as A_3, A_6, A_7 or A_8.

Detailed results of the inheritance of resistance factors in the F_1 and the segregation of these resistance factors in the F_2 generation are given in Appendix Tables 5a - 5e and are summarised in Table 13.

All crosses between plants which were resistant to isolates X, Y, 4 or 5 and plants which were susceptible to these isolates gave F_1 hybrids which were resistant and the infection type was similar to that of the resistant parent (Table 13, crosses A - E). Thus, resistance to isolates X, Y, 4 or 5 in these crosses was dominant over susceptibility. In the cases of isolates Y and 5, the F_2 generation segregated in all crosses to give a ratio of 3 resistant plants to 1 susceptible plant indicating that resistance to each race was due to a single dominant gene . Resistance to the other three isolates segregated in the F_2 generation to give different ratios in different crosses. In the case of isolate 4, the F_2 generation in three crosses (1d x 4h, 10j x 1d and 14c x 3g) segregated in the ratio of 3 resistant plants to 1 susceptible plant, indicating that in plant lines 4h, 10j and 14c resistance to isolate 4 was controlled by a single dominant gene or possibly by two or more closely linked genes. In the cross 10j x 1d, resistance to isolate x also segregated in the F_2 generation to give a ratio of 3 resistant plants to 1 susceptible plant. Thus plant line 10j also appears to contain a single dominant gene conferring

resistance to isolate x as well. In the crosses 11 x 8h and 11 x 10j, resistance to isolates X and 4 in the F_2 generations segregated to give a ratio of 15 resistant plants to 1 susceptible plant. Thus resistance to isolates X and 4 in plant lines 8h and 10j appears to be determined by either one or both of two unlinked dominant genes.

The F_1 hybrids of the crosses between plant lines resistant and susceptible to isolate 3 were resistant except in two crosses, both involving plant line 10j as the resistant parent. Thus in the crosses 1d x 4h and 11 x 8h, the F_1 hybrids were resistant and the F_2 generation segregated in a ratio of 3 resistant plants to 1 susceptible plant indicating that resistance to isolate 3 in plant lines 4h and 8h was conditioned by a single dominant gene.

However, when plant line 10j which was resistant to isolate 3 was crossed with the susceptible plant lines 1d or 11, F_1 hybrids were obtained which were susceptible. Thus resistance to isolate 3 in plant line 10j was recessive to susceptibility (Table 13, crosses C and D). In the cross between 10j and 1d in which the seed was collected from the susceptible parent (1d), the possibility that the F_1 was the result of a self fertilization rather than a cross fertilization was ruled out by the morphology of its capitulum. The F_1 plant was heterozygous for the ray floret character, and the corolla was bright yellow in colour (Appendix, Table 5a). The F_2 generation of these two crosses segregated in ratios of 7 resistant plants to 9 susceptible plants indicating that two recessive genes were involved, with resistance being conferred by either one or both of them.

TABLE 13

The segregation of resistance factors in the F_2 generations of crosses 1d x 4h, 1l x 8h, 10j x 1d, 1l x 10j and 14c x 3g.

Cross A 1d (♂) x 4h (♀)

Isolates	Reaction Types			Observed No. of plants in the F_2		Expected ratio of plants in the F_2	$X^2_{(1)}$	P
	P_1(♂)	P_2(♀)	F_1	R	S	R : S		
Y	4	0	0	59	17	3 : 1	.2807	NS
3	3	0	0	81	25	3 : 1	.1132	NS
4	3	0	0	79	27	3 : 1	.0126	NS
5	4	0	0	79	27	3 : 1	.0126	NS

Cross B 1L (♂) x 8h (♀)

Isolates	Reaction Types			Observed No. of plants in the F_2		Expected ratio of plants in the F_2	$X^2_{(1)}$	P
	P_1(♂)	P_2(♀)	F_1	R	S	R : S		
X	2	0	0	64	4	15 : 1	.0157	NS
Y	0	0	0	68	0	No segregation		
3	4	0	0	92	32	3 : 1	.0430	NS
4	2-3	0	0	116	8	15 : 1	.0086	NS
5	4	0	0	92	32	3 : 1	.0430	NS

Cross C 10j (♂) x 1d (♀)

Isolates	Reaction Types			Observed No. of plants in the F_2		Expected ratio of plants in the F_2	$X^2_{(1)}$	P
	P_1(♂)	P_2(♀)	F_1	R	S	R S		
X	0	3	0	16	8	3 : 1	0.8889	NS
Y	0	4	0	20	4	3 : 1	0.8889	NS
3	0	4	4	10	14	7 : 9	0.0423	NS
4	0	3	0	15	9	3 : 1	2.000	NS
5	0	4	0	18	6	3 : 1	0.000	NS

Cross D 1L (♂) x 10j (♀)

Isolates	Reaction Types			Observed No. of plants in the F_2		Expected ratio of plants in the F_2	$X^2_{(1)}$	P
	P_1(♂)	P_2(♀)	F_1	R	S	R S		
X	2	0	0	25	3	15 : 1	0.9524	NS
Y	0	0	0	28	0	No segregation		
3	4	0	4	13	15	7 : 9	0.0816	NS
4	2-3	0	0	25	2	15 : 1	0.0381	NS
5	4	0	0	18	10	3 : 1	1.7143	NS

Cross E 14c (♂) x 3g (♀)

Isolates	Reaction Types			Observed No. of plants in the F_2		Expected No. of plants in the F_2		$X^2_{(1)}$	P
	P_1(♂)	P_2(♀)	F_1	R	S	R	S		
Y	0	4	0	24	8	3 :	1	0	NS
4	0	4	0	22	10	3 :	1	0.6667	NS

Where,

P_1 = male parent

P_2 = female parent

R = resistant

S = susceptible

NS = not significantly different

Crosses in which both parents were resistant to isolate Y (Table 13, crosses B and D), gave F_1 and F_2 generations in which all plants were uniformly resistant and their infection type very closely resembled that of the parents. Thus plant lines 1L, 8h and 10j contained the same gene or closely linked genes for resistance to isolate Y.

Linkage between resistance factors

In a number of the crosses, the parental combinations of resistance factors occurred more frequently than expected and the non-parental combinations were less frequent than expected indicating that some of the resistance factors were linked. Accordingly, a precise test for association between the resistance factors was made by a X^2 analysis.

These analyses were only carried out on the results of the crosses between plant lines 1d x 4h and 1L x 8h. In all other crosses the numbers of plants in the F_2 generation were too few to be analysed.

Cross 1d x 4h

The resistance factors were considered in pairs and the results for each pair of factors are given in Table 14. The test for association by the X^2 analysis was found to be highly significant. The recombination values were estimated using the product method (Allard, 1956) and the results are listed in Table 14.

TABLE 14

Linkage relationship between pairs of resistance factors in plant lines 4h and 8h.

Linkage relationship between resistance factors R_3 and R_4

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
R_3R_4 - parental type	70	59.625	24.9853	<0.001
R_3r_4) no recombinants	11	19.875		
r_3R_4)	9	19.875		
r_3r_4 - parental type	16	6.625		
TOTAL	106	106.000		

Recombination value = 18.2990 ± 4.2%

Linkage relationship between resistance factors R_3 and R_5

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
R_3R_5 - parental type	79	59.625	93.1111	<0.001
R_3r_5) recombinants	1	19.875		
r_3R_5)	1	19.875		
r_3r_5 - parental type	25	6.625		
TOTAL	106	106.000		

Recombination value = 1.5099 ± 1.52%

Linkage relationship between resistance factors R_4 and R_5

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
R_4R_5 - parental type	70	59.625	28.9098	<0.001
R_4r_5) recombinants	9	19.875		
r_4R_5)	10	19.875		
r_4r_5 - parental type	17	6.625		
TOTAL	106	106.000		

Recombination value = 17.2044 ± 4.1%

Linkage relationship between resistance factors Y and R_3

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
YR_3 - parental type	53	42.75	34.1286	<.001
Yr_3) recombinants	3	14.25		
yR_3)	6	14.25		
yr_3 - parental type	14	4.75		
TOTAL	76	76.00		

Recombination value = 11.81 ± 3.8%

Linkage relationship between resistance factors Y and R_4

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
YR_4 - parental type	55	42.75	32.5848	<.001
Yr_4) recombinants	4	14.25		
yR_4)	4	14.25		
yr_4 - parental type	13	4.75		
TOTAL	76	76.00		

Recombination value = 10.5 ± 3.2%

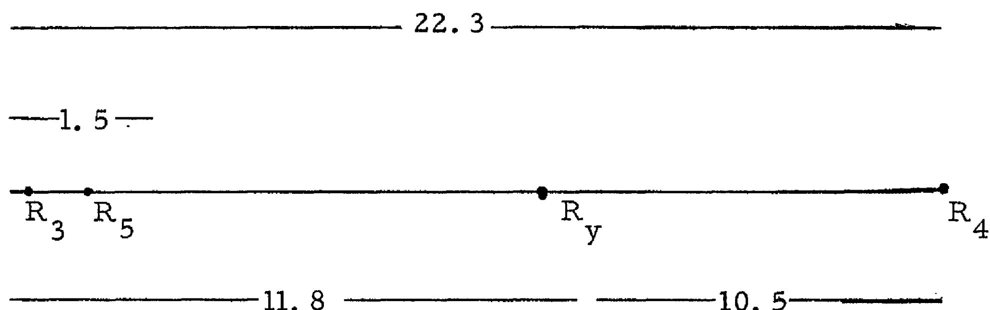
Linkage relationship between resistance factors Y and R_5

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
YR_5 - parental type	53	42.75	34.1286	<.001
Yr_5) recombinants	3	14.25		
yR_5)	6	14.25		
yr_5 - parental type	14	4.75		
TOTAL	76	76.00		

Recombination value = 11.81 ± 3.8%

The map distance between resistance factors R_y and R_3 is the same as that between R_y and R_5. This indicates that R_3 and R_5 are closely linked.

These results thus show that in plant line 4h resistance factors R_y, R_3, R_4 and R_5 are in the same linkage group and can be arranged as shown in the diagram:



Cross 1L x 8h

Linkage relationship between resistance factors R_3 and R_5

Resistance Factors	Observed No. of plants	Expected No. of plants (9 : 3 : 3 : 1)	$X^2_{(3)}$	P
R_3R_5 - parental type	92	69.75	129.4767	< 0.001
R_3r_5) recombinants	0	23.25		
r_3R_5)	0	23.25		
r_3r_5 - parental type	32	7.75		
TOTAL	124	124.00		

In this cross only the parental types were found to occur in the F_2 generation indicating that in plant line 8h, resistance factors R_3 and R_5 were very closely linked. In the previous cross resistance to isolates 3 and 5 were also shown to be closely linked with a recombination value of 1. 5 $\pm$ 1. 5%.

The association between resistance factors and other genetic factors

Of all the possible marker gene investigated, only two; one determining the production of ray florets and the other determining the

colour of the strap shaped corolla were involved in crosses which gave a sufficient number of progeny in the F_2 generation to enable an analysis of linkage between them and the resistance genes to be carried out.

Cross ld x 4h was between a homozygous radiate plant with creamy yellow ray florets (ld) and a homozygous non-radiate plant with bright yellow disc florets (4h). The radiate and non-radiate inflorescence characters segregated in a simple Mendelian manner in the F_2 generation* as described in the Introduction. The corolla of the F_1 hybrid was bright yellow and in the F_2 generation, the colour of the corolla of the ray florets segregated into 3 bright yellow : 1 creamy yellow, in both the homozygous and heterozygous radiate plants, Table 15B. This indicates that the bright yellow colour of the corolla of the ray florets is determined by a single dominant gene. The colour difference in the non-radiate plants was, however, not distinct enough to be classified.

It was then considered necessary to determine if the genes determining the ray floret character and that determining the ray floret corolla colour are in the same linkage group as resistance genes R_3, R_4, R_5 and R_y. Accordingly, a X^2 test for trihybrid ratio should have been used, but the non-radiate plants which possessed resistance or susceptible genes to isolate 3 could not be separated into distinct groups on the basis of the presence or absence of the bright yellow or creamy yellow corolla colour determining genes as was possible for the radiate plants. Thus, the ratio expected in the F_2 generation was not a 27 : 9 : 9 : 9 : 3 : 3 : 3 : 1, but one of 27 : 12 (9 + 3) : 9 : 9 : 3 : 4 (3 + 1). The results of this analysis are given in Table 16.

* (Table 15A)

The results show that the incompletely dominant gene determining the inflorescence character, the dominant gene determining the colour of the corolla of the ray florets and the dominant gene conferring resistance to isolate 3 belong in different linkage groups (resistance factors R_3, R_4, R_5 and R_y are linked).

TABLE 15 A

The segregation of the inflorescence character determining gene in the F_2 generations of the cross 1d x 4h.

Crosses		Observed No. of plant lines			Expected Ratio			$X^2_{(2)}$	P
♂	♀	RR	Rr	rr	RR	Rr	rr		
1d	x 4h	26	53	25	1 :	2 :	1	0.0192	NS

TABLE 15 B

The segregation of the ray floret corolla colour determining gene in the F_2 generation of the cross 1d x 4h.

Radiate inflorescence character	RR		Rr		$X^2_{(3)}$	P
Ray floret colour	CY	cy	CY	cy		
Observed number of plant lines	18	8	37	16	1.1225	NS
Theoretical ratio	3	1	6	2		

TABLE 16

Association between resistance factor R_3, inflorescence character determining gene and the ray floret corolla colour determining gene in the cross 1d x 4h.

Inflorescence character	Ray colour	Gene	Observed No. of plants	Expected ratio	$X^2_{(5)}$	P
Radiate	CY	R_3	44	27	2.5356	NS
Non-radiate	CY	R_3)				
		)	19	12		
Non-radiate	cy	R_3)				
Radiate	cy	R_3	15	9		
Radiate	CY	r_3	12	9		
Radiate	cy	r_3	8	3		
Non-radiate	CY	r_3)				
		)	6	4		
Non-radiate	cy	r_3)				

Where, RR = homozygous ray floret
Rr = heterozygous ray florets
rr = homozygous disc florets
CY = bright yellow corolla of the ray florets
cy = creamy yellow corolla of the ray florets

DISCUSSION

Genetic studies on a limited number of plant lines of groundsel indicated that resistance to isolates of E. fischeri is inherited in a simple Mendelian manner. In most cases resistance appears to be conditioned by single dominant genes. Thus plant line 4h appears to possess single dominant genes governing resistance to isolates 3, 4, 5 and Y; plant line 8h appears to possess single dominant genes conferring resistance to

isolates 3 and 5; plant line 10j appears to possess a single dominant gene conferring resistance to isolate 5 and plant line 14c appears to possess single dominant genes conferring resistance to isolates 4 and Y. The F_1 and F_2 generations of crosses between parents which were resistant to isolate Y were also uniformly resistant to this isolate. Thus both parents appear to contain the same gene conditioning resistance to isolate Y. However, no other crosses were made to determine if resistance to any isolate in any other plant lines were conditioned by the same resistance genes.

Resistance to an isolate in a particular plant line may not necessarily be conferred by single dominant genes. For example, resistance to isolates X and 4 in the F_2 generation of crosses between infection type 2 and infection type 0 gave a ratio of 15 resistant plants : 1 susceptible plant indicating that either one or both of two independently inherited dominant genes conferred resistance. However, the cross was not carried through to the F_3 generation to determine if the resistant plants segregated to give the expected ratio of 7 pure breeding resistant plants : 4 segregating (15 resistant : 1 susceptible plant) : 4 segregating (3 resistant : 1 susceptible plant) plants, which would have confirmed the involvement of two dominant genes.

Also, in plant line 10j resistance to isolate 3 appeared to be conferred by two independently inherited recessive genes, either or both of which conferred resistance to isolate 3. However, this cross was not carried through to the F_3 generation to confirm whether the 7 resistant plants were true breeding. It thus appears that isolates X, 3 and 4 may

have two avirulent factors to match the two resistance factors in these plant lines.

Genetic studies on the recombination values of resistance factors R_3, R_4, R_5 and R_y reveal that they are linked in plant line 4h and that R_3 and R_5 are closely linked. In plant line 8 h, resistance factors R_3 and R_5 are also closely linked, thus confirming the results obtained in plant line 4h.

Similar linkage relationships have been found to exist among powdery mildew or rust resistance genes in cultivars of wheat and barley, (Allard and Shands, 1954; Roane and Starling, 1970).

However, the single dominant gene determining the inflorescence character, the single dominant gene determining the ray florets' corolla colour and those conferring resistance to isolates 3, 4, 5 and Y in plant line 4h belong in different linkage groups.

These results are thus in general agreement with the mode of inheritance of resistance genes reported by Flor (1942), Moseman (1958), Sears _et al._ (1954), Hooker and Saxena (1967), Udeogalanya and Clifford (1978) and several other investigators. Thus _Senecio vulgaris_ possesses major gene resistance against its parasite _Erysiphe fischeri_.

PART II: HOST/PARASITE INTERACTION

CHAPTER 1 GENERAL INTRODUCTION

A parasite is an organism or virus existing in intimate association with another living organism from the functioning tissues of which it derives part or all of the material for its nutrition (Anon, 1973). The relationship between a parasite and its host is thus basically a nutritional one. The parasite due to its feeding activities constitutes a drain on the nutrient resources of its host and in this way alone can have an effect on the host's growth and development. Besides any deleterious effects due to this basic nutritional relationship a parasite may cause other physiological disorders in its host. These effects, probably resulting from the actions of substances secreted by the parasite or produced by the host in response to stimuli originating in the parasite, are in principle quite separate from the effects caused by the depletion of materials used as nutrients. The first group of activities may be classified as 'essential' (parasitic) and the second group as 'inessential' (pathogenic). Thus the total effect of the activities of a parasite on its host will be in general terms the sum of the effects of both of these activities.

Some of the physiological and morphological effects which occur in the host as the result of infection, have been reviewed by Crowdy and Manners (1971), Wood (1967) and Wheeler (1976) and these may be summarised as follows:

1. Effects on carbon assimilation.
2. Effects on translocation of organic and inorganic substances including water.
3. Effects on respiration

4. Effects on growth regulatory mechanism.

It is intended here to discuss these effects in broad terms, limiting the discussion to a few examples that are of direct relevance in the context of this thesis.

It has been demonstrated by many investigators that biotrophic parasites increase the rate of photosynthetic assimilation of carbon dioxide by host tissues around the infection site, during the first 24 - 48 hours after inoculation, but thereafter the rate rapidly declines (Allen, 1942; Last, 1963; Livne, 1964; Doodson *et al.*, 1965; Scott and Smillie, 1966; Edwards, 1970; Devlin and Barke, 1971). Thus, Scott and Smillie (1966), reported that in barley infected with *Erysiphe graminis f. sp. hordei,* there was an initial stimulation of photosynthesis followed by a rapid decline. The mechanism by which the initial increase in the rate of photosynthesis is brought about is not clear, but Ayres (1976), found that the stomata of the leaves of *Pisum sativum,* 24 hours after inoculation with *Erysiphe pisi,* opened more widely in the light than did those of healthy leaves. Thereafter the stomata were progressively reduced by infection, until the seventh day after inoculation, all movements ceased and the stomata remained partly open. It may thus be that the initial increase in the rate of photosynthesis is partly due to an increased uptake of carbon dioxide due to the increase in the stomata.

That infection reduced photosynthetic carbon dioxide assimilation has been demonstrated by Ayres (1976). He observed a reduction in net photosynthesis in the leaves of *Pisum sativum,* 48 hours after inoculation with *Erysiphe pisi* and attributed this to the reduced stomata which increased

the stomatal resistance to the diffusion of carbon dioxide into the leaf and partly to the reduced ability of the leaves to fix the carbon dioxide. Magyarosy et al. (1976) also demonstrated that 15 minutes after supplying labelled carbon dioxide ($^{14}CO_2$) to leaf discs of Beta vulgaris infected with Erysiphe polygoni the rate of the assimilation of the label was about 50% that of un-infected discs. The decrease in the rate of photosynthesis in wheat leaves infected with Puccinia recondita was, however, attributed to a reduction in the amount of chlorophyll per chloroplast, (Manners and Gandy, 1954). In wheat leaves infected with Puccinia striiformis, the reduction in the rate of photosynthesis was greater than the rate at which chlorophyll was lost indicating that the assimilation rate per unit amount of chlorophyll was falling (Doodson et al., 1965). Decrease in the rate of carbon dioxide assimilation has also been shown to occur in barley, 48 hours after inoculation with Erysiphe graminis f. sp. hordei and was attributed to a loss of enzymic activity associated with both electron transfer and carbon dioxide fixation (Scott and Smillie, 1966). Thus the reduction in the rate of photosynthesis may in part be due to a reduction in the amount of chlorophyll and in part due to a reduction in the efficiency of CO_2 fixation.

In many host-parasite systems the decrease in the rate of photosynthesis in the infected leaves has been shown to be compensated for by an enhanced rate of assimilation of carbon dioxide by un-infected leaves on the infected plants (Wang, 1960; Livne, 1964). Thus Livne (1964) demonstrated that un-infected leaves of Phaseolus vulgaris infected with Uromyces phaseoli assimilated as much as 50% more labelled carbon

than the corresponding leaves on healthy plants. Thrower and Thrower (1966) found that in broad bean infected with *Uromyces fabae*, photosynthates were translocated out of the un-infected leaves on infected plants at a greater rate than was the case for similar leaves on un-infected plants, thus lowering the levels of carbohydrates which remained. This may have stimulated photosynthesis, in the un-infected leaves of infected plants.

Many investigators have shown that infections can affect translocation. Often they not only reduce the rate of translocation of carbohydrates out of an infected organ (Doodson *et al.*, 1965), but they also promote imports into such organs (Livne and Daly, 1966; Shaw and Samborski, 1956). Thus Doodson *et al.* (1965) found that when a single leaf of a wheat plant was infected with *Puccinia striiformis*, the proportion of the assimilates exported was only 0.4% compared with 20% from a corresponding healthy leaf on a control plant. It has also been shown that healthy primary leaves of *Phaseolus vulgaris* when fed with labelled carbon, exported 50% of the label in a five-hour period whereas primary leaves infected with *Uromyces phaseoli* exported less than 2% in a similar time interval (Livne and Daly, 1966). They further showed that the labelled carbon accumulated in infected primary leaves when it was fed to healthy trifoliate leaves of the otherwise infected plants. In the control experiment little carbon accumulated in the healthy primary leaves. Inorganic ions have also been shown to preferentially move to infected parts of the plant (Gerwitz and Durbin, 1965; Yarwood and Jacobsen, 1955).

In contrast to the biotrophic parasites, necrotrophic pathogens cause little or no accumulation in the host tissue, much of the accumulation

occurring in the fungal tissue (Thrower, 1965).

The causal factors of the accumulation of carbohydrates in infected organs are not clear but there is evidence that alterations in hormone balance frequently accompany attack by biotrophic pathogens (Sequeira, 1963; Bailiss and Wilson, 1967; Kiraly et al., 1967). It is thus possible that some part of the observed alterations in translocatory patterns are due to hormonal effects although some may be due to sink effects due to the utilization of the compounds by the parasite.

In many studies photosynthates have been shown to be translocated into the fungus where they are converted into fungal carbohydrates. Thus Edwards and Allen (1966), using the powdery mildew-barley complex, have shown that the parasite was the major site of the accumulation of carbon and that the transfer of carbon from host to parasite was very rapid. Smith, Muscatine and Lewis (1969), describe how in a number of diseases caused by biotrophic pathogens, soluble carbohydrates were taken from the plant by the fungus and converted to forms metabolically inaccessible to the host.

In addition to the altered translocatory patterns in infected plants, parasites have also been shown to interfere with the transport of water. Most foliar diseases have been shown to considerably reduce the proportion of healthy leaf cells essential for the development of a transpiration stream adequate to cause a flow of water into the leaves (Horsfall and Dimond, 1959). It has also been shown that in bacterial wilt of cucurbits caused by *Erwinia tracheiphila*, the bacteria produce extracellular slimy polysaccharides

which together with the bacterial cells cause a mechanical obstruction of the xylem cells and so reduce the efficiency of the water conducting system of diseased plants (Enlows, 1920; Yu, 1933; Burkholder, 1960).

That infection increases respiration in a host plant has been reported by several workers. Measuring total respiration over a period of three days, Yarwood (1934), reported a 50% increase in respiration in clover leaves infected with Erysiphe polygoni compared with that in non-infected leaves. Pratt (1938), followed the course of respiration of wheat after infection with Erysiphe graminis f. sp. tritici and found that although the rate of respiration of healthy leaves declined with age, it gradually increased in infected leaves to a value about three times that of non-infected leaves, and was maintained at this level for a week before it decreased to zero as the leaf died. Allen and Goddard (1938), showed a four to five fold increase in respiration rate in mildewed wheat leaves and most of this increase appeared to be localized in the mesophyll cells of the host. These workers interpreted their results as strong evidence for the production of diffusible substances by the fungus which changed the rate of cellular oxidation in mesophyll cells in the immediate vicinity of the fungus.

Metabolites such as toxins and growth regulators produced by parasites can induce changes in the overall metabolism of the host, the end result of which becomes manifest in the altered growth and development of the host and thus in its final morphology. For example, foliar deformities such as potato leaf roll or tobacco leaf curl which are caused by viruses result from the abnormal growth and expansion of the leaf lamina (Bawden,

1950). In club root of Cruciferous plants caused by Plasmodiophora brassicae, the pathogen stimulates the infected cells to enlarge and induce cell division. Infected cells may be five or more times larger than adjacent un-infected cells and their nuclei and nucleoli 5 and 30 times larger respectively than those of adjacent un-infected cells (Williams, 1964). It has been shown that the plasmodium infected clubs constitute a drain on the economy of the plant by utilizing much of the food stuff required for the normal growth of the plant. They also interfere with absorption and translocation of mineral nutrients and water through the root system and can result in the gradual stunting and wilting of the above ground parts (Sherf, 1964; Colhoun, 1958).

In most of these examples the studies were limited to measurements of the effects of the parasites on individual tissues or organs of the infected plants only and did not include measurements of the effects of infection on the overall growth of the plant. This aspect of host-parasite relationship has been investigated by Last (1962), Fric (1975), Harrison and Isaac (1969).

Using growth analytical methods, Last (1962) studied the effects of localized infections of Erysiphe graminis f. sp. hordei on the growth of barley. He showed that although the total dry weight of infected plants continued to increase throughout the period of investigation, control plants contained 59% more dry matter than infected plants, 80 days after inoculation. At this stage the infected plants had about 30% total mildew cover. The relatively greater reduction in root growth compared to leaves in infected plants was perhaps the most unexpected feature of this host

parasite system, since powdery mildews are usually discussed in relation to their leaf environment with little thought of possible effects on the roots. The work of Edwards (1971) may offer an explanation of the phenomenal reductions in root dry weight of mildew infected barley plants. He reported that in healthy primary leaf of barley, labelled carbon fixed in the tip section of the blade was preferentially translocated to the roots, whereas label fixed in the base section was primarily translocated to the shoot. When sporulating powdery mildew infection was present in the mid section of the primary leaf, label fixed in that region or in the acropetal healthy tip section readily accumulated in the infection area, thus depriving the roots of photosynthates. However, label fixed in the healthy basal section was translocated into the other parts of the plant with only a small fraction moving acropetally into the infected mid section. Last (1962) further showed that infection reduced the numbers of tillers and their extension growth. Loss of dry weight of tops showed a close relationship to loss in total leaf area which was attributable to decreases in the numbers and sizes of leaves per infected plant. The mean net assimilation rate of infected plants was reduced by about 26%. Ear development was similarly affected; the average dry weight per ear being 25% less in the infected plants than in the controls.

The work of Harrison and Isaac (1969), is an outstanding example of an application of growth analytical techniques to plant pathological problems. They found that during the first five to six weeks of growth, potato plants infected with Verticillium dahliae or V. albo-atrum were morphologically identical with the un-infected controls. However,

during this period the growth rate of the infected plants was affected as was the distribution of dry matter between organs. It was also found that the total green leaf area was greatly reduced, due to defoliation and decrease in size of the remaining leaves. The growth indices : unit leaf rate and relative growth rates were found to be reduced by infection.

However, there are a number of associations known between plant hosts and fungal parasites which do not lead to any of these marked deleterious effects on the host, but on the contrary may prove very advantageous to the host. Ectotrophic mycorrhiza are examples of host/parasite systems in which the photosynthesizing host plant is the primary source of carbohydrate to both components of the relationship. The fungal component absorbs nutrients from the substrate and is the source of inorganic nutrients for the system. Using Beech mycorrhiza, Lewis and Harley (1965 a, b and c) demonstrated that carbohydrates applied as sucrose to the host tissue is translocated to the fungal component where it is converted to the fungal carbohydrates trehalose, mannitol and glycogen. Carbohydrate applied directly to the fungal tissue was little translocated to the host, but was mainly assimilated in the fungal sheath. However, the host gains from its association with the fungi. As Harley (1971) points out, in addition to offering protection against invading organisms some mycorrhizal fungi may also help to increase nutrient absorption by the host. Recent work has shown that plant growth may be improved by infection with vesicular-arbuscular mycorrhizal fungi and the growth response is normally associated with an improved supply of phosphorus from the soil (Mosse, 1973; Khan, 1975 and Cooper, 1975). This trend

has been shown experimentally on a wide range of agricultural crops. Thus Kleinschmidt and Gerdeman (1972) found that the dry weight of citrus plants in a fumigated field plot inoculated with Endogone mosseae were significantly greater than that of non-inoculated control plants. Ross and Harper (1970) obtained a 34 to 40% increase in the yield of soybean plants infected with Endogone. This was substantiated by the results of Khan (1975) who also found that maize inoculated with Endogone with at least 60% of the root length infected increased the root/shoot fresh weight ratios by 50%; stimulated the rate of increase of leaf area and caused a twelve-fold increase in grain weight.

Mycorrhizal infection of Ericaceous plants has also been shown to increase the efficiency with which roots of the host plant absorb nitrogen from heathland soils of low fertility (Stribley, Read and Hunt, 1975). The mycorrhizae have been shown to participate directly by absorbing inorganic nitrogen present at low concentrations or by providing access to a source of nitrogen (possibly organic) not available to infected roots (Stribley and Read, 1974). Thus in mycorrhizal associations between fungi and plant roots, any inessential activities of the fungal partner would appear to be minimal or even non-existent.

In these cases, plant growth was increased rather than being depressed by infection although the fungus might have obtained a large part of its carbon assimilation from its host.

Thus in theory a host should be able to tolerate a parasite without its growth being greatly affected. This would occur if the host has sufficient

extra assimilatory capacity over that required to support its own growth and if the parasite does not produce toxins or other metabolites which may cause marked disturbances in its host.

In most crop plants fairly low levels of infection appear to have marked effects on host plant growth and yield (Chester, 1950; James, 1974; Caldwell et al., 1958; Ellis, 1954), but the effect of particular levels of infection by a specific parasite may vary between cultivars, some cultivars being more affected than others. For example, Caldwell et al. (1958) showed that with similar maximum levels of infection, the losses of wheat grain attributable to Puccinia coronata were not significant on the variety Benton but were significant on the variety Clinton 59. The cultivars which are least affected are thus said to be tolerant. Tolerance has been defined by Schafer (1971) as "that capacity of a cultivar resulting in less yield or quality loss relative to disease severity or pathogen development when compared with other cultivars of that crop". Since the parasite grows and sporulates well on these tolerant cultivars, it follows that the marked physiological disturbances induced in the less tolerant cultivars are not essential to the establishment of an effective relationship by the parasite with its host. The evolutionary trend in host-parasite relationships would thus be expected to be towards increasing tolerance since parasite isolates which have least effect on their host would have a selective advantage over those which have more deleterious effects. Such a situation would be expected to lead to the maximum survival of both the host and the parasite. Thus it is commonly observed that wild plants sometimes become almost totally infected yet continue to grow and set

viable seed (Tarr, 1972; Ben-Kalio, 1976). They thus appear to exhibit a high degree of tolerance to their parasites.

However, until the work of Ben-Kalio (1976) no studies had been undertaken (to my knowledge) to establish the level of tolerance in wild plants. Thus from the studies on the effects of Erysiphe fischeri on the growth and development of Senecio vulgaris, Ben-Kalio (1976) showed that low levels of infection in which up to 10% of the aerial parts of the host were colonized significantly increased the rate at which the lower leaves senesced. There was however no overall effect on growth because the loss was compensated for by the expansion of further leaves. High levels of infection in which between 75% and 100% of the aerial parts became colonized, did not kill the host and the plants continued to grow to flower and set viable seed. Dry matter production was found to be reduced by about 80% due to reduction in both the green leaf area and the unit leaf rate, but its overall distribution throughout the plant was not affected, the growth of the roots, stems and leaves being stunted in equal proportion. Thus infection did not appear to disrupt the translocatory system of the host. The number of flowers produced was also found to be reduced by about 75%, but the weight of individual fruits was reduced by only 25%. Thus Senecio vulgaris appears to possess a high level of tolerance to Erysiphe fischeri.

It is likely that conscious or unconscious selection among crop plants for improved yielding capacities following the development of agriculture over the years has caused considerable disruption to the physiological balance that would have existed between these plants and their

parasites. The lack of tolerance generally shown by crop plants to their parasites, may therefore be due to the erosion of the tolerance which was almost certainly present in their wild ancestors during attempts at crop improvement by conventional breeding methods. The extent to which a host plant can endure the activities of its parasite may be best investigated using wild species which have not suffered such interferences through breeding programmes as test plants. Thus it should be possible to re-select for tolerance and utilize it probably in combination with resistance, to help reduce crop losses caused by parasites. However, its full potential in this field cannot be properly evaluated until the levels of tolerance it will be possible to achieve are known. The results of such studies may bear considerable relevance to the potential value of tolerance as a breeding aim for controlling yield losses in crop plants.

The extent to which Senecio vulgaris endured the activities of its parasite Erysiphe fischeri has been partly investigated by Ben-Kalio (1976). This study was intended to investigate further the levels of tolerance in S. vulgaris to E. fischeri. Thus although Ben-Kalio (1976) reported a reduction in dry matter production in the infected plants, which he attributed to a reduction in leaf area, little is known of the events resulting in the reduction in dry matter production and the reduction in leaf area. Dry matter production may be reduced due to effects of infection on the chlorophyll content of the leaves or on the efficiency of the leaves as photosynthesizing organs. The leaf area may be reduced due to a reduction in cell division and/or cell expansion in the leaf lamina.

The aim of this study was therefore to investigate further the

effects of infection on the growth, development and functioning of the leaf tissues of S. vulgaris by comparing the growth of healthy and E. fischeri infected plants using standard methods of growth analysis (Hunt, 1978).

CHAPTER 2: THE EFFECT OF INFECTION ON THE GROWTH, DEVELOPMENT AND FUNCTIONING OF THE LEAF TISSUE OF GROUNDSEL

MATERIALS AND METHODS

All the experiments described here were carried out in a growth room maintained at a temperature of about 19°C with a 16 hour photoperiod.

The groundsel plants used in this study were obtained from a highly susceptible inbred line, in an attempt to avoid genetic variability within the experimental plants which might have resulted in variable responses to the pathogen, (see Part I, Chapter 2).

Production of Plant Material

Seedlings were raised in seed trays containing Levington potting compost and two weeks after germination, those of approximately equal vigour were selected and transplanted singly into 11.4 cm plastic pots containing similar compost. The plants were then divided into two groups of 60 plants each.

Maintenance of mildew-free plants

For this study it was necessary to maintain disease-free plants for comparison with infected plants. Freshly prepared 0.05% aqueous Benomyl (methyl-1 (butylcarbamoyl)-2-benzimidazole-carbamate) was poured into the pots of one set of 60 seedlings at the time of transplanting until the compost was saturated. This treatment was repeated at four weekly intervals since preliminary studies had indicated that a single

treatment did not protect the plants throughout their 12 - 14 weeks' life cycle.

Benomyl has been generally considered non-phytotoxic and has been reported to give good control of mildew (Tarr, 1972; Crowdy, 1971; Edgington _et al._, 1972; Brooks, 1970; Ben-Kalio, 1976). It has also been reported to have no effect on the growth of groundsel plants (Ben-Kalio, 1976).

Inoculation procedures

The non-benomyl treated plants were inoculated by introducing heavily infected plants into the growth room and allowing them to become naturally infected. In the first experiment the infected plants were introduced one week after transplanting and in the second experiment they were not introduced until two weeks after transplanting. All plants were moved around the growth room at weekly intervals to ensure even growth.

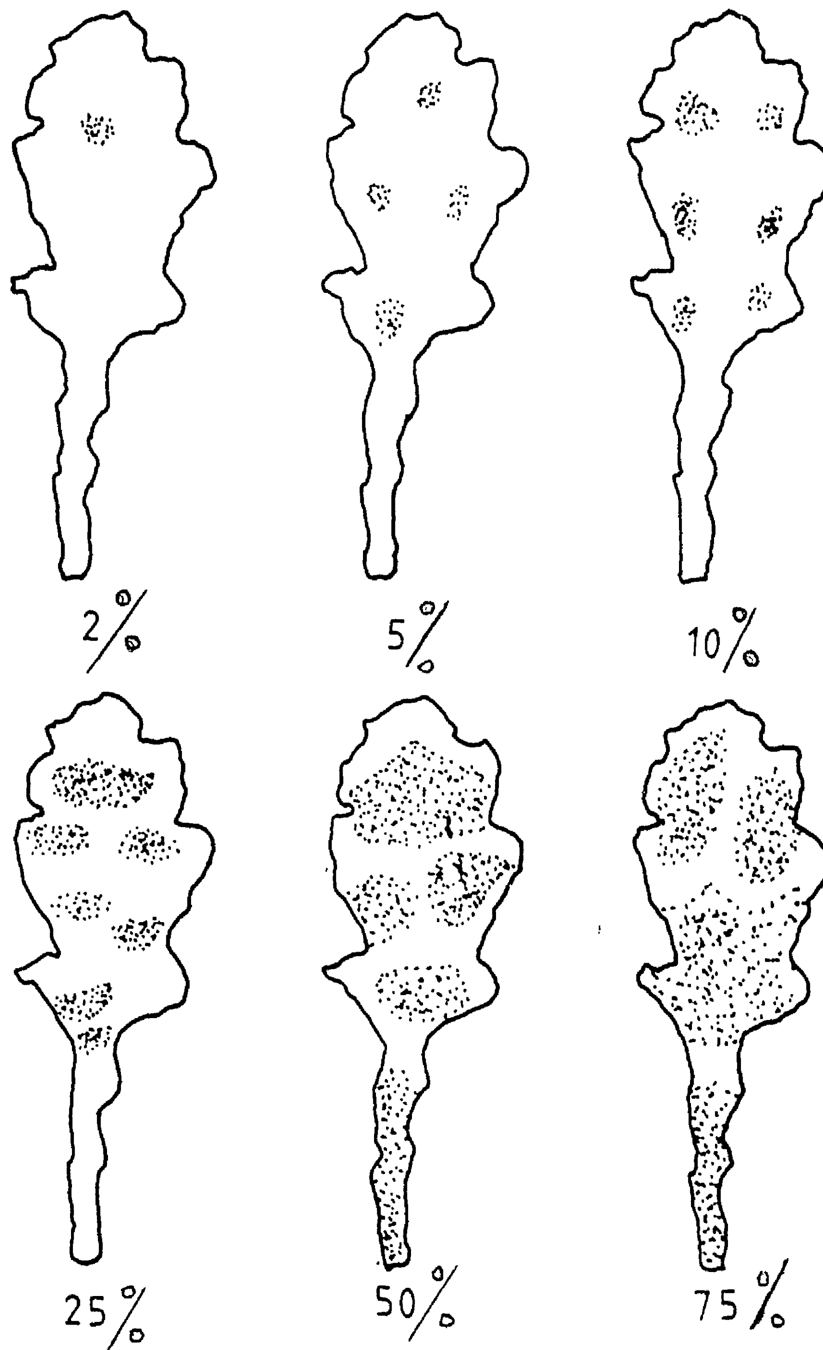
Assessment of mildew intensity

An accurate measure of the percentage mildew cover on the infected plants is an important prerequisite for a study of the effects of infection on the growth and development of the host plant. A diagrammatic scale (Ben-Kalio, 1976) was therefore used for the assessment of mildew intensity because it is easy to use in practice and makes for rapid assessment. Figure 1 shows the six disease gradings used.

Harvest

Five plants from each of the infected and un-infected (control) groups of plants were sampled at each harvest. In the first experiment

FIG. 1



Diagrammatic scale for appraising mildew intensity on groundsel leaves

the first harvest was at the third week after transplanting and in the second experiment it was at the fifth week after transplanting. Subsequent harvests in both experiments were made at weekly intervals so that five harvests were made in each of the experiments.

Post harvest treatment

Before division of the plants into root and shoot systems, the root system was carefully rinsed under running tap water to remove the adhering compost particles and then blotted dry.

Measurement of leaf area

A portable photoelectric leaf area meter (Lambda Instruments Corporation, Model LI-3000) was used for the measurement of leaf area. In the first experiment total leaf area only was measured for each plant at a particular harvest. In the second experiment individual leaf areas were measured. The cotyledons were numbered one and two and the foliage leaves were numbered in order up the main axis. The area of each leaf was measured three times and the mean calculated. The mean of means of the areas of corresponding leaves on the plants of a particular treatment at each harvest was regarded as the area of that leaf. Total leaf area only was measured for each lateral branch.

Fresh and dry weights determination

The fresh weights of the root system; the stem including lateral branches and floral parts and the leaves of each plant were determined separately. These plant parts were then wrapped separately in pre-

weighed aluminium foil envelopes and dried to constant weight at 80°C. Their dry weights were then determined after a cooling period of about 10 minutes in a desiccator.

Determination of chlorophyll content

The chlorophyll of both infected and un-infected plants was extracted separately after dry weight measurements had been taken. Because in groundsel all aerial parts of the plant contain chlorophyll and are thus capable of photosynthesis, the chlorophyll was extracted from the stems including lateral branches and green floral parts as well as from the leaves, using the methods of MacKinney (1941) and Arnon (1949).

The dried plant parts were ground to a powder and then warmed in 80% acetone on a water bath (approximately 38 - 40°C) for about 10 minutes. The supernatant was then decanted and filtered through Whatman No. 1 filter paper. Fresh acetone was added to the residue and re-extracted. This process of extraction was repeated until the acetone no longer showed a green colouration. All chlorophyll extract from a particular plant part were combined, re-filtered and the volume measured. The optical density of the chlorophyll solution was measured at 652 nm in a spectrophotometer (Perkin Elmer) and the chlorophyll content in mg/ml was calculated using the following formula:

$$\text{Chlorophyll concentration} = \frac{\text{OD x dilution}}{34.5} \text{ mg/ml}$$

Where: OD = optical density

34.5 = specific absorption co-efficient for the chlorophyll at 652 nm

Computation of the growth indices : specific leaf area (SLA), leaf weight ratio (LWR), leaf area ratio (LAR), Unit leaf rate or net assimilation rate (ULR, NAR or E) and relative growth rate (RGR)

Growth in plants may be defined as the increase in dry weight of the plant (Sestal *et al.*, 1971). Growth analysis thus makes it possible to follow the formation and accumulation of biomass resulting from the interactions between external environmental factors and the internal factors of the plant. Fresh and dry weights of various organs; size of the assimilatory apparatus and chlorophyll content are some of the attributes which are employed in the analysis of growth. From some of these primary values the various indices can be calculated.

Specific leaf area (SLA)

SLA is defined as the mean area of leaf displayed per unit weight of leaf (Hunt, 1978). It usually reflects leaf thickness and the relative proportions of the assimilatory and conductive or mechanical tissues in the leaf. It is derived from the formula:

$$SLA = \frac{LA}{LW}$$

Where: LA = the total leaf area

LW = the leaf dry weight

Leaf weight ratio (LWR)

Leaf weight ratio is the ratio between leaf dry weight and total plant dry weight. In analytical terms it represents the average fraction of the plant's total stock of organic material divided between the photosynthetic system and the rest of the plant. It is derived from the formula:

$$LWR = \frac{LW}{W}$$

Where: W = the total plant dry weight

Leaf area ratio (LAR)

Leaf area ratio is defined as the ratio of total leaf area to whole plant dry weight (Hunt, 1978). It describes the relative size of the assimilatory apparatus (Briggs, Kidds and West, 1920b) and is calculated from the formula:

$$LAR = \frac{LA}{W}$$

Unit leaf rate (E)

The unit leaf rate is a measure of increment in dry weight per unit leaf area per day. It thus gives a measure of the photosynthetic efficiency of the leaves. Williams (1946) provided a formula for the estimation of mean unit leaf rate (E) over a period of time:

$$_{1\text{-}2}\bar{E} = \frac{_2W - _1W}{_2T - _1T} \cdot \frac{\log_e {_2LA} - \log_e {_1LA}}{_2LA - _1LA}$$ (Williams, 1946)

Where:

$_1W$ = the total plant dry weight at time $_1T$

$_2W$ = the total plant dry weight at time $_2T$

$_2T - _1T$ = the time interval between harvests

e = the base of natural logarithms

$_1LA$ = the total leaf area at time $_1T$

$_2LA$ = the total leaf area at time $_2T$

This expression makes the assumption that weight and leaf area are linearly related over the period of observation. Hunt (1978) however, states that there are two conditions under which the above equation is likely to be untrue. They are:

1. when plants are growing quickly,
2. when the harvest intervals are long.

Various other formulae are, however, available in cases where total plant weight is not linearly related to leaf area. A method for solving problems of non-linearity has been put forward by Combe (1960), Evans and Hughes (1962) and elaborated by Evans (1972) as:

$$_{1\text{-}2}\bar{\bar{E}} = \frac{_2W - {_1W}}{_2T - {_1T}} \cdot \frac{2}{_2LA - {_1LA}}$$

For the present investigation the equation:

$$_{1\text{-}2}\bar{\bar{E}} = \frac{_2W - {_1W}}{_2T - {_1T}} \times \frac{\text{Log}_e\, {_2LA} - \text{Log}_e\, {_1LA}}{_2LA - {_1LA}}$$

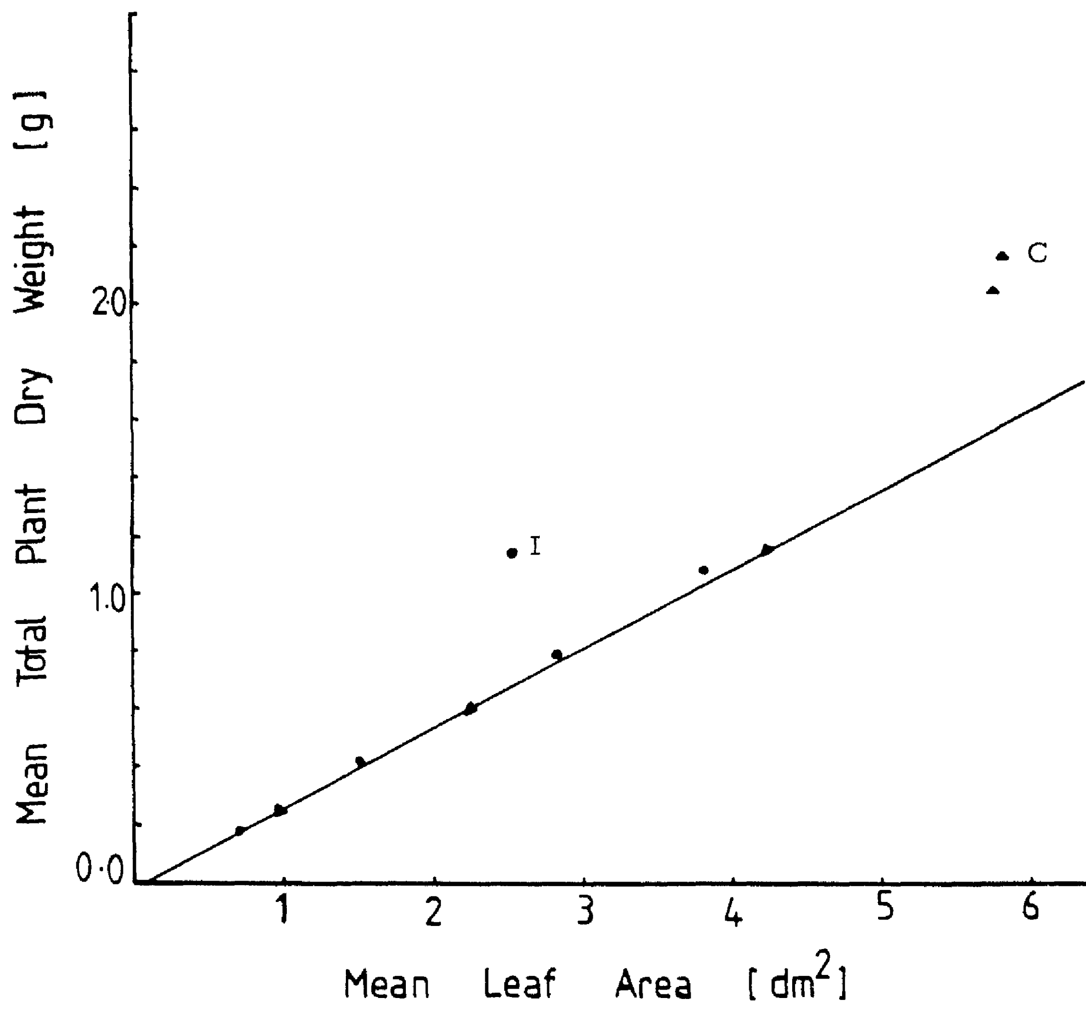
was adopted because the total plant weight and leaf area are linearly related (See Figure 2) and for the additional reason that the time interval between harvests was relatively short being only seven days.

<u>The relative growth rate (RGR)</u>

The relative growth rate represents the efficiency of the plant as a producer of new material, (Blackman, 1919). It is defined as the increase in plant weight per unit weight per unit time, (Fisher, 1921). The mean relative growth rate is derived from the formula:

$$_{1\text{-}2}\bar{R} = \frac{\text{Log}_e\, {_2W} - \text{Log}_e\, {_1W}}{_2T - {_1T}}$$

FIG. 2



The relationship between total plant dry weight and leaf area of infected and un-infected plants.

I = Infected

C = Control

All the growth indices described above can be shown to be related as follows:

$$LAR = SLA \times LWR$$

$$RGR = ULR \times LAR$$

Other ratios have also been constructed (Hunt, 1978) for the determination of the distribution of dry matter along the plant axis. The more frequently used ones are the ratio of root weight to shoot weight, the ratio of root weight to total dry weight, and the ratio of shoot weight to total dry weight.

Determination of the effect of infection on the size of the lower epidermal cells

Leaves from positions 9, 11, 8, 12 and 13 on the main axis showing 40%, 55%, 60%, 70% and 100% mildew cover respectively were excised from five infected plants showing 70%, 75%, 70%, 80% and 100% total mildew cover respectively. Leaves from corresponding positions were excised from five un-infected plants. The area of all the leaves in both groups were measured with the leaf area meter. Lower epidermal strips were then obtained by gently peeling off the lamina of each leaf, taking care to remove the strips from similar areas on both infected and un-infected leaves. The lower epidermis was used in preference to the upper epidermis because it could be removed more easily. After removal, the strips were separately immersed in distilled water and fungal mycelia and conidia were brushed off the infected strips with a camel hair brush. Each lower epidermal strip was stained in 1% cotton blue in lactophenol and then mounted in glycerol. The numbers of guard cells per unit area were determined microscopically by counting the numbers per field of

view (0.09083 mm^2) at a magnification of x 320. Five counts per strip were made and the mean of these was taken as the number of guard cells per unit area for that epidermal strip. A total of ten strips per leaf were determined. For leaf 13, a count of the other epidermal cells was also made.

The sizes of about 25 guard cells from the leaves on position 13 were determined by measuring their length and breadth at a magnification of x 400 using an ocular micrometer.

Measurements of water loss per unit area of infected and un-infected leaves

Leaves with 10%, 25%, 50%, 75% or 95% mildew cover were excised from a series of infected plants. Three leaves with each level of infection were taken. Fifteen leaves were then excised from corresponding positions on un-infected plants. Each leaf was weighed immediately after detachment and its area measured. The leaves were then suspended by paper clips on a string in the growth room. The leaves were re-weighed at two-hourly intervals over a period of 12 hours to determine water loss.

RESULTS

The effect of infection on the functioning of the leaf tissue

Infection of the leaf tissue could affect dry matter production by interfering with photosynthesis. This was investigated by examining the effects of infection on the chlorophyll content per unit area of leaf; the unit leaf rate and other growth parameters.

Development of infection during the course of the experiment

Within five to six days of introducing heavily infected plants into the growth room containing three week old healthy plants, colonies of Erysiphe fischeri appeared on the lower leaves of non-benomyl treated plants. The subsequent spread of infection was very rapid and ranged from 17.2% at the sixth week after sowing to 90% at the tenth week after sowing.

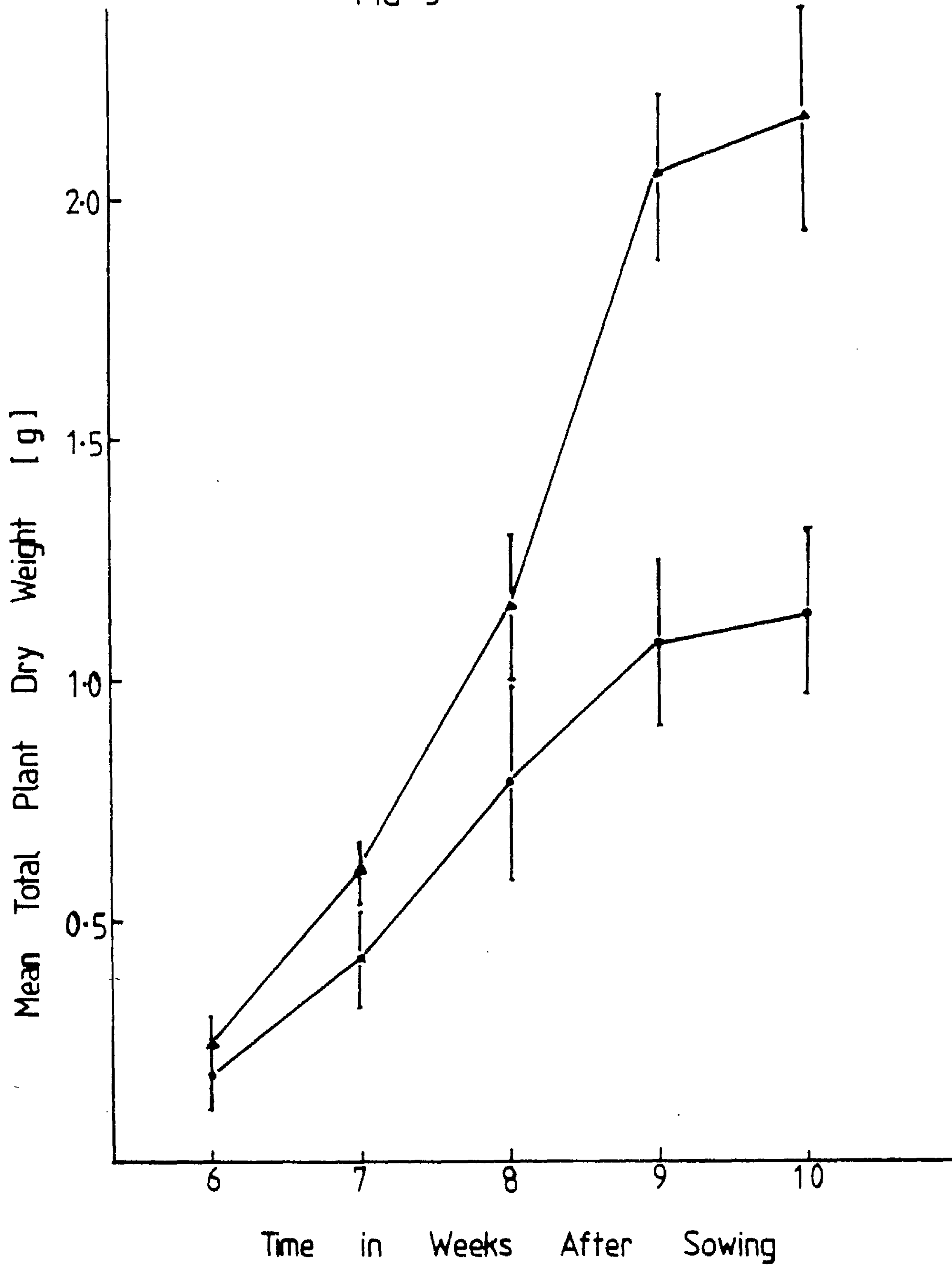
Mildew intensity during the course of this study was much higher than that which is usually found under natural conditions. Colonies of the mildew covered all the aerial parts of infected plants including the inflorescences (Plate 1). The results are presented in Appendix Tables 6A - 6E and summarised in Text Tables 1 - 4.

Effect of infection on the accumulation and distribution of dry matter

Total plant dry weights and dry weights of roots, stems and leaves of infected and un-infected plants at each harvest are given in Table1A and the results on total plant dry weights only are plotted graphically in Figure 3.

The results show that although the total plant dry weights of infected plants are smaller than those of un-infected plants at harvests, none of the differences were significant until the seventh week after sowing when total mildew cover was about 30%. The dry weight of infected plants showed a 30% reduction over that of un-infected plants by the seventh week and a reduction of about 47.25% by the tenth week when total mildew cover was about 90%.

PLATE 1: Photomicrograph illustrating morphological differences between groundsel plants heavily infected with *E. fischeri* (more than 95% infection) and those treated with benomyl.



Mean total plant dry weights of E. fischeri infected (●———●) and un-infected (▲———▲) plants determined at weekly intervals.

TABLE 1A

Data on dry weights of _E. fischeri_ infected (I) and non-infected (C) plants. Each reading is a mean of five plants.

Harvests	Time in weeks after sowing		% Infection	Dry Weights (g)			
				Root	Stem	Leaves	Total
1st	6	I	17.2	0.01814 $\pm$ .007	0.01514 $\pm$ .01	0.14458 $\pm$ .05	0.17786 $\pm$ .07
		C	0	0.02508 $\pm$ .008	0.02414 $\pm$ 0.01	0.19008 $\pm$ 0.05	0.23930 $\pm$ .06
		t		NS	NS	NS	NS
		P					
2nd	7	I	30	0.0487 $\pm$ 0.01	0.05104 $\pm$ 0.01	0.31760 $\pm$ .080	0.41734 $\pm$ .106
		C	0	0.07648 $\pm$ 0.01	0.07232 $\pm$ 0.01	0.45148 $\pm$ .050	0.60028 $\pm$ .065
		t		S	S	S	S
		P		$<$ 0.001	$<$ 0.01	$<$ 0.05	$<$ 0.01
3rd	8	I	62	0.08708 $\pm$ 0.01	0.15612 $\pm$ 0.06	0.55094 $\pm$ 0.13	0.79414 $\pm$ .200
		C	0	0.12872 $\pm$ 0.02	0.21596 $\pm$ 0.05	0.80942 $\pm$ 0.12	1.15410 $\pm$ .150
		t		S	NS	S	S
		P		$<$ 0.001		$<$ 0.01	$<$ 0.01
4th	9	I	73	0.10482 $\pm$ 0.01	0.25896 $\pm$ 0.03	0.72076 $\pm$ 0.14	1.08456 $\pm$ .170
		C	0	0.19404 $\pm$ 0.03	0.58956 $\pm$ 0.11	1.26284 $\pm$ 0.07	2.04642 $\pm$ .170
		t		S	S	S	S
		P		0.001	0.001	0.001	0.001
5th	10	I	90	0.09700 $\pm$ 0.01	0.30900 $\pm$ 0.06	0.73740 $\pm$ 0.14	1.14340 $\pm$ .171
		C	0	0.16818 $\pm$ 0.03	0.60812 $\pm$ 0.10	1.39116 $\pm$ 0.17	2.16746 $\pm$ .230
		t		S	S	S	S
		P		$<$ 0.001	$<$ 0.001	$<$ 0.001	$<$ 0.001

NS = Not significant at P = 0.05

The ratios of root dry weights to shoot dry weights, and of root dry weights, shoot dry weights and leaf dry weights to total plant dry weight are given in Table 1B and the results are plotted graphically in Figures 4A - 4D.

These results show that the proportion of the total dry matter contained in the roots or shoots of infected plants was not significantly different from that of un-infected plants at any harvest. This experiment was repeated two further times and similar results were obtained in each case. Thus in general mildew infection reduces dry matter production when the level of infection reaches about 30%, but it has little effect on the distribution of dry matter; the roots, stems and leaves being stunted in equal proportions.

Effect of infection on the development of leaf tissue and to photosynthetic efficiency

(i) Effect of Infection on the numbers of expanded leaves

The total numbers of leaves present at each harvest on infected and un-infected plants are given in Table 2A and the results on total number of leaves are plotted graphically in Figure 5A.

The total numbers of leaves on infected plants were significantly less ($P < 0.05$) at the seventh and subsequent weeks after sowing when the total mildew cover was over 30%.

The first senescent leaves were observed on both infected and un-infected plants seven weeks after sowing and the number of senescent leaves on infected plants was always significantly greater than that on un-infected plants (Table 2A).

FIGURES 4A - 4D: The distribution of dry matter along the plant axis of *E. fischeri* infected (●———●) and non-infected (▲———▲) plants in the progression of (A) root dry weight to shoot dry weight (B) root dry weight to total plant dry weights; (C) shoot dry weight to total plant dry weights and (D) leaf dry weight to total plant dry weights.

FIG. 4.

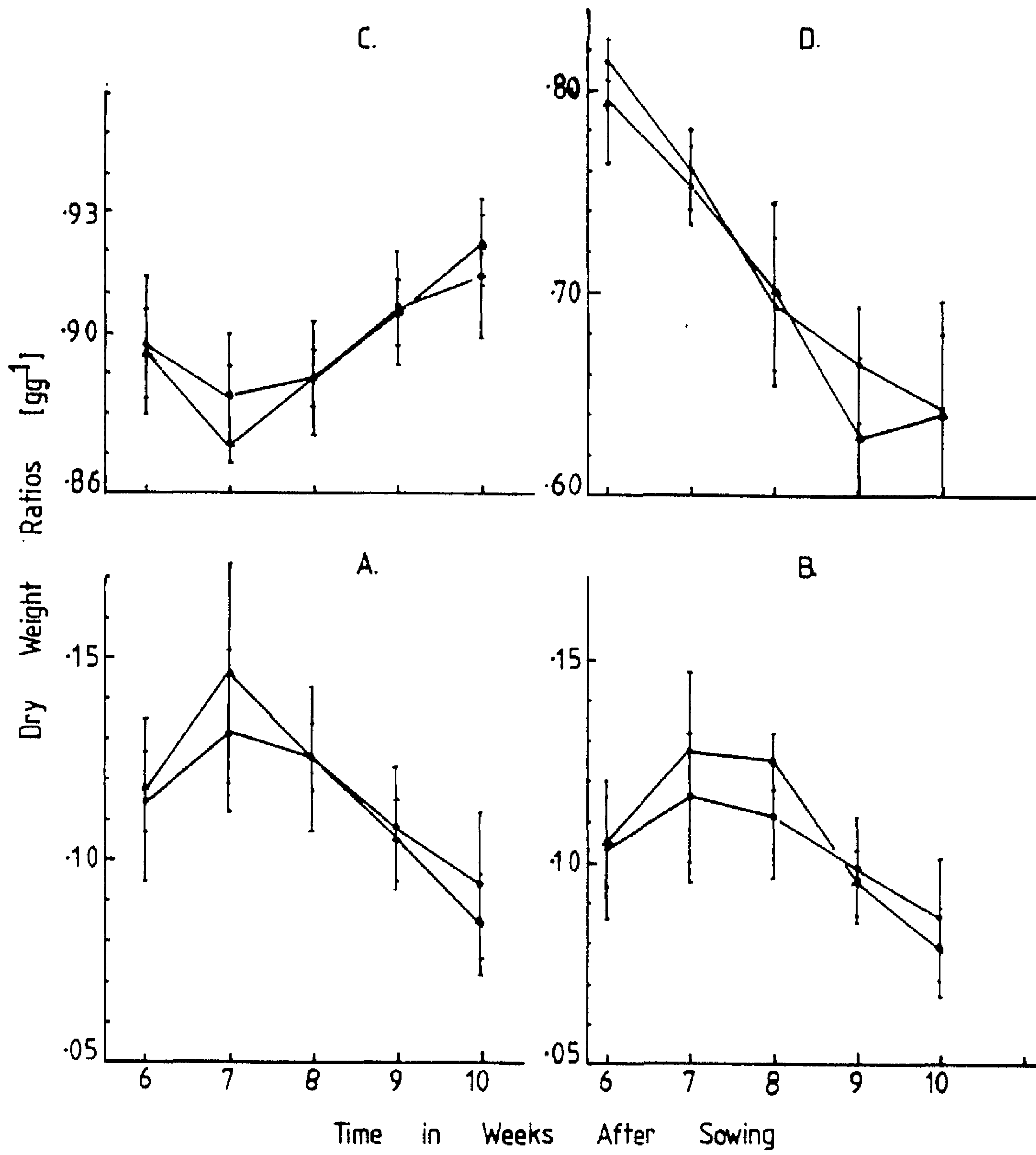


TABLE 1B

Date on dry weight ratios of *E. fischeri* infected (I) and non-infected (C) plants. Each reading is a mean of five plants

Harvests	Time in weeks after sowing		% Infection	Dry Weight Ratios (gg^{-1})				
				Root/Shoot	Root/W	Shoot/W	Stem/W	Leaves/W
1st	6	I	17.2	0.11507 ± .022	0.10302 ± 0.017	0.89698 ± 0.017	0.08332 ± 0.016	0.81366 ± 0.0[illegible]
		C	0	0.11694 ± 0.014	0.10462 ± 0.011	0.89538 ± 0.011	0.10078 ± 0.022	0.79460 ± 0.0[illegible]
		t		NS	NS	NS	NS	NS
2nd	7	I	30	0.13192 ± 0.021	0.11638 ± 0.016	0.88362 ± 0.016	0.12309 ± 0.013	0.76052 ± 0.0[illegible]
		C	0	0.14650 ± 0.027	0.12752 ± 0.021	0.87247 ± 0.021	0.12062 ± 0.014	0.75186 ± 0.0[illegible]
		t		NS	NS	NS	NS	NS
3rd	8	I	62	0.12503 ± 0.018	0.11102 ± 0.014	0.88898 ± 0.014	0.19447 ± 0.028	0.69451 ± 0.0[illegible]
		C	0	0.12531 ± 0.009	0.11138 ± 0.007	0.88867 ± 0.007	0.18799 ± 0.043	0.70068 ± 0.0[illegible]
		t		NS	NS	NS	NS	NS
4th	9	I	73	0.10826 ± .015	0.09794 ± .013	0.90559 ± .014	0.24047 ± .020	0.66513 ± .0[illegible]
		C	0	0.10459 ± .01	0.09465 ± .008	0.90535 ± .008	0.28691 ± .037	0.61843 ± .0[illegible]
		t		NS	NS	NS	NS	NS
5th	10	I	90	0.09377 ± 0.018	0.08560 ± 0.015	0.91419 ± 0.015	0.27098 ± 0.043	0.64341 ± 0.0[illegible]
		C	0	0.08432 ± 0.013	0.07769 ± 0.011	0.92231 ± 0.011	0.28034 ± 0.032	0.64197 ± 0.0[illegible]
		t		NS	NS	NS	NS	NS

NS = Not significant at P = 0.05

FIGURE 5A: Effect of infection on total number of leaves:

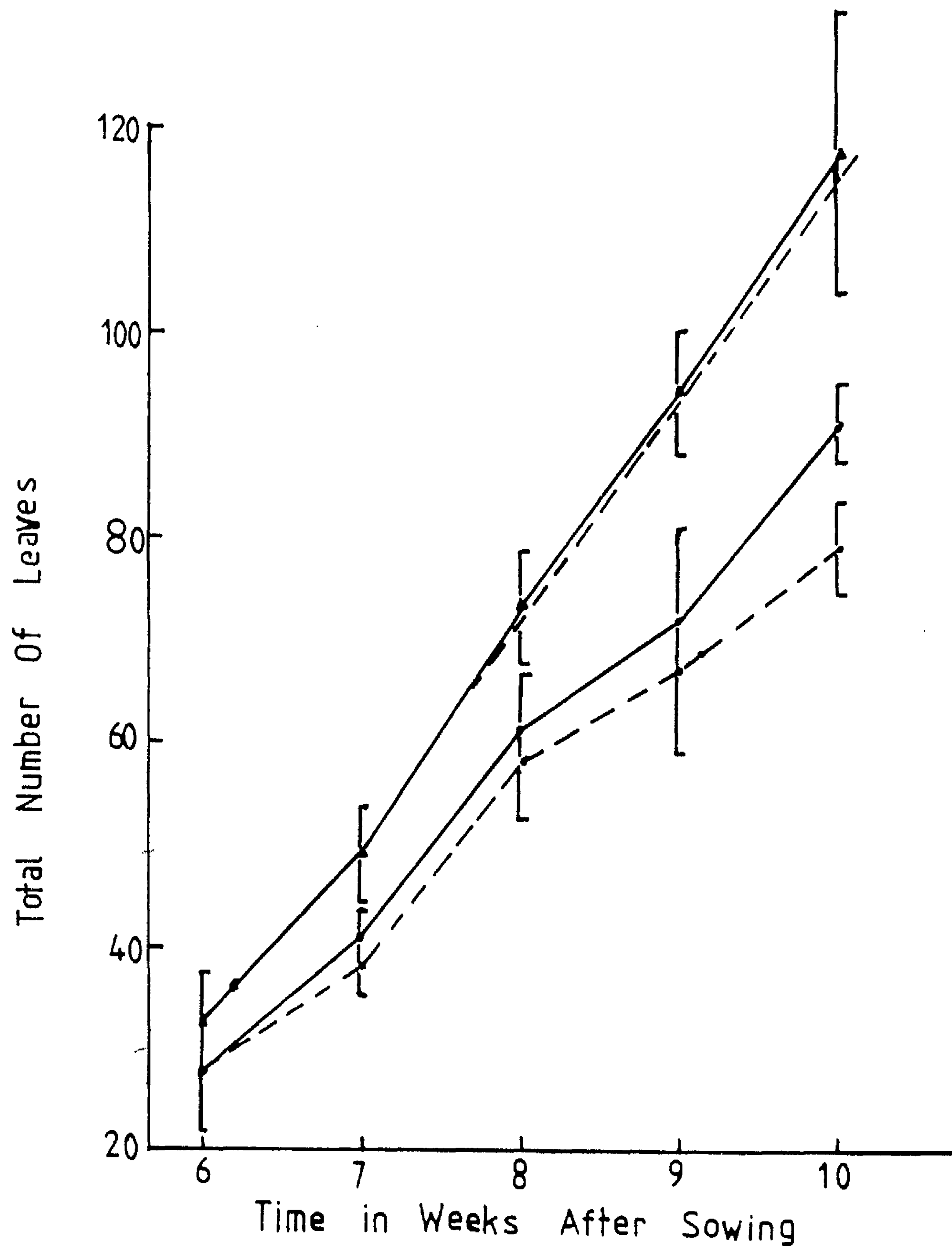
▲———▲ Total number of leaves (Control)

▲- - - -▲ Total number of green leaves (Control)

●———● Total number of leaves (Infected)

●- - - -● Number of green leaves (Infected)

FIG. 5A



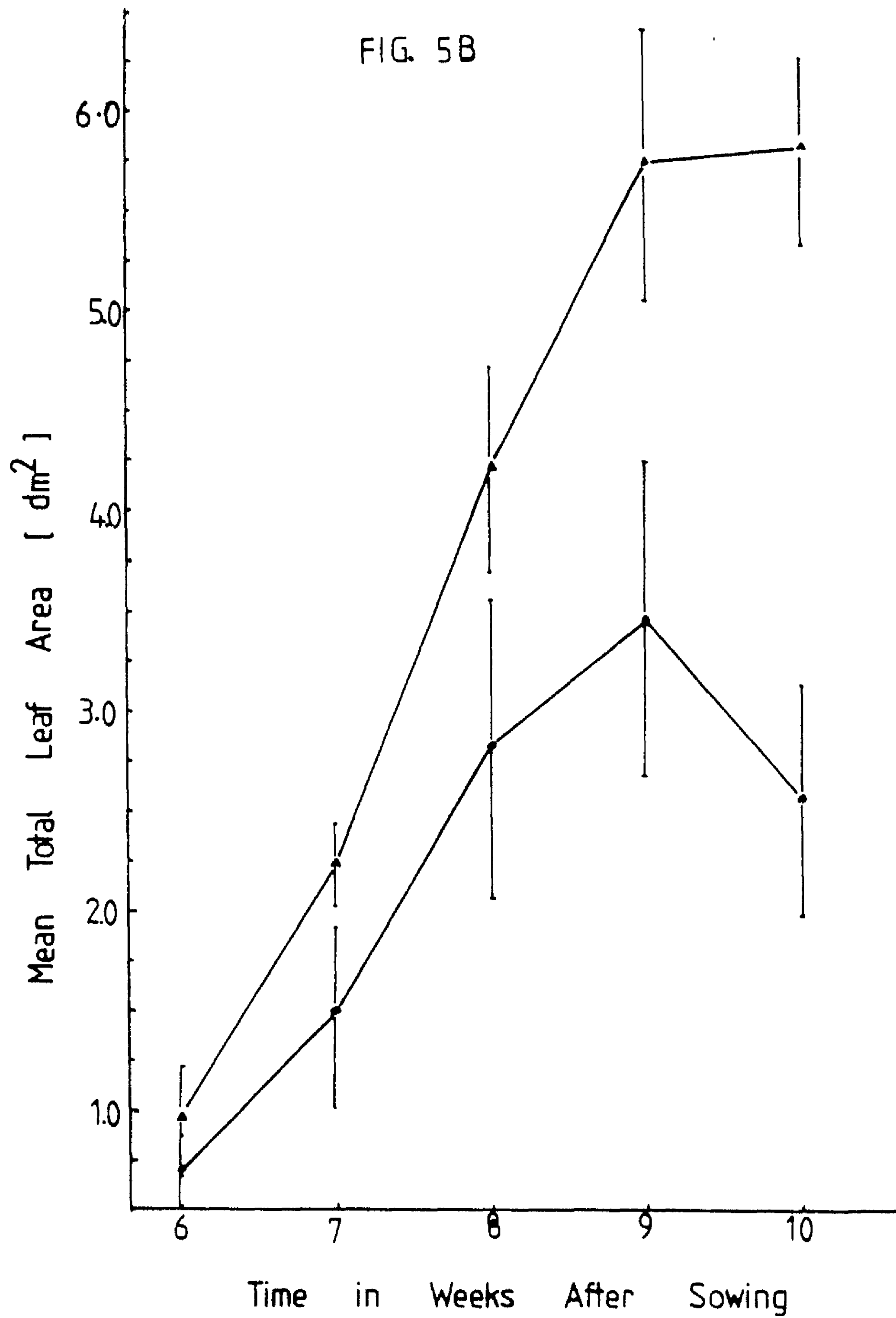
(ii) Effect of Infection on total leaf area

Total leaf areas of infected and un-infected plants at every harvest are given in Table 2A and the results are plotted graphically in Figure 5B. The leaf areas of un-infected plants increased throughout the period of investigation, but those of infected plants increased up to the ninth week after sowing and then declined when total mildew cover was about 73%. The decline may have been partly due to a reduction in the number of leaves expanding at the apex but was mainly due to the loss of the lower leaves through senescence. Although total leaf area of infected plants was smaller than that of un-infected plants at every harvest, the differences did not become significant until seven weeks after sowing when total mildew cover was over 30%. Thus the effects of infection on leaf expansion became manifest at about the same time as the effects on dry matter production. This experiment was repeated two further times and similar results were obtained.

Effect of infection on the relative growth rate of the leaves

The relative growth rates of the leaves on infected and un-infected plants are given in Table 3.

The results show a distinct ontogenetic drift in the relative growth rates of the leaves of both infected and un-infected plants, the relative growth rate being high during the early stages of growth and low as the plants started flowering at about the tenth week after sowing. A comparison of the relative growth rates of the leaves of infected and un-infected plants showed a significant difference ($P < 0.01$) between the ninth and tenth weeks after sowing when total mildew cover was over 73%.



Leaf area development of E. fischeri infected (●———●) and un-infected (▲———▲) plants taken at weekly intervals.

TABLE 2A

Primary values of growth of _E. fischeri_ infected (I) and non-infected (C) plants taken at weekly intervals. Each reading is a mean of five plants.

Harvests	Time in weeks after sowing	% Mildew cover		Total No. of leaves	Mean No. of Senescent leaves	Mean No. of Non-senescent leaves	Mean Leaf area (dm^2)
1st	6	17.2	I	27.8 $\pm$ 6.40	-	27.8 $\pm$ 6.40	0.7128 $\pm$ .18
			C	32.8 $\pm$ 5.52	-	32.8 $\pm$ 5.52	0.9613 $\pm$ .28
			t	NS		NS	NS
			P				
2nd	7	30	I	40.8 $\pm$ 2.28	2.60 $\pm$ 1.42	38.2 $\pm$ 2.69	1.4870 $\pm$ 0.46
			C	49.0 $\pm$ 4.12	0.20 $\pm$ 0.56	48.8 $\pm$ 4.52	2.2334 $\pm$ 0.22
			t	S	S	S	S
			P	< 0.01	< 0.01	< 0.001	< 0.01
3rd	8	62	I	61.0 $\pm$ 5.50	2.60 $\pm$ 1.11	58.4 $\pm$ 5.31	2.8156 $\pm$ 0.76
			C	72.6 $\pm$ 5.66	0.60 $\pm$ 0.68	72.0 $\pm$ 5.63	4.2180 $\pm$ 0.51
			t	S	S	S	S
			P	< 0.01	< 0.01	< 0.01	< 0.01
4th	9	73	I	72.2 $\pm$ 8.76	5.6 $\pm$ 1.42	66.6 $\pm$ 9.85	3.4525 $\pm$ 0.79
			C	94.2 $\pm$ 6.4	1.8 $\pm$ 1.04	92.4 $\pm$ 7.74	5.7339 $\pm$ 0.68
			t	S	S	S	S
			P	< 0.001	< 0.001	< 0.001	< 0.001
5th	10	90	I	90.6 $\pm$ 3.69	11.6 $\pm$ 5.46	79.0 $\pm$ 4.57	2.5545 $\pm$ 0.57
			C	117.4 $\pm$ 13.60	2.8 $\pm$ 2.22	114.6 $\pm$ 12.60	5.7985 $\pm$ 0.47
			t	S	S	S	S
			P	< 0.001	< 0.01	< 0.001	< 0.001

NS = Not significant at $P = 0.05$

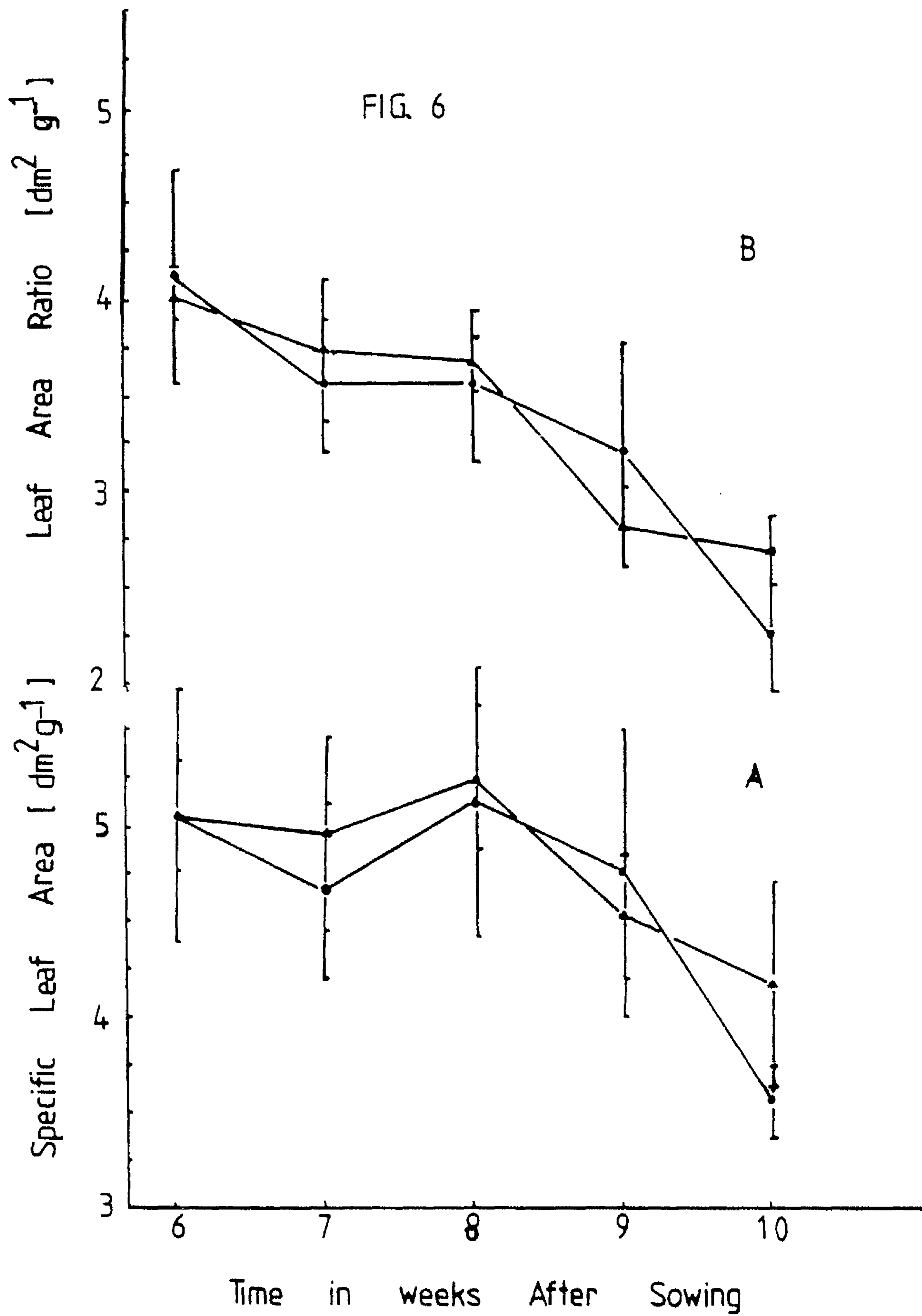
The values of specific leaf area and leaf area ratio of infected and un-infected plants are given in Table 2B and plotted graphically against time in Figures 6A and 6B.

In general these two growth indices showed a negative correlation with time, their values being high during the early stages of growth and low as the plants matured. The results in Table 2B show that the specific leaf areas and leaf area ratios of infected plants were not significantly different from those of un-infected plants at any harvest. It thus appears that although infection reduced the leaf area, the size of the leaves was proportional to the size of the plant, and infection did not appear to alter the growth pattern of the infected plants.

Effect of infection on chlorophyll content

The chlorophyll content per unit area of leaf and per unit dry weights of leaf and stem at each harvest are given in Table 2B and the results on the chlorophyll content per unit area only is plotted graphically in Figure 7.

A comparison of the chlorophyll content per unit area or per unit weight of leaf or stem of infected and un-infected plants showed no significant differences at any harvest. Similar results were obtained in the two repeat experiments. It is thus unlikely that the reduction in dry matter production by the infected plants was due to any effects of infection on the chlorophyll content of the leaf tissue.



Effect of infection on specific leaf area (6A) and leaf area ratio (6B) of groundsel, taken at weekly intervals.

●———● infected

▲———▲ un-infected

TABLE 2B

Primary values of growth of *E. fischeri* infected (I) and non-infected (C) plants taken at weekly intervals. Each reading is a mean of five plants.

Harvests	Time in weeks after sowing	% Mildew cover		Mean Chlorophyll Unit Area (mg/dm^2) SE	Mean chlorophyll per unit weight (mg/g)	
					Stem SE	Leaves SE
1st	6	17.2	I	0.2345 ± 0.04	0.4004 ± .28	0.1619 ± .16
			C	0.2387 ± 0.03	0.3867 ± .10	0.1968 ± .14
			t	NS	NS	NS
2nd	7	30	I	0.3520 ± 0.07	0.3715 ± .16	1.6260 ± .64
			C	0.3302 ± 0.05	0.3304 ± .15	1.6411 ± .51
			t	NS	NS	NS
3rd	8	62	I	0.3184 ± 0.06	0.5031 ± 0.06	1.6228 ± 0.30
			C	0.2936 ± 0.03	0.4351 ± 0.09	1.5354 ± 0.16
			t	NS	NS	NS
4th	9	73	I	0.3062 ± 0.05	0.5435 ± 0.09	1.5014 ± 0.29
			C	0.2598 ± 0.06	0.4940 ± 0.20	1.3079 ± 0.28
			t	NS	NS	NS
5th	10	90	I	0.2422 ± 0.04	0.5613 ± 0.08	1.1154 ± 0.16
			C	0.2508 ± 0.03	0.4692 ± 0.07	1.0118 ± 0.06
			t	NS	NS	NS

Harvests		Mean SLA ($dm^2 g^{-1}$) SE	Mean LAR ($dm^2 g^{-1}$) SE
1st	I	5.0469 ± 0.67	4.1089 ± 0.58
	C	5.0526 ± 0.29	4.0107 ± 0.12
	t	NS	NS
2nd	I	4.6712 ± 0.47	3.5524 ± 0.37
	C	4.9673 ± 0.52	3.7328 ± 0.38
	t	NS	NS
3rd	I	5.1251 ± 0.70	3.5518 ± 0.40
	C	5.2366 ± 0.36	3.6624 ± 0.15
	t	NS	NS
4th	I	4.7640 ± .76	3.1759 ± .59
	C	4.5336 ± .34	2.8027 ± .21
	t	NS	NS
5th	I	3.5556 ± 0.18	2.2272 ± 0.29
	C	4.1936 ± 0.54	2.6819 ± 0.19
	t	NS	NS

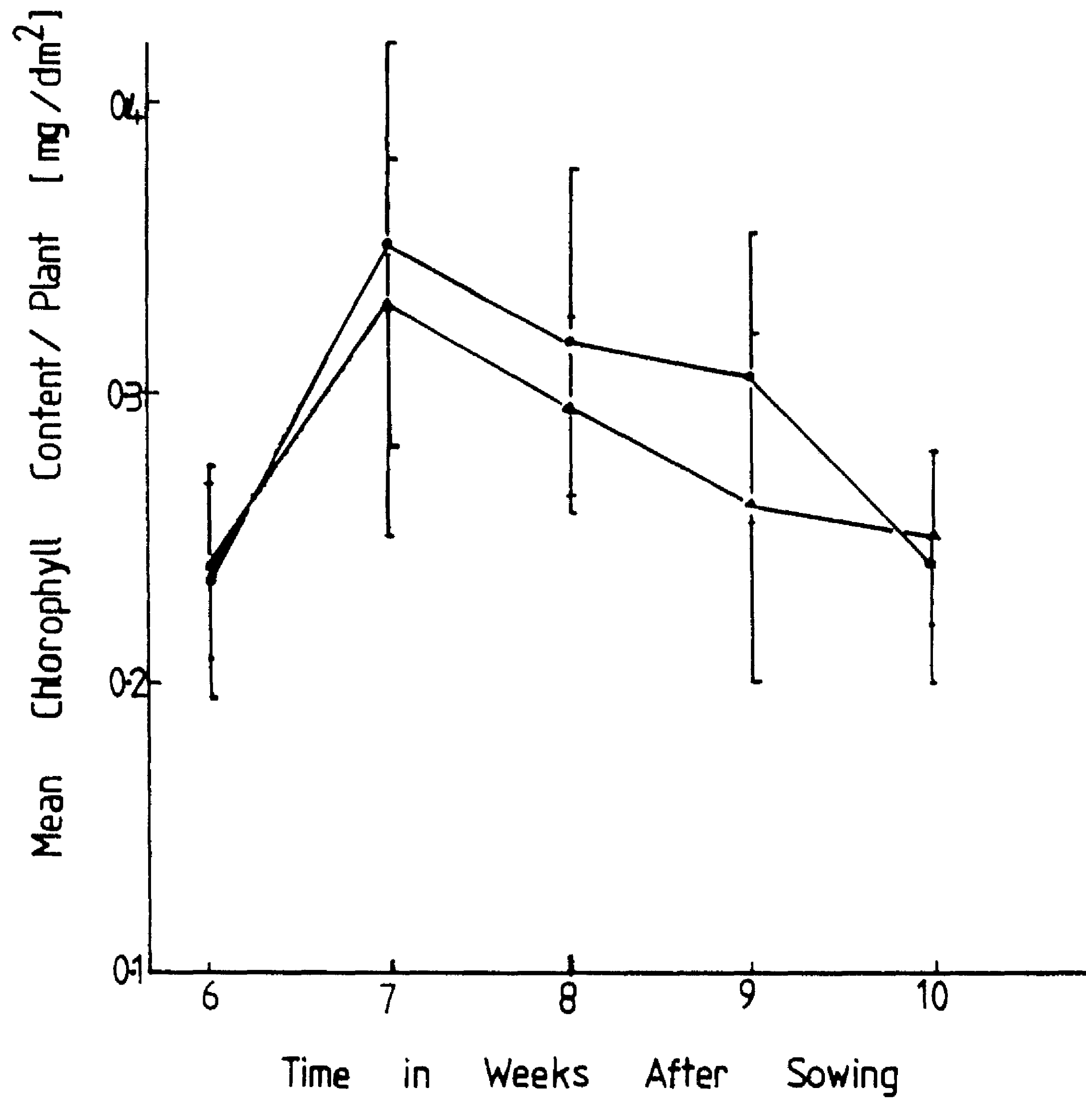
NS = Not significant at $P = 0.05$

TABLE 3

The mean relative growth rates of infected (I) and un-infected (C) leaves.

Time in weeks after sowing	% Infection	Mean relative growth rate of leaves				t
		I	SE	C	SE	
6 - 7	17.2 - 30	0.1044	± 0.020	0.1228	± 0.027	NS
7 - 8	30 - 62	0.0919	± 0.008	0.0906	± 0.005	NS
8 - 9	62 - 73	0.0284	± 0.023	0.0439	± 0.003	NS
9 - 10	73 - 90	-0.0417	± 0.028	0.0019	± 0.011	S

FIG. 7



Mean chlorophyll content of E. fischeri infected (•——•) and un-infected (▲——▲) groundsel leaves, taken at weekly intervals.

Effect of infection on unit leaf rate

The values of the unit leaf rates of infected and un-infected plants are given in Table 4 and the results are plotted graphically against time in Figure 8.

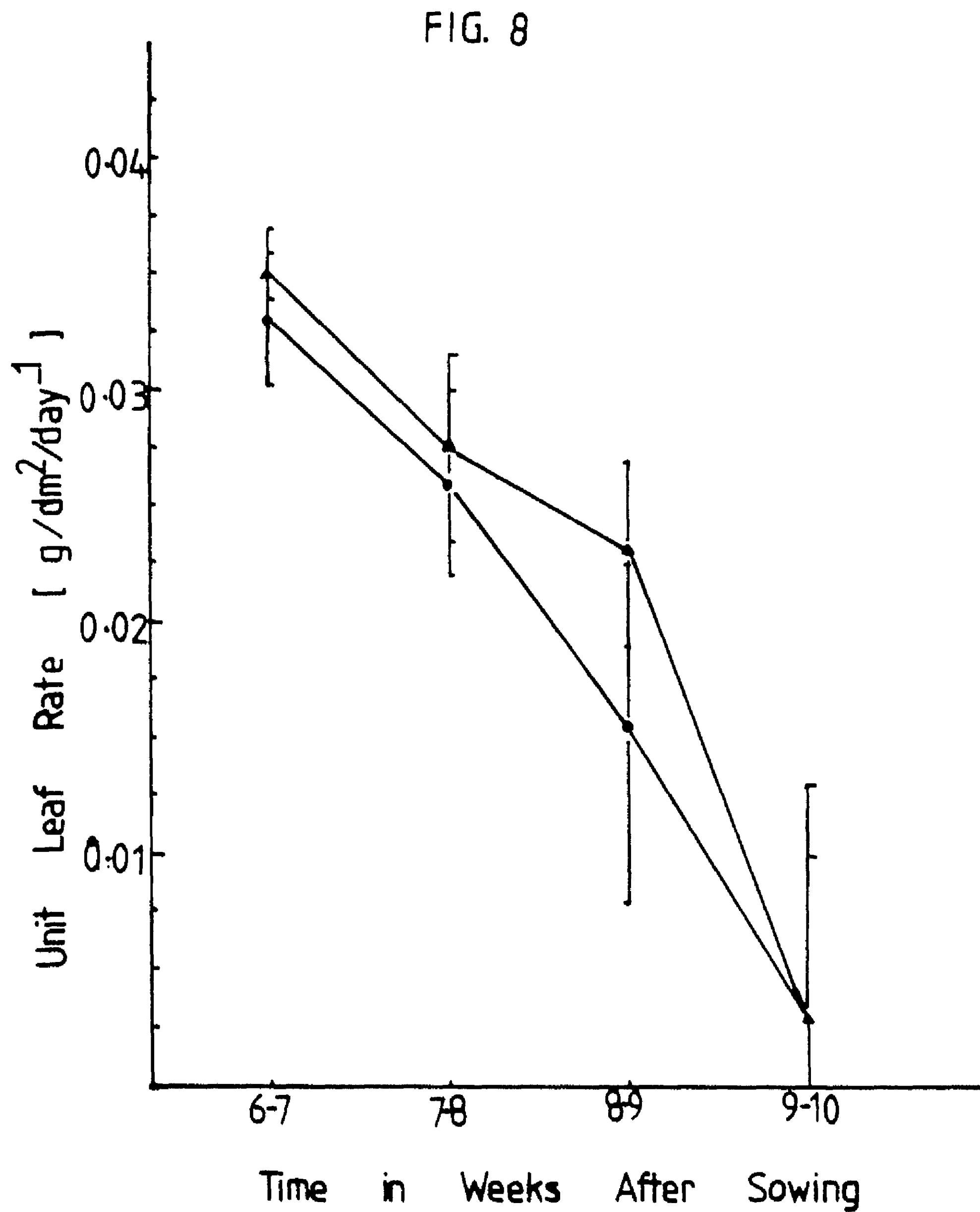
TABLE 4

The unit leaf rates of infected (I) and un-infected (C) plants.

Time in weeks after sowing	% Mildew cover	Mean unit leaf rates (gdm^2 day^{-1})		t
		I SE	C SE	
6 - 7	15 - 29	0.0331 ± 0.004	0.0350 ± 0.001	NS
7 - 8	29 - 62	0.0261 ± 0.004	0.0252 ± 0.004	NS
8 - 9	62 - 73	0.0155 ± 0.007	0.0232 ± 0.004	NS
9 - 10	73 - 90	0.0030 ± 0.009	0.0030 ± 0.007	NS

The curves for both infected and un-infected plants show a negative correlation with time and total plant dry weight. The downward trend of the curves however, indicates that as the plants grow older the amount of photosynthates produced per unit area of leaf decreases.

Results in Table 4 show that the unit leaf rates of infected plants was not significantly smaller than that of un-infected plants at levels of infection up to 90% cover. In the two repeat experiments the unit leaf rates of the infected plants were significantly (0.01) smaller than that of the control at levels of infection over 95% cover whereas dry matter production was reduced at about 30% infection. This indicates that the



Effect of infection on the unit leaf rates of E. fischeri infected (●———●) and un-infected (▲———▲) plants, taken at weekly intervals.

reduction in dry matter production was not primarily due to a reduction in the efficiency of the leaves as photosynthesizing organs but appears to be due to the reduction in leaf area since leaf area and dry matter production were significantly reduced at the same time.

The reduction in leaf area may be due to a reduction in cell division or a reduction in cell expansion during leaf development. These aspects of growth were then investigated and the results are reported in the next Section.

Effect of infection on the development and expansion of the leaf tissue

This study was carried out with a view to establishing more clearly the events responsible for the reduction in leaf number and leaf area observed in the previous experiments. In this experiment the effects of infection on the development of individual leaves on the main axis and on total leaf area on the lateral branches were examined.

Development of infection during the course of the experiments

Infection was first observed on the lower leaves of the non-benomyl treated plants, five to six days after introducing the infected plants into the growth room. By this time the plants were about five weeks old and most of their lower leaves appeared fully expanded. The subsequent spread of infection was very rapid and ranged from about 33.3%, eight weeks after sowing to about 99.97% at the twelfth week.

Infection was first observed on the lower internodes of the stem at about the ninth week after sowing and by the twelfth week the entire stem surface was colonized.

Effect of infection on the expansion of leaves

(a) On the main axis

The number of leaves on the main axis of infected and un-infected plants are given in Appendix Tables 7AA - 7EA and the results are summarised in Text Table 5.

Both the infected and un-infected plants had produced about twenty-five leaves by the fifth harvest after which leaf initiation was terminated by terminal inflorescence development. There was, however, no significant difference between the number of leaves initiated on the main axis of infected and un-infected plants at any harvest, Table 5. This was probably due to leaf initiation being completed in the apical meristem before infections were established.

The areas of individual leaves along the main axis of infected and un-infected plants at each harvest are given in Appendix Tables 7AA - 7EA and the results are plotted as histograms in Figures 9A - 9E.

Although infection did not significantly affect the areas of apparently fully expanded lower leaves, it did significantly ($P < 0.02$) reduce the areas of the developing upper leaves. The extent of the reduction in leaf areas appears to be dependent upon the time the leaves became infected, because the leaves in the lower position were reduced in area less than the leaves at higher positions on the main axis.

At levels of infection above 67% total mildew cover, even the apparently un-infected leaves or slightly infected leaves (about 5%

TABLE 5

Effect of infection on the development of leaves on the main axis and on the lateral branches.

Time in weeks after sowing		Total mildew cover %	Mean total No. of leaves on main axis	Mean total No. of lateral branches	Mean total No. of leaves on lateral branches
8	I	33.33	20.33±1.43	7.67±1.43	30.00± 4.96
	C	0.00	20.67±1.43	10.33±2.87	39.00±13.67
	t		NS	NS	NS
9	I	67.50	22.00±0.00	9.50±6.35	45.00±12.71
	C	0.00	22.50±6.35	12.50±6.35	60.00±12.71
	t		NS	NS	NS
10	I	85.00	25.00±2.48	11.67±5.17	55.67±22.25
	C	0.00	24.33±1.43	17.33±3.79	91.00± 8.74
	t		NS	NS	S (P<0.001)
11	I	91.67	24.00±0.00	18.67±1.43	77.67±17.96
	C	0.00	26.00±2.48	20.33±1.43	114.00± 8.60
	t		NS	NS	S(P<0.001)
12	I	99.97	24.67±2.87	17.67±5.23	74.33± 11.19
	C	0.00	25.00±2.48	20.33±3.79	133.67± 1.43
	t		NS	NS	S(P<0.001)

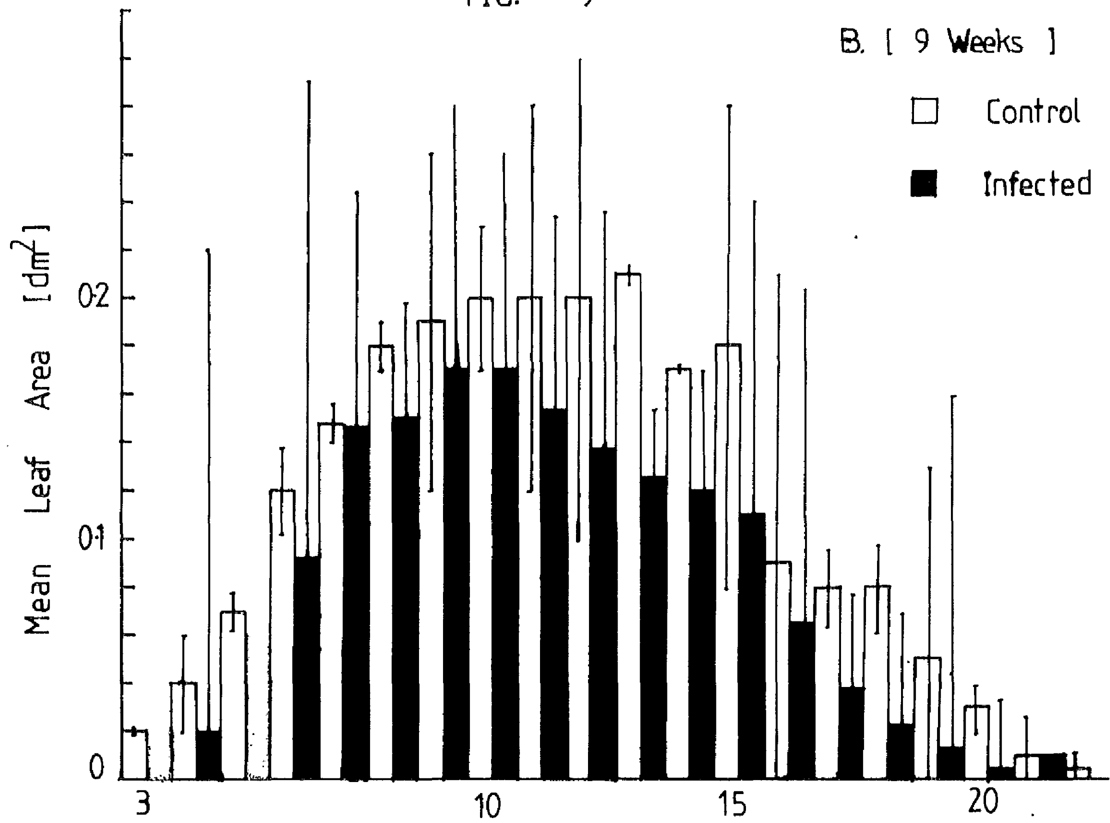
NS = Not significant at P = 0.05

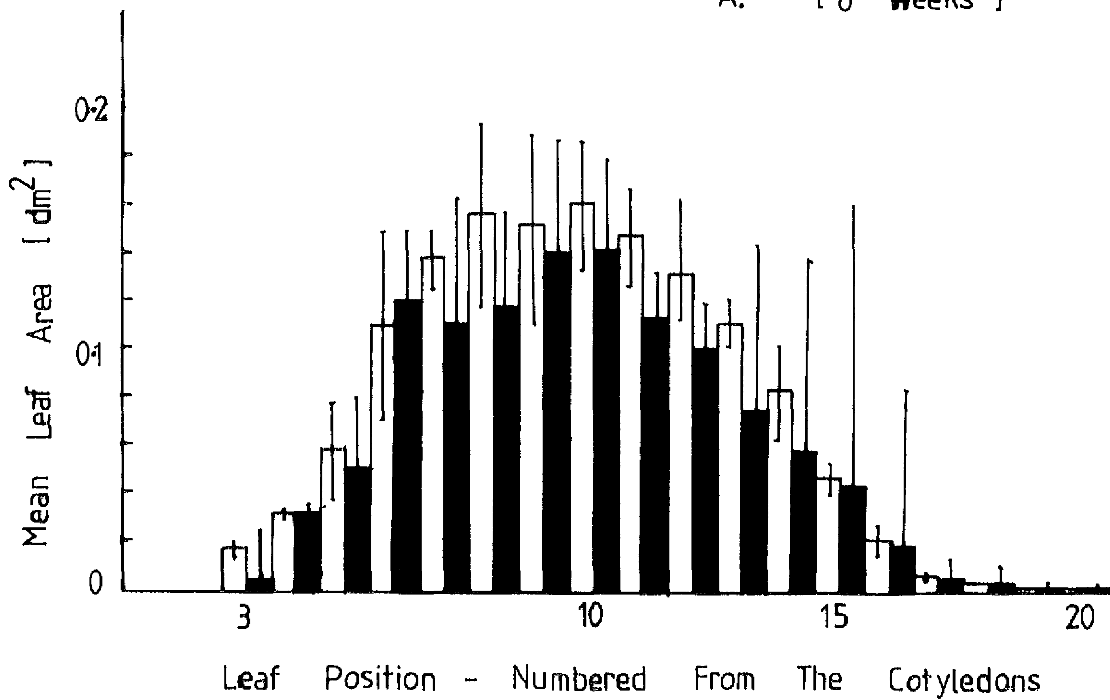
I = Infected

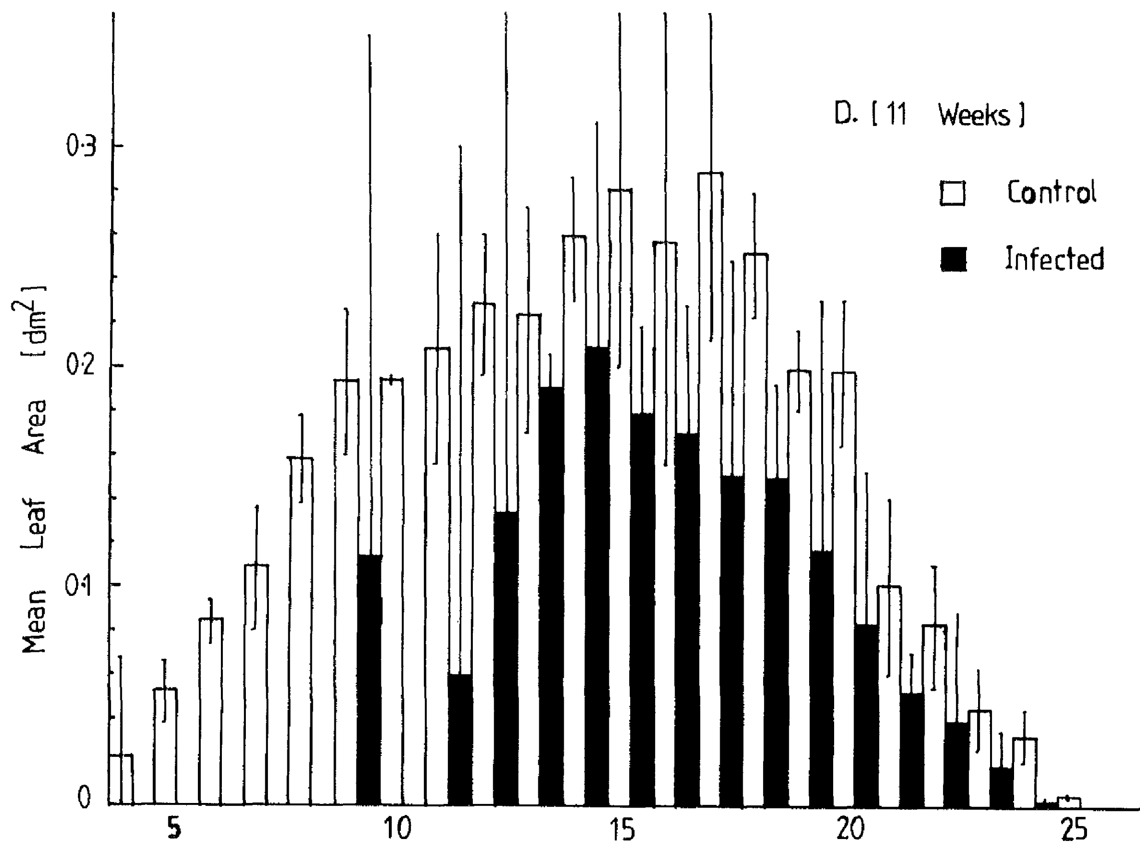
C = Control

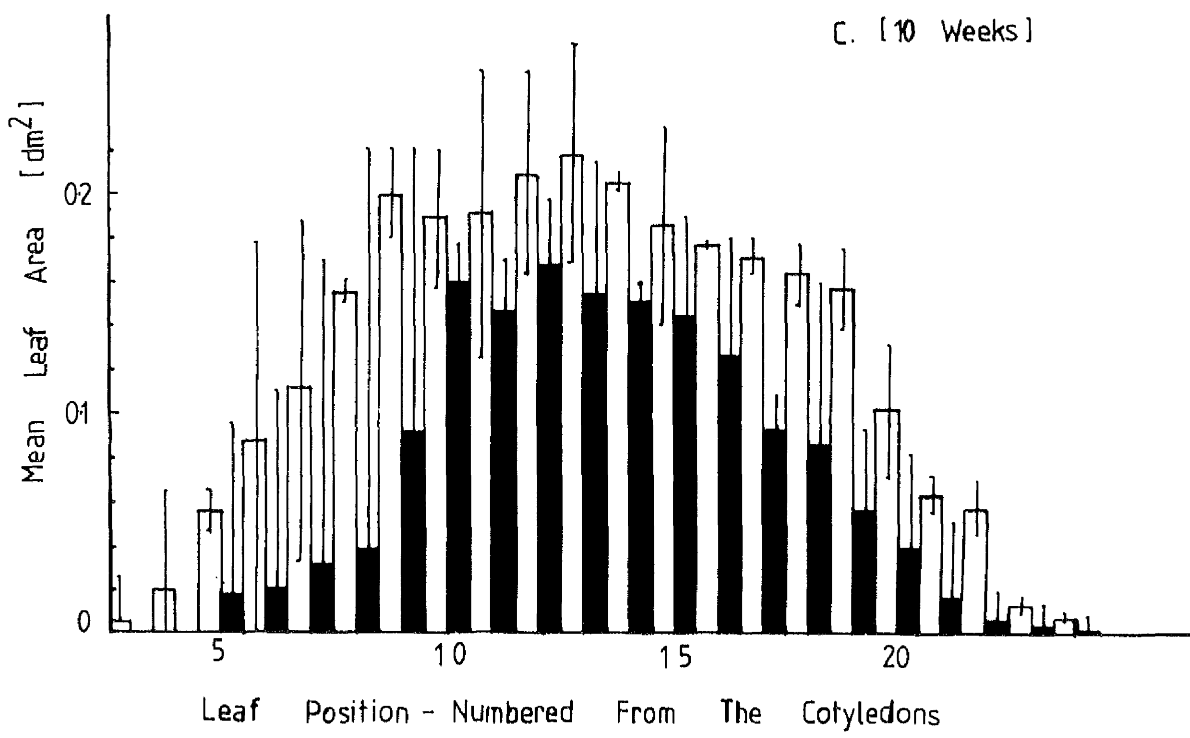
FIGURES 9A - 9E: Effect of infection on the number and areas of leaves present on the main axis of infected and un-infected plants during the growth period between the eighth and twelfth weeks after sow

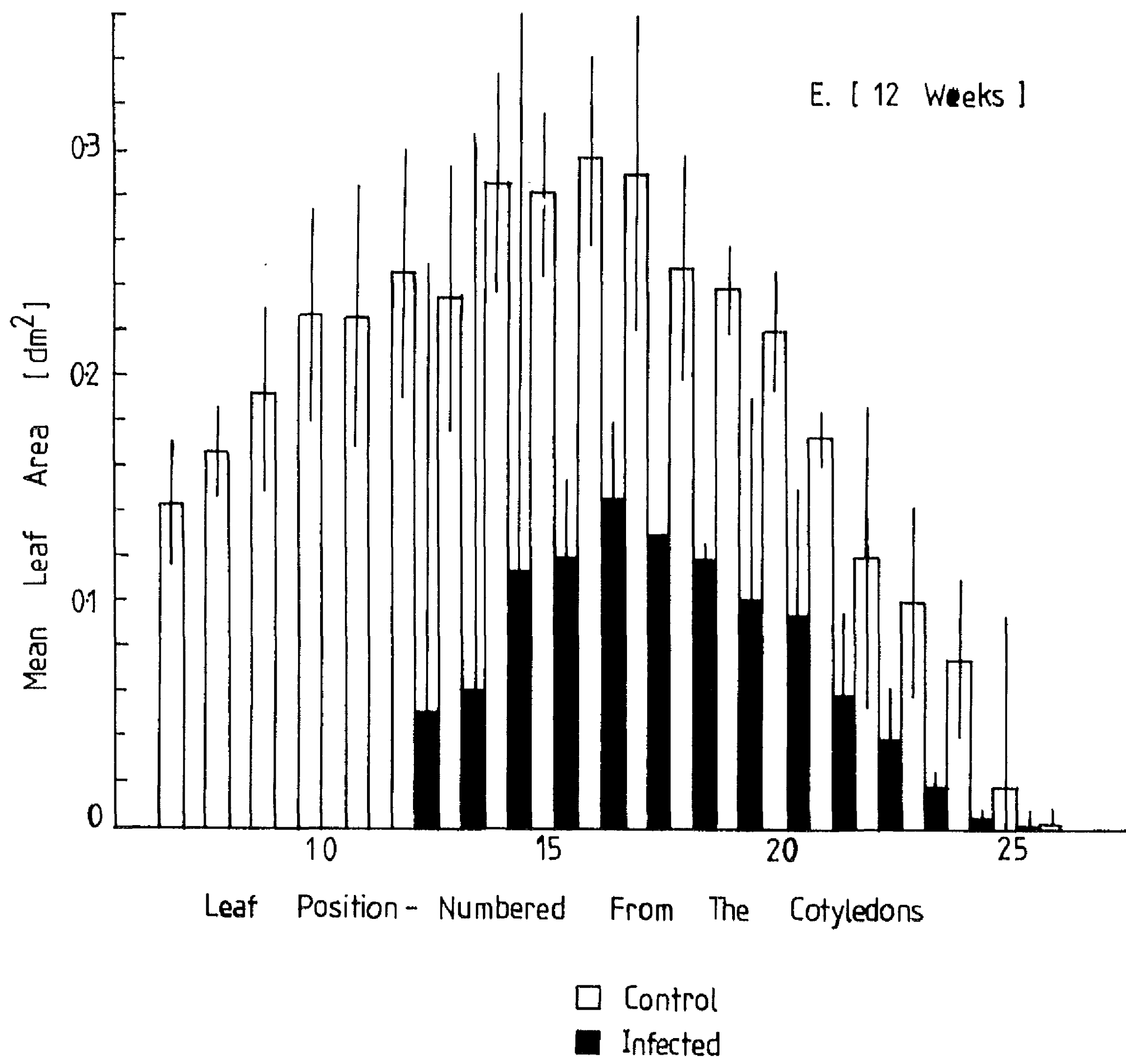
FIG. 9






D. [11 Weeks]
Control
Infected
Mean Leaf Area [dm^2]
0·3
0·2
0·1
0
5
10
15
20
25


C. [10 Weeks]
Mean Leaf Area [dm^2]
0·2
0·1
0
5
10
15
20
Leaf Position - Numbered From The Cotyledons



E. [12 Weeks]
Mean Leaf Area [dm2]
0·3
0·2
0·1
0
10
15
20
25
Leaf Position - Numbered From The Cotyledons
Control
Infected

infection) near the apex of the main axis were greatly reduced in area (Appendix Tables 7C - 7E). Whether this was due to the reduced size of the infected plants or due to translocatable substances secreted by the parasite acting on the leaf tissues in advance of infection was not determined.

(b) Effect of infection on the development of leaves on the lateral branches

The number of leaves produced by individual lateral branches at each harvest are given in Appendix Tables 7AB - 7EB and the results are plotted graphically in Figures 10A - 10E.

In general, the lateral branches on the infected plants produced fewer leaves than those on un-infected plants at every harvest. This may be partly because the infected plants produced fewer lateral branches than did un-infected plants, and partly because the lateral branches developed from buds in the axils of the lower leaves which were already heavily infected and as a result the laterals themselves were infected at a very young age, thus infection may have affected the apical meristem of the branches so that either fewer leaf initials were formed or fewer of the initials expanded to form leaves.

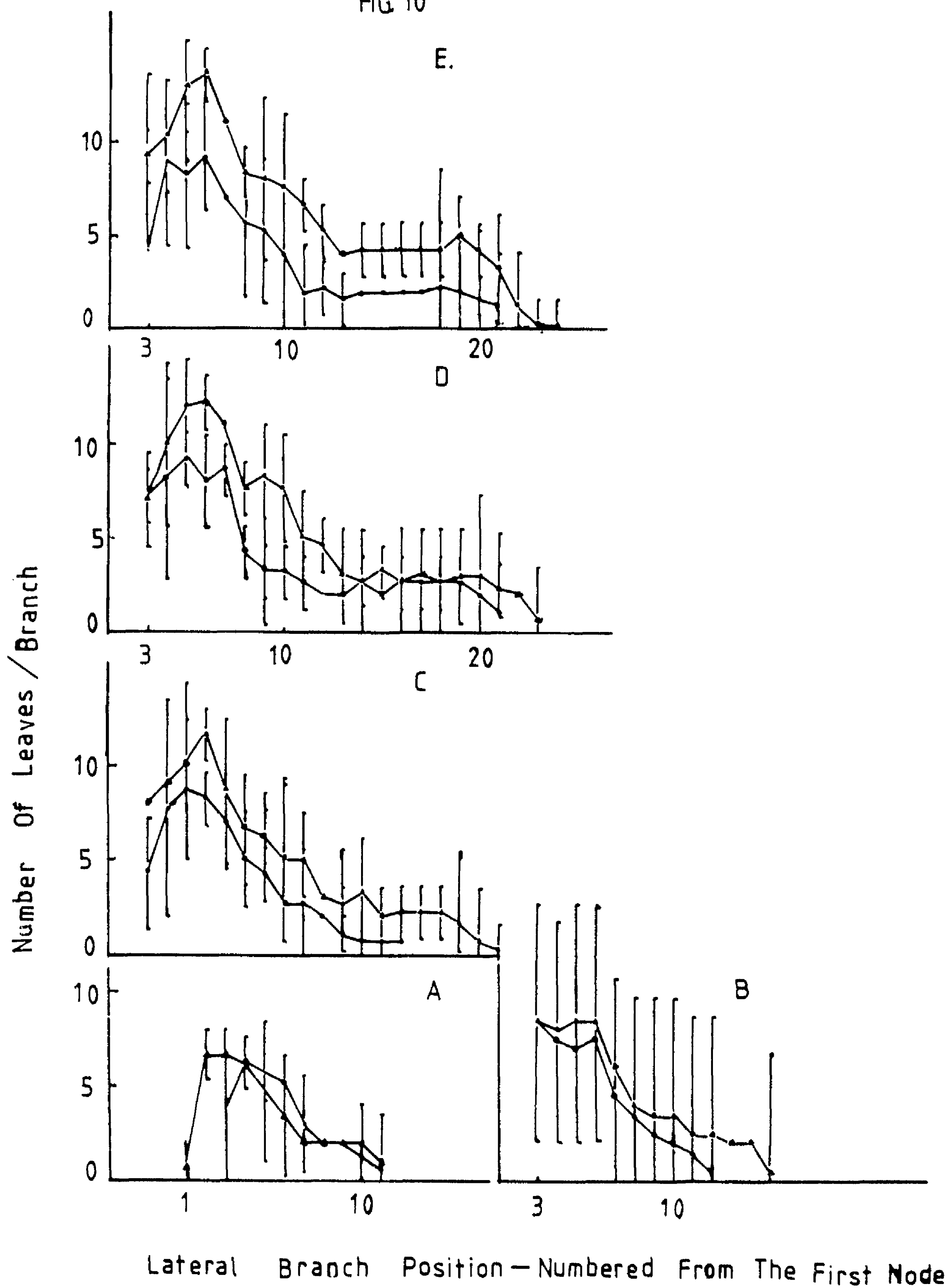
The total areas of leaves produced by each lateral branch of infected and un-infected plants are given in Appendix Tables 7AB - 7EB, and the results plotted as histograms in Figures 11A - 11E.

The reduction in the area of the lateral branch leaves of the infected plants followed the same pattern as that on the main axis, the

FIGURES 10A - 10E: Effect of infection on the development of leaves on individual lateral branches of infected (●———●) and un-infected (▲———▲) plants during the growth period between the eighth and twelfth weeks after sowing.

A	=	eight weeks
B	=	nine weeks
C	=	ten weeks
D	=	eleven weeks
E	=	twelve weeks

FIG 10



FIGURES 11A - 11E: Effect of infection on the expansion of leaves present on the lateral branches of infected and un-infected plants during the growth period between the eighth and twelfth weeks after sowing.

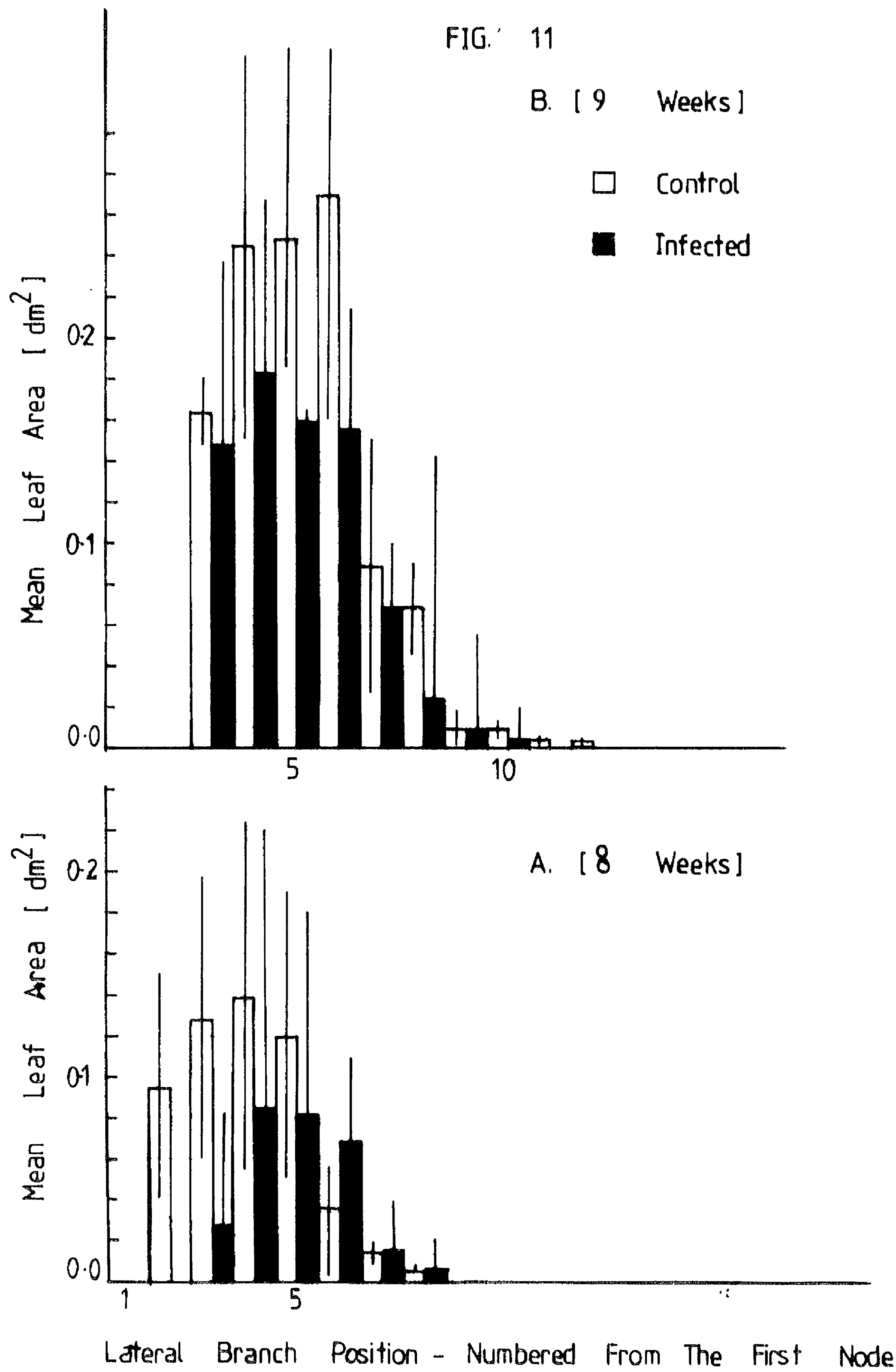
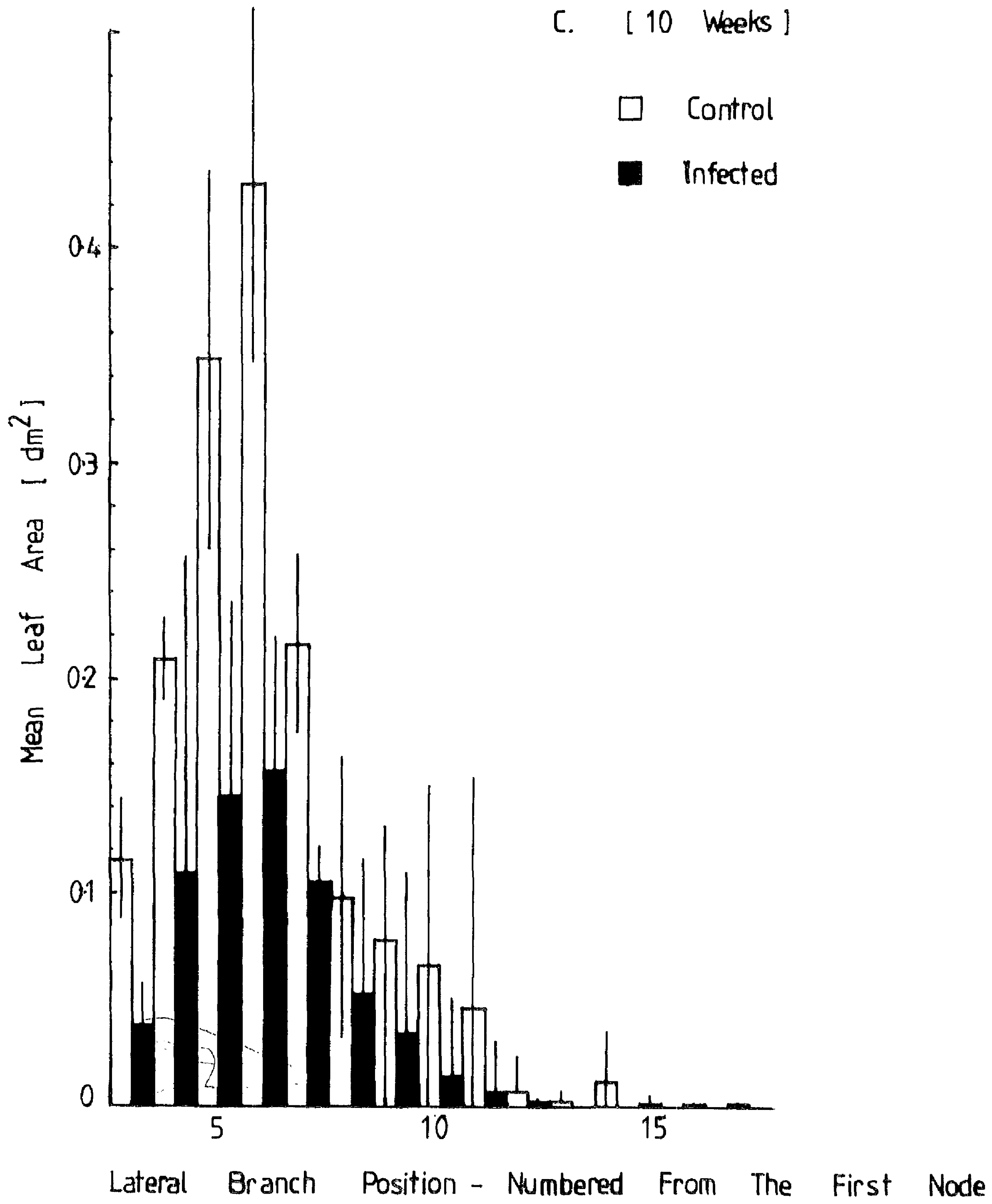
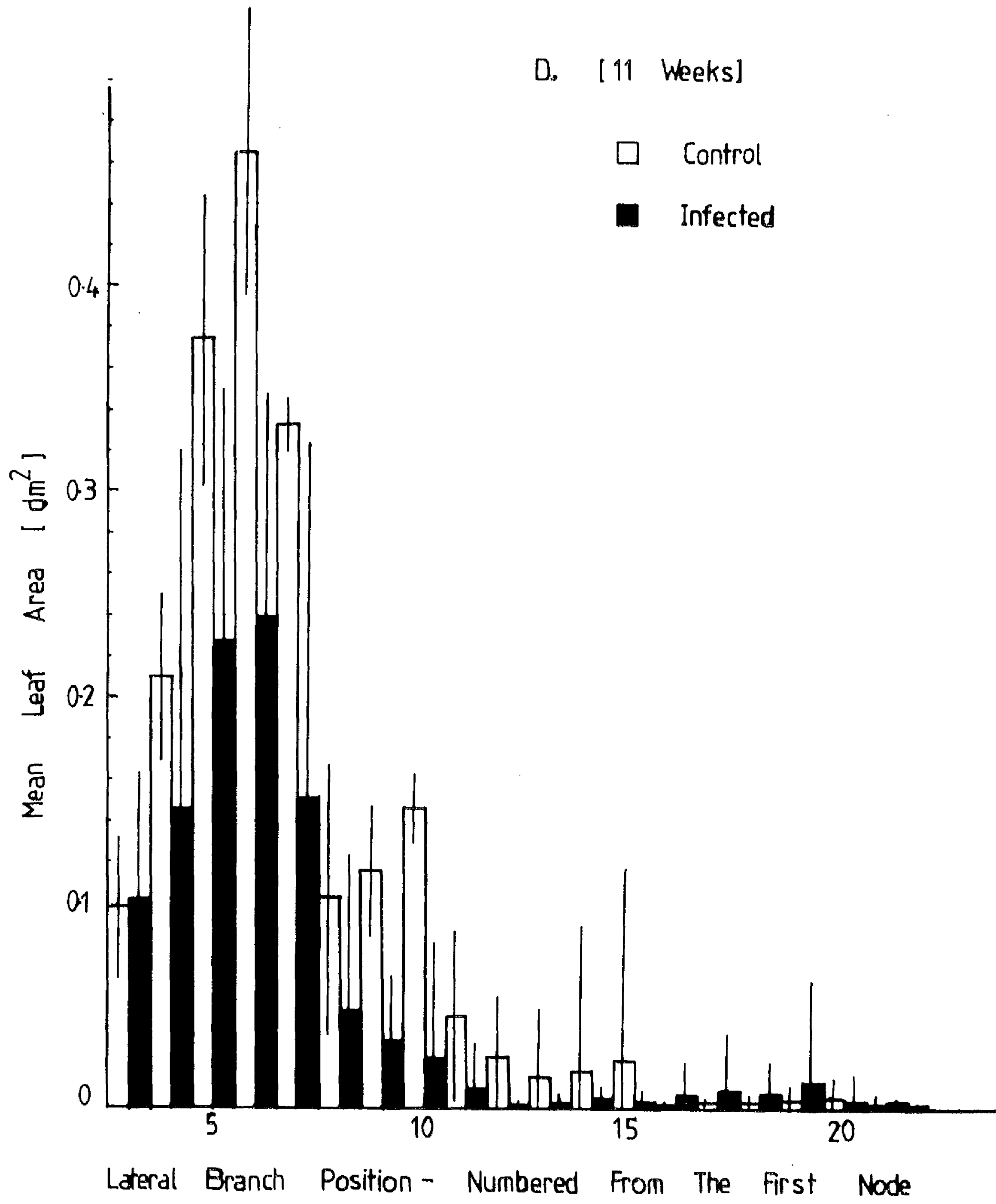
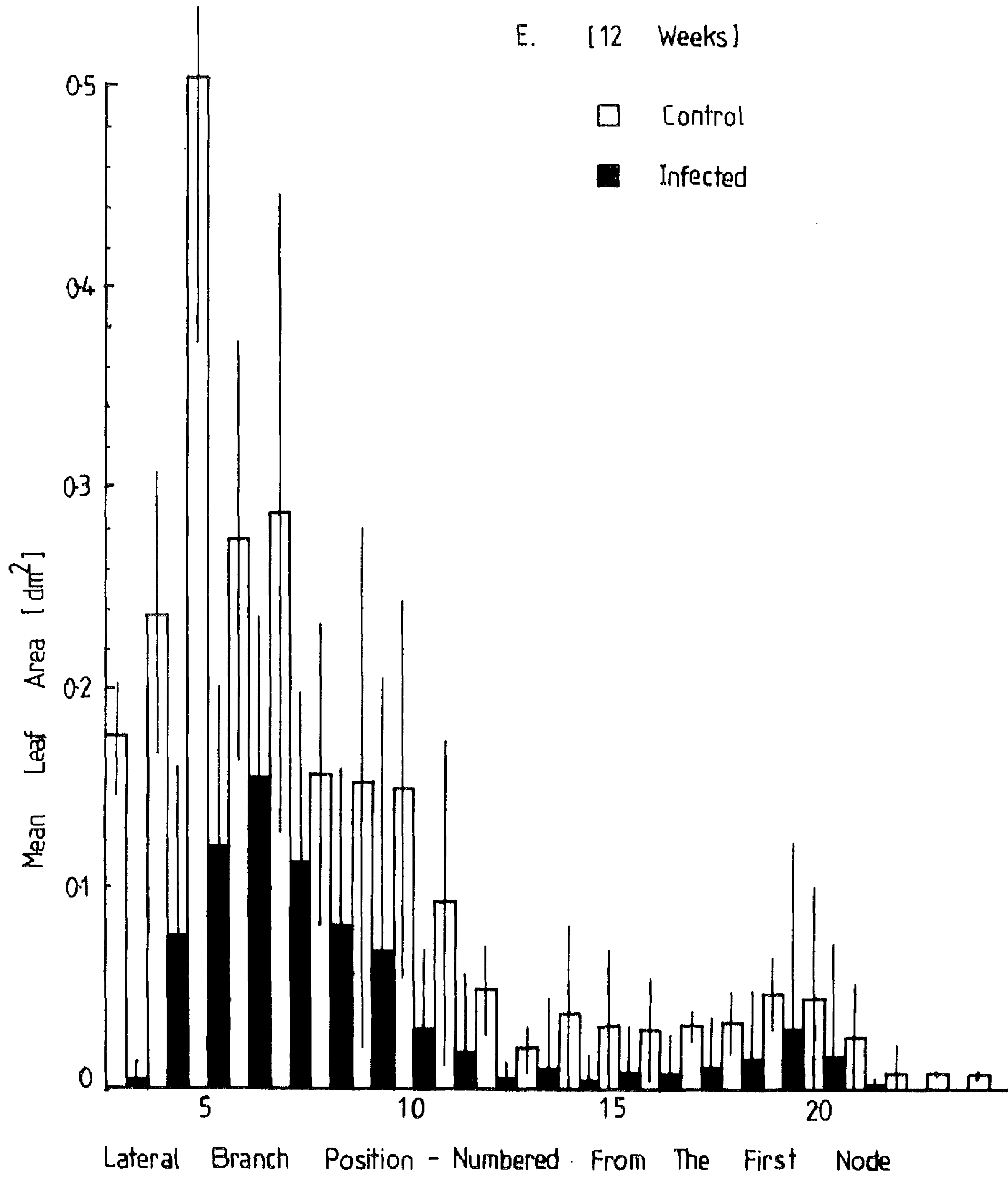


FIG. 11
B. [9 Weeks]
Control
Infected
Mean Leaf Area [dm^2]
0·2
0·1
0·0
5
10
A. [8 Weeks]
Mean Leaf Area [dm^2]
0·2
0·1
0·0
1
5
Lateral Branch Position - Numbered From The First Node


C. [10 Weeks]
Control
Infected
Mean Leaf Area [dm2]
0·4
0·3
0·2
0·1
0
5
10
15
Lateral Branch Position - Numbered From The First Node


D. [11 Weeks]
Control
Infected
Mean Leaf Area [dm^2]
0·4
0·3
0·2
0·1
0
5
10
15
20
Lateral Branch Position - Numbered From The First Node


E. [12 Weeks]
Control
Infected
Mean Leaf Area [dm^2]
0·5
0·4
0·3
0·2
0·1
0
5
10
15
20
Lateral Branch Position - Numbered From The First Node

only difference being that the branch leaves were more reduced in area than were leaves with similar amounts of mildew cover on the main axis.

From these results it can be concluded that reduction in the number of expanded leaves on the infected plants was due not to any reduction on the main axis but to a reduction in the number of leaves expanding on the lateral branches. The extent to which the infected leaf area was reduced was dependent mainly on the time, extent and duration of infection.

Factors affecting the expansion of leaf tissue

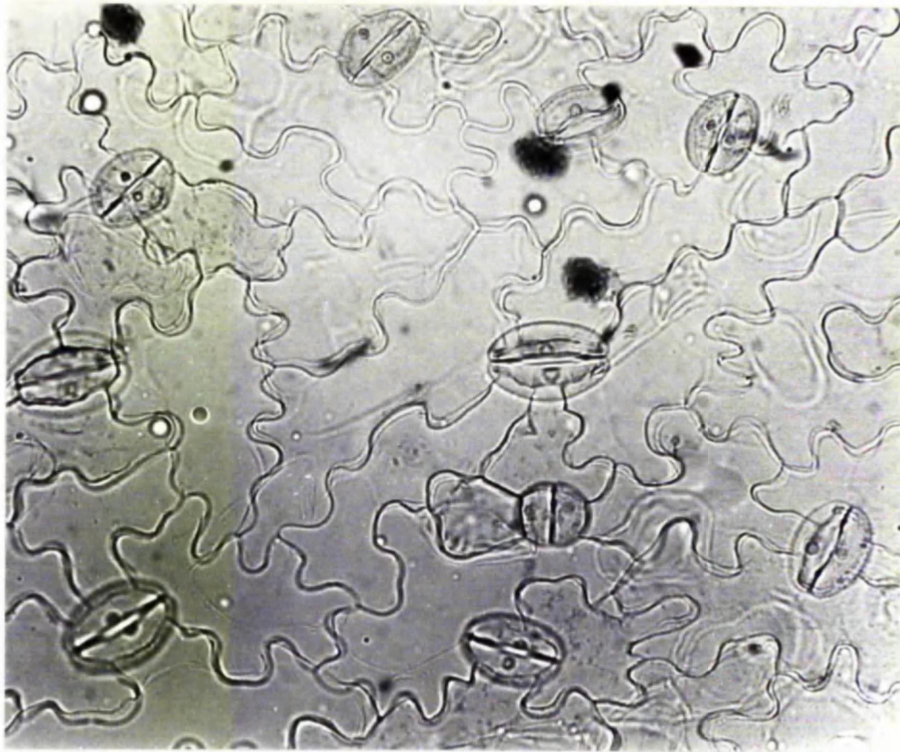
This study was carried out with a view to establishing the events resulting in the reduction in leaf area. Leaf area may be reduced because the cells of the lamina expand less than normal and/or because the leaf lamina contains fewer cells. The effect of infection on cell expansion was thus investigated by comparing the numbers of guard cells per unit area in infected leaves having different levels of infection with those of leaves from corresponding positions on un-infected plants. The results are given in Table 6, and are illustrated in Plate 2.

TABLE 6

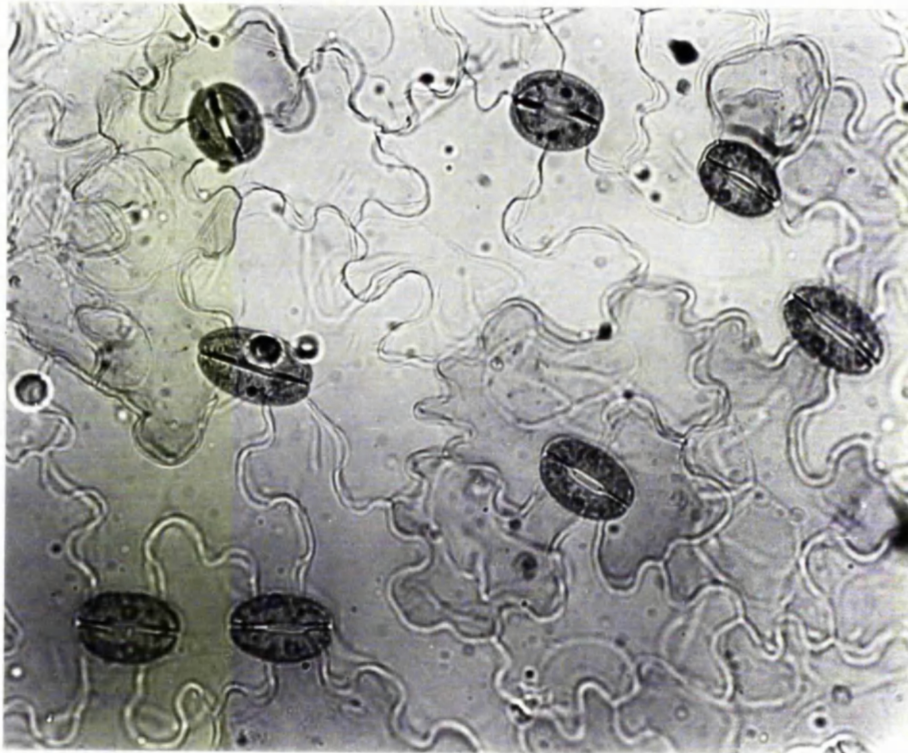
The effect of infection on the number of guard cells per unit area of leaf.

	Infected Leaves					Un-infected Leaves				
Leaf Position	9	11	8	12	13	9	11	8	12	13
Mean leaf area (mm^2)	1314.26	1566.06	1298.12	1586.54	1828.38	1917.19	2282.65	1860.31	2713.65	2970.83
% Infection	40	55	60	70	100	0	0	0	0	0
	No. of guard cells per unit area					No. of guard cells per unit area				
	12	12	9	15	16	9	12	8	12	12
	11	14	13	19	15	10	9	9	8	14
	9	15	12	17	14	10	11	12	10	15
	12	13	16	19	17	9	9	9	11	14
	10	10	14	16	14	10	13	8	13	9
	13	13	13	14	18	12	11	8	12	10
	16	14	13	15	15	8	10	12	10	9
	14	17	14	17	16	10	8	11	11	10
	15	15	11	16	18	9	10	9	10	11
	14	13	10	14	11	12	10	10	12	8
			13							
$\overline{X}$	12.6	13.6	12.5	16.2	15.4	9.9	10.3	9.6	10.9	11.2
SE	1.6	1.4	1.3	1.3	1.4	0.9	1.1	1.1	1.0	1.7

PLATE 2: Photomicrographs of _E. fischeri_ infected (I) and un-infected (C) lower epidermal cells of groundsel leaves.



I



C

An analysis of variance of the number of guard cells per unit area of infected and un-infected leaves was determined and the results are given in Table 7.

TABLE 7

Analysis of variance of the data from Table 6.

Source of variance	Sum of Squares	Degrees of Freedom	Mean Squares	F	P
Infected leaves					
Between columns	111.52	4	27.88	7.97	< 0.001
Between rows	59.22	9	6.58	1.88	NS
Residual	126.08	36	3.50		
TOTAL	296.82	49			
Un-infected leaves					
Between columns	17.88	4	4.47	1.37	NS
Between rows	12.98	9	1.44	0.44	NS
Residual	116.92	36	3.25		
TOTAL	147.78	49			

The results show that there was a significant difference ($P < 0.001$) in the number of guard cells per unit area between leaves with different mildew intensities, but there was no significant difference between the un-infected leaves.

The results in Table 6 show that the number of guard cells per unit area ($0.09083\ mm^2$) of leaf was always significantly higher ($P < 0.02$) in infected leaves than in the corresponding un-infected leaves.

The size of about 25 guard cells from leaves in the 13th position was then determined by measuring their length and breadth. The size of the guard cells in the infected leaf ranged from about 21.6 x 14.4 μ to about 38.4 x 19.2 μ with a mean size of about 29.86 $\pm$ 1.28 x 14.98 $\pm$ 0.52 μ. Those in the corresponding un-infected leaf ranged from about 26.4 x 14.4 μ to about 36.0 x 19.2 μ with a mean size of about 30.34 $\pm$ 0.85 x 15.26 $\pm$ 0.56 μ. There was thus no significant difference in the size of guard cells from infected and un-infected leaves. It is thus likely that the other epidermal cells were significantly smaller in the infected leaves than in the corresponding un-infected leaves. These were however not measured.

The numberof the other epidermal cells per unit area of the leaves from the 13th position was determined by counting the total numbers of cells in 50 different fields of view per leaf. There were an average of about 46.8 cells per 0.09083 mm^2 on the infected leaf and about 33.2 cells per 0.09083 mm^2 on the corresponding un-infected leaf. The infected leaf thus contained significantly more ($P < 0.02$) cells per unit area than did the corresponding un-infected leaf. This shows that the infected leaves were smaller in size because the epidermal cells other than the guard cells were smaller in size than those in the corresponding un-infected leaves.

<u>The effect of infection on the number of the epidermal cells per lower epidermis of the leaf</u>

The total numbers of guard cells per lower epidermis of whole leaves were calculated and the results are given in Table 8.

TABLE 8

Effect of infection on total number of guard cells per lower epidermis of leaves.

Leaf position on main axis	I	C	Reduction (C - I)
	Number of guard cells per lower epidermis	Number of guard cells per lower epidermis	
9	182315.05	208963.78	26,648.73
11	234486.57	258849.44	24,362.87
8	178646.92	196619.79	17,972.87
12	282967.60	325649.95	42,682.35
13	309997.26	366324.95	56,327.69

As can be seen, infected leaves contained fewer guard cells per lower epidermis of leaves than did corresponding un-infected leaves. The other epidermal cells from the lower epidermis of theleaf in the 13th position on the infected plant were also found to be reduced in number by about 143822 cells. It thus appears that infection reduces the numbers of cells per leaf. Thus, the smallness of the leaves on the infected plants appears to be partly due to a reduction in expansion of some of the cells and partly due to inhibition of cell division in the expanding leaf.

Reduction in cell size may be due to the effects of water stress and this was investigated by determining the effects of infection on the water content of the roots, stems and leaves.

Effect of infection on the water content of the roots, stems and leaves

The succulence of the component parts of the plant was calculated and the results are given in Table 9. Succulence expresses the water of a plant as a percentage of the dry weight (Last, 1962). Fresh and dry weight data from which the succulence was calculated are given in Appendix Tables 6A - 6E.

The results show that infection did not affect the water content of the roots or stems since there was no significant difference between succulence of the roots or stems of infected and un-infected plants at any harvest. However, the water content of the infected leaves was always less than that of the un-infected leaves. These differences were however not significant until after the ninth week after sowing when total mildew cover was over 73%.

These results, thus show that the infected leaves contained less water at high levels of infection. This may be due to their obtaining less water from the stem or that they lost more water due to transpiration than they obtained from the stem than did un-infected leaves. The rate of water loss from infected leaves was then investigated.

Loss in fresh weight per unit area of infected and un-infected leaves

Changes in loss in weight per unit area of detached infected and un-infected leaves at two hourly intervals for a period of twelve hours are given in Table 10.

The results show that the total loss in weight per unit area was

TABLE 9

Determination of the effects of infection on the succulence of the roots, stems and leaves.

Time in weeks after sowing		% Infection	Percentage Succulence					
			Roots	SE	Stem	SE	Leaves	SE
6	I	17.2	1302.36	± 116	1069.39	± 65	1403.94	± 48
	C		1290.98	± 155	1027.58	± 32	1402.44	± 87
	t		NS		NS		NS	
7	I	30	1299.57	± 194	909.25	± 80	1164.82	± 85
	C		1390.14	± 97	965.57	± 104	1333.13	± 152
	t		NS				NS	
8	I	62	1086.16	± 237	1091.22	± 36	1118.75	± 195
	C		1089.08	± 150	1082.66	± 69	1203.08	± 55
	t		NS		NS		NS	
9	I	73	1242.28	± 126	1127.73	± 67	1078.75	± 201
	C		1406.03	± 69	1140.41	± 41	1307.08	± 167
	t		NS		NS		NS	
10	I	90	984.11	± 253	947.31	± 53	736.69	± 102
	C		946.11	± 171	981.76	± 67	1027.02	± 57
	t		NS		NS		S	

NS = Not significant at P = 0.05

S = Significant at P = 0.001

TABLE 10

Changes in fresh weight per unit area of leaves taken at two hourly intervals after detachment

% Infection		Changes in fresh weight/unit area (g/dm^2)					
		2 hours	4 hours	6 hours	8 hours	10 hours	12 hours
10	I	*0.4658 ± .13	0.2917 ± .10	0.2490 ± .10	0.1976 ± .12	0.1763 ± .12	0.0905 ± .17
0	C	0.4405 ± .11	0.2478 ± .10	0.2433 ± .07	0.2593 ± .19	0.1969 ± .04	0.1334 ± .15
	t	NS	NS	NS	NS	NS	NS
25	I	0.5329 ± .19	0.2942 ± .05	0.2384 ± .03	0.2153 ± .02	0.1855 ± .01	0.1642 ± .03
0	C	0.4540 ± .06	0.2554 ± .06	0.2277 ± .03	0.2039 ± .10	0.1766 ± .06	0.1315 ± .04
	t	NS	NS	NS	NS	NS	NS
50	I	0.7299 ± .57	0.3392 ± .18	0.2518 ± .09	0.2089 ± .02	0.1630 ± .05	0.1290 ± .04
0	C	0.4926 ± .11	0.2693 ± .10	0.2485 ± .06	0.1960 ± .12	0.1718 ± .02	0.1369 ± .08
	t	NS	NS	NS	NS	NS	NS
75	I	0.6662 ± .25	0.3802 ± .16	0.3006 ± .08	0.2465 ± .01	0.1924 ± .05	0.1704 ± .06
0	C	0.5025 ± .06	0.2849 ± .03	0.2533 ± .04	0.2419 ± .04	0.1957 ± .005	0.1480 ± .05
	t	NS	NS	NS	NS	NS	NS
95	I	0.9491 ± .14	0.5700 ± .15	0.4166 ± .12	0.3260 ± .06	0.1975 ± .01	0.1761 ± .03
0	C	0.6774 ± .13	0.3321 ± .09	0.2639 ± .05	0.1881 ± .04	0.0952 ± .04	0.0373 ± .03
	t	S	S	S	S	S	S

Control leaves are leaves from identical positions as those on infected plants.

* = Each reading is a mean of four leaves
I = Infected leaves
C = Un-infected leaves
NS = Not significant at P = 0.05
S = Significant at P = 0.01

greater than the higher level of infection. Although the infected leaves lost more weight per unit area than the un-infected leaves, none of these differences was significant except for the leaves with over 75% mildew cover.

Thus infection leads to greater rates of water loss from the leaves, and the water stress which results could be responsible for the reduced cell expansion and also for any inhibition in cell division and, therefore, the reduction in leaf size.

DISCUSSION

That infected plants were producing less dry matter than un-infected plants became apparent at about 30% mildew cover and the difference became greater as the experiment continued. It is noteworthy, however, that dry matter production was reduced without the chlorophyll content per unit area of the leaves being affected and before the unit leaf rate was reduced. Thus, the reduction in the rate of dry matter production did not appear to be due to a direct effect on photosynthesis.

The absence of any relation between reduction in dry matter production and chlorophyll content per unit area is in agreement with the work of Magyarosy *et al.*, (1976) who demonstrated that although, *Erysiphe polygoni* reduced dry matter production in the leaves of *Beta vulgaris* the chlorophyll content per unit area of the leaves was not affected.

Last (1962) found that the unit leaf rates in barley when infected with *Erysiphe graminis f. sp. hordei* was reduced when the plant had only about 20% cover. However, in the present investigation, infection did not

reduce the unit leaf rate until more than 90% of the plant was infected. This is indicative of high levels of tolerance in groundsel to Erysiphe fischeri.

Since leaf area and dry matter production were reduced by infection at about the same time, the reduction in dry matter production in the present investigation appears to be more directly related to the reduction in the green leaf area. This reduction in green leaf area is due in part to increased leaf senescence, in part to a reduction in the number of leaves expanding on the lateral branches, and in part, to a reduction in the expansion of individual leaves.

Normally, senescence commences on the older lower leaves and progresses to the upper leaves and mildew infection which occurs first on the lower leaves appears to increase the rate at which the leaves senescenced. Since leaf senescence is characterised by loss of chlorophyll and the chlorophyll content of normal leaves in many plants is a function of the age of the leaf, the presence of the parasite on the lower leaves may have accentuated the natural sequence of events leading to leaf senescence. Thus reducing the number of green functional leaves.

Infection did not affect the total number of leaves expanding on the main axis. A possible reason could be that by the time the apex on the main axis was infected all the leaf primordia were formed and the terminal inflorescence were setting seeds. Infection, however, reduced the numbers of leaves expanding on the lateral branches with levels of infection up to 37% drastically reducing ($P < 0.01$) the total number of expanded leaves. Since lateral branches were produced first from the most heavily infected

lower nodes and since they were infected during their period of active growth, infection may have reduced the rate of cell division at the apices of these branches, so that either fewer leaf primordia were produced or the infected plants produced similar numbers to the control, but fewer of them expanded.

The extent to which infection reduced leaf area was found to be dependent upon the time of infection. Thus, although infection did not affect fully expended leaves, it drastically reduced the area of leaves which became infected while they were still expanding. Such leaves contained fewer and smaller lower epidermal cells than un-infected leaves, indicating that infection probably inhibited both cell division and cell expansion.

Infection was found to reduce the succulence of the leaves. This could either be due to their obtaining less water from the stem or that they lost more water per unit area of leaf than they obtained from the stem than did un-infected leaves. In such leaves, loss of water per unit area of leaves was proportional to mildew intensity. Thus, in addition to any water used directly by the fungus, water was lost at a greater rate than in the un-infected leaves. This enhanced loss of water per unit area of infected leaf may be responsible for the reduced expansion of the ordinary epidermal cells of the leaf lamina.

Ayres (1976) reported that the increased rate of transpiration of leaves of Pisum sativum infected with Erysiphe pisi was due to water loss through the fungus mycelium rather than directly through the cuticle. It may be that this is also the case in this investigation.

That reduction in dry matter production in S. vulgaris infected with E. fischeri was due to a reduction in leaf area has been reported by Ben-Kalio (1976). Also, in barley infected with E. graminis f. sp. hordei, reduction in dry matter production was partly attributed to a reduction in the assimilatory surface and partly to a reduction in the net assimilation rate of the leaves (Last, 1962).

Although E. fischeri reduced dry matter production, the distribution of the dry matter in relation to the development of various organs was not affected even at 90% total mildew cover. The roots, stems and leaves were equally stunted. These results are thus in general agreement with Ben-Kalio's (1976) findings.

In contrast, root development in mildew infected cereal plants has been shown to be drastically reduced, even at low levels of infection of about 30% (Last, 1962). This has been attributed to a distortion of the translocation system resulting in the accumulation of carbohydrates in the infected organs. Recent researches with radioactive materials have produced results in support of this (Edward, 1971).

Minarcic and Pauleck (1975) attributed the reduction in root development of powdery mildew infected barley to the inhibition of mitotic cell division in the apical root meristems. This inhibition could have been due to a shortage of essential carbohydrates or to the presence of a fungal toxin.

The growth indices: specific leaf area, leaf weight ratio, leaf area ratio and unit leaf rate in S. vulgaris exhibited similar ontogenetic changes

in infected and un-infected plants. Although the rates of change were different, the differences were not significant at any harvest. Thus the mildew did not alter the growth pattern of the infected plants. These results are thus in agreement with Ben-Kalio's (1976) findings.

In conclusion, it is clear that the reduction in dry matter production in mildew infected groundsel is primarily dependent upon the extent to which the assimilatory surface is reduced and not to any obvious direct effects on the efficiency of the leaves as photosynthesizing organ. Reduction in leaf area was partly due to a reduction in the number of green leaves and partly due to a reduction in their expansion. Such leaves were found to contain fewer epidermal cells. The reduction in leaf expansion appears to be due to water stress. Although infection reduced dry matter production, the pattern of dry matter distribution and the overall growth patterns were not affected.

Since such high levels of infection as obtained in this investigation rarely occurs in nature, it is unlikely that highly susceptible lines of groundsel will be eliminated due to the activities of the parasite. It thus appears that long association between S. vulgaris and E. fischeri has led to the development of mutual tolerance. A situation in which the host is little affected by infection and in which the parasite grows and reproduces freely. Such a situation has led to the maximum survival of both the host and the parasite, (Part I).

APPENDICES

Appendix Table 1

The interaction between *E. fischeri* and the stems and leaves of inbred lines of *S. vulgaris*

Plant Lines	Date of first infection on leaves	Mean Infection type Stem	Leaves
1a	31.7.78	4	4
b	27.7.78	4	4
c	1.8.78	4	4
d	1.8.78	4	4
e	29.7.78	4	4
f	29.7.78	4	4
g	30.7.78	4	4
h	30.7.78	4	4
i	30.7.78	4	4
j	31.7.78	4	4
k	1.8.78	4	4
l	2.8.78	4	3
m	2.8.78	4	3
n	1.8.78	4	4
o	4.8.78	4	3
p	2.8.78	4	4
q	30.7.78	4	4
r	4.8.78	4	4
s	1.8.78	4	3
2a	29.7.78	4	3
b	30.7.78	4	4
c	1.8.78	4	2
d	30.7.78	4	4
e	29.7.78	4	4
f	2.8.78	4	4
g	3.8.78	4	4
h	1.8.78	4	2
i	17.8.78	2	2
j	18.8.78	2	2
3a	30.7.78	4	4
b	29.7.78	4	4
c	29.7.78	4	4
d	31,7,78	4	4
e	31.7,78	4	4
f	30,7.78	4	4
g	30.7.78	4	4
h	30.7.78	4	4
i	31,7,78	4	4
j	31.7.78	4	4
4a	1,8.78	4	3
b	31,7.78	4	3
c	1.8,78	4	4
d	31.7.78	4	4
e	1,8,78	4	4
f	2.8,78	4	4
g	2.8,78	4	4
h	31.7.78	4	2
i	30,7.78	4	4

Appendix Table 1 cont.

Plant Lines	Date of first Infection on leaves	Mean Infection type stem	Mean Infection type leaves
j	1.8.78	4	4
5a	1.8.78	4	3
b	3.8.78	4	3
c	31.7.78	4	4
d	30.7.78	4	4
e	30.7.78	4	4
f	31.7.78	4	4
g	16.8.78	4	4
h	2.8.78	4	4
i	3.8.78	4	4
j	2.8.78	4	4
6a	1.8.78	4	4
b	-	0	0
c	5.8.78	4	3
d	5.8.78	2	2
e	30.7.78	4	4
f	29.7.78	4	4
g	31.7.78	4	4
7a	1.8.78	4	4
b	-	0	0
c	30.7.78	0	4
d	31.7.78	4	4
e	1.8.78	4	4
f	29.7.78	4	4
g	30.7.78	4	4
h	1.8.78	4	4
i	29.7 78	4	4
j	30.7.78	4	4
8a	30.7.78	4	4
b	30.7.78	4	4
8c	30.7.78	4	4
d	30.7.78	4	4
e	31.7.78	4	4
f	30.7.78	4	4
g	31.7.78	4	4
h	-	0	0
i	-	0	0
j	2.8.78	4	4
k	1.8.78	4	4
9a	16.8.78	0	2
b	2.8.78	0	2
c	6.8.78	2	2
d	3.8.78	2	2
e	11.9.78	2	2
f	7.8.78	2	2
g	13.8.78	0	2
h	1.8.78	2	3
i	3.8.78	0	2
j	2.8.78	0	2
10a	3.8.78	4	4
b	1.8.78	4	4
c	30.7.78	4	4

Appendix Table 1 cont.

Plant Lines	Date of first Infection on Leaves	Mean Infection type Stem	Leaves
d	30.7.78	4	4
e	31.7.78	4	4
f	2.8.78	4	4
g	31.7.78	4	4
h	3.8.78	4	4
i	17.8.78	4	4
j	-	0	0
11a	1.8.78	2	2
b	30.7.78	2	2
c	31.7.78	2	2
d	31.7.78	2	2
e	30.7.78	2	2
f	1.8.78	2	2
g	31.7.78	2	2
h	1.8.78	2	2
i	31.7.78	2	2
j	1.8.78	2	2

Appendix Table 2

The interactions between five isolates of *E. fischeri* and groundsel seedlings giving infection types O, 1, 2, 3 and 4

Plant line	Locality	ISOLATES 1	2	3	4	5
1a	Glasgow	4	O	4	4	3
b		4	4	4	4	4
c		4	O	4	4	4
d		4	4	4	O	4
e		4	O	4	4	4
f		4	4	4	4	3
g		4	4	4	4	4
h		4	4	3	4	1
i		4	4	4	4	3
j		4	4	4	4	2
k		4	3	3	O	2
l		3	4	2	2	3
m		4	O	3	O	2
n		4	O	4	4	3
o		4	O	3	O	4
p		4	4	4	4	4
q		4	3	4	3	2
r		O	4	4	O	4
s		4	O	4	4	4
2a	Crail	4	O	4	4	4
b		4	3	4	3	O
C		O	2	4	O	O
d		4	3	4	2	2
e		4	4	4	2	3
f		O	4	4	O	O
g		3	O	3	3	3
h		4	O	4	2	4
i		O	2	2	O	O
j		O	4	2	O	O
3a	Ayr	4	4	4	3	3
b		4	4	4	3	2
c		4	4	4	4	2
d		3	3	4	3	3
e		4	2	4	4	2
f		3	4	3	O	2
g		2	2	4	3	2
h		4	4	4	2	4
i		1	2	4	4	2
j		4	1	2	2	4
4a	Dublin	4	3	2	4	O
b		1	3	O	3	O
c		2	3	2	O	O
d		3	3	3	2	O
e		2	2	O	O	2

Appendix Table 2 cont.

Plant Line	Locality	ISOLATES 1	2	3	4	5
f	Dublin	0	2	2	4	4
g		3		1	4	0
h		0	0	0	3	4
i		4	4	4	4	0
j		0	2	4	2	4
5a	Far Sawrey	2	3	4	3	2
b		2	3	4	4	4
c		3	2	4	4	4
d		0	2	3	4	2
e		2	0	4	4	4
f		4	3	0	4	4
g		4	3	2	2	0
h		4	4	4	4	2
i		4	4	4	4	2
j		2	0	2	0	2
6a	Ulverston	4	3	4	2	4
b		0	0	0	1	0
c		4	0	0	1	2
d		0	0	4	4	2
e		0	4	4	4	3
f		4	0	2	4	4
g		4	4	2	3	4
7a	Coinston	4	3	4	3	2
b		0	0	0	0	0
c		4	4	4	4	2
d		0	2	2	0	0
e		4	4	4	4	3
f		4	4	0	2	3
g		4	4	4	4	4
h		4	4	2	4	4
i		4	4	4	4	4
j		4	4	4	2	3
8a	Wellesburne	4	4	4	4	4
b		4	4	4	2	2
c		3	4	4	0	4
d		4	4	4	4	4
e		3	3	2	4	3
f		4	3	2	4	4
g		3	3	4	4	2
h		0	0	0	0	0
i		0	3	0	2	4
j		2	4	2	4	2
k		4	3	4	4	4
9a	Perth	0	0	0	0	0
b		0	4	4	2	0
c		0	4	2	0	0
d		0	4	4	0	0
e		0	3	2	2	0
f		0	4	3	0	0
g		0	0	0	0	0
h		1	2	2	4	2
i		0	0	2	0	0
j		0	2	4	0	0

Appendix Table 2 cont.

Plant Lines	Locality	ISOLATES 1	2	3	4	5
10a	Stranraer	0	0	2	4	2
b		3	2	4	2	2
c		4	4	3	4	4
d		4	3	4	2	3
e		2	3	4	4	4
f		3	4	4	3	4
g		4	0	4	4	4
h		2	0	4	3	3
i		3	3	4	1	4
j		0	0	0	0	0
11a	Abington	0	2	0	4	4
b		4	2	4	4	2
c		3	4	3	4	4
d		2	3	4	2	3
e		2	0	3	3	2
f		4	3	4	4	3
g		0	2	2	0	0
h		0	4	2	0	0
i		0	4	4	0	0
j		0	4	4	0	0

Table 3a

The distribution of mildew resistance factors within the total host population.

NUMBER OF PLANTS PER SITE

SITES \ Resistance factors/plant	R_0	R_1	R_2	R_3	R_4	R_5	R_6	R_7	R_8	R_1R_2	R_1R_3	R_1R_4	R_1R_5	R_1R_6	R_2R_4	R_2R_5
GLASGOW	7		2		1	1	2					1				
CRAIL	2		3			1										
AYR	7		1		1											
DUBLIN	2	1		1		2							1			
FAR SAWREY	4		1	2			1								1	
ULVERSTON	1	1						1								
CONISTON	6			1												
WELLESBURN	8	1			1											
PERTH	0										1					
STRANRAER	5				1			1		1						
ABINGTON	4										1					
PEEBLES	5				1											
HAWICK	0		1	1	1		4		1							
DUMFRIES	3													1		
ABERDEEN	5				1											
PITLOCHERY	1						2									
KINGUSSIE	5						1									1
INVERNESS	5				1		1			1						
LAIRG	3					1										
PORTREE	5	1			2					1						
MALLAIG	7			2												
FORT WILLIAM	7														1	1
CRAINLARICH	2			1												
OBAN	3	1	1	1									1			
STIRLING	8															
TOTAL	107	5	9	9	10	5	11	2	1	3	2	1	2	1	2	2

NUMBER OF PLANTS PER SITE

SITES \ Resistance Factors/plant	R_2R_7	R_3R_4	R_3R_5	R_3R_6	R_4R_5	R_4R_6	R_5R_6	R_5R_7	R_7R_8	$R_1R_2R_4$	$R_1R_3R_5$	$R_1R_4R_5$	$R_1R_5R_6$	$R_2R_3R_4$	$R_2R_4R_5$	$R_2R_4R_6$
GLASGOW	1															
CRAIL												1				
AYR									1							
DUBLIN		1			1											
FAR SAWREY							1									
ULVERSTON				1										1		
CONISTON																
WELLESBURN																
PERTH												2	1			
STRANRAER																2
ABINGTON	1											1				
PEEBLES																
HAWICK								1								
DUMFRIES						1	1									1
ABERDEEN																
PITLOCHRY						1										1
KINGUSSIE		1														
INVERNESS														1		
LAIRG																
PORTREE												1				
MALLAIG																
FORT WILLIAM															1	
CRAINLARICH			2							1	1					
OBAN														1		
STIRLING																
TOTAL	2	2	2	1	1	2	2	1	1	1	1	5	1	3	1	4

Resistance Factors/plant	SITES	GLASGOW	CRAIL	AYR	DUBLIN	FAR SAWREY	ULVERSTON	CONISTON	WELLESBURN	PERTH	STRANRAER	ABINGTON	PEEBLES	HAWICK	DUMFRIES	ABERDEEN	PITLOCHRY	KINGUSSIE	INVERNESS	LAIRG	PORTREE	MALLAIG	FORT WILLIAM	CRAINLARICH	OBAN	STIRLING	TOTAL
		N	U	M	B	E	R		O	F		P	L	A	N	T	S		P	E	R		S	I	T	E	
$R_2R_4R_7$		1																									1
$R_2R_5R_7$																	1										1
$R_3R_4R_5$													1			1											2
$R_3R_4R_6$													1					1									2
$R_4R_5R_6$														1													1
$R_4R_5R_7$																1											1
$R_1R_2R_3R_4$																									1		1
$R_1R_2R_4R_5$																										1	1
$R_1R_2R_4R_6$													1														1
$R_1R_2R_5R_6$																	1										1
$R_1R_3R_4R_5$																										1	1
$R_1R_4R_5R_6$			3							3		3															9
$R_2R_3R_4R_6$		1																									1
$R_2R_4R_6R_7$															1												1

NUMBER OF PLANTS PER SITE

SITES \ Resistance Factors/plant	$R_3R_4R_5R_6$	$R_1R_2R_3R_4R_5$	$R_1R_2R_3R_5R_6$	$R_1R_2R_4R_5R_6$	$R_1R_2R_4R_6R_7$	$R_1R_3R_4R_5R_6$	$R_2R_3R_4R_5R_6$	$R_2R_4R_5R_6R_8$	$R_1R_2R_3R_4R_5R_6$	$R_2R_3R_4R_5R_6R_8$	$R_1R_2R_3R_4R_5R_7R_8$	$R_1R_2R_3R_4R_5R_6R_7R_8$
GLASGOW												
CRAIL												
AYR												
DUBLIN											1	
FAR SAWREY												
ULVERSTON			1		1						1	
CONISTON				1								1
WELLESBURN									1			
PERTH				1					1	1		
STRANRAER												1
ABINGTON												
PEEBLES									1			
HAWICK												
DUMFRIES	1											
ABERDEEN	1						1					
PITLOCHRY	1					1		1				
KINGUSSIE		1										
INVERNESS												
LAIRG												
PORTREE												
MALLAIG												
FORT WILLIAM												
CRAINLARCH		2										
OBAN												
STIRLING												
TOTAL	3	3	1	2	1	1	1	1	3	1	2	2

Appendix Table 3b

The distribution of mildew resistance factors within the coastal plant population

Resistance factors/plant	GLASGOW	CRAIL	AYR	DUBLIN	ULVERSTON	STRANRAER	ABERDEEN	INVERNESS	PORTREE	MALLAIG	FORT WILLIAM	OBAN	TOTAL
				NO OF PLANTS PER SITE									
r_0	7	2	8	2	1	5	5	5	5	7	7	3	
R_1				1	1				1			1	4
R_2	2	3	1									1	7
R_3				1						2		1	4
R_4	1		1			1	1	1	2				7
R_5	1	1		2									4
R_6	2							1					3
R_7					1								1
R_1R_2							1	1	1				3
R_1R_4	1												1
R_1R_5				1								1	2
R_2R_4											1		1
R_2R_5											1		1
R_2R_6	2				1			1					4
R_2R_7	1												1
R_3R_4				1									1
R_3R_6					1								1
R_4R_5				1									1
$R_1R_4R_5$		1							1				2
$R_2R_3R_4$					1			1				1	3
$R_2R_4R_5$											1		1
$R_2R_4R_6$						2							2
$R_2R_4R_7$	1												1

Appendix Table 3b cont.

Resistance factors/plant	GLASGOW	CRAIL	AYR	DUBLIN	ULVERSTON	STRANRAER	ABERDEEN	INVERNESS	PORTREE	MAILAIG	FORT WILLIAM	OBAN	TOTAL
				NO OF PLANTS PER SITE									
$R_3R_4R_5$							1						1
$R_4R_5R_7$							1						1
$R_1R_2R_3R_4$												1	1
$R_1R_4R_5R_6$		3											3
$R_2R_3R_4R_5$	1												1
$R_3R_4R_5R_6$							1						1
$R_1R_2R_3R_5R_6$					1								1
$R_1R_2R_4R_6R_7$					1								1
$R_2R_3R_4R_5R_6$							1						1
$R_1R_2R_3R_4R_5R_7R_8$				1	1								2
$R_1R_2R_3R_4R_5R_6R_7R_8$						1							1

Appendix Table 3 c

The distribution of mildew resistance factors within the Inland Plant Population

Resistance Factors/plant	SITES	FAR SAWREY	CONISTON	WELLESBOURNE	PERTH	ABINGTON	PEEBLES	HAWICK	DUMFRIES	PITLOCHRY	KINGUSSIE	LAIRG	CRAINLARICH	STIRLING	TOTAL
					NO OF PLANTS PER SITE										
r_0		4	6	8	0	4	5	0	3	1	5	3	2	8	
R_1				1											1
R_2		1						1							2
R_3		2	1					1					1		5
R_4				1			1	1							3
R_5												1			1
R_6		1						4		2	1				8
R_7			1												1
R_8								1							1
R_1R_3					1	1									2
R_1R_6									1						1
R_2R_4		1													1
R_2R_5											1				1
R_2R_6									1						1
R_2R_7						1									1
R_3R_4											1				1
R_3R_5													2		2
R_4R_6									1	1					2
R_5R_6		1							1						2
R_6R_7								1							1
$R_1R_2R_4$													1		1
$R_1R_3R_5$													1		1

Appendix Table 3c cont.

Resistance factors/plant — SITES	FAR SAWREY	CONISTON	WELLESBOURNE	PERTH	ABINGTON	PEEBLES	HAWICK	DUMFRIES	PITLOCHRY	KINGUSSIE	LAIRG	CRAINLARICH	STIRLING	TOTAL
NO OF PLANTS PER SITE														
$R_1R_4R_5$				2	1									3
$R_1R_5R_6$				1										1
$R_2R_4R_6$								1	1					2
$R_2R_5R_7$									1					1
$R_3R_4R_5$						1								1
$R_3R_4R_6$						1				1				2
$R_4R_5R_6$							1							1
$R_1R_2R_4R_5$													1	1
$R_1R_2R_4R_6$						1								1
$R_1R_2R_5R_6$									1					1
$R_1R_3R_4R_5$													1	1
$R_1R_4R_5R_6$				3		3								6
$R_2R_4R_6R_7$								1						1
$R_3R_4R_5R_6$								1	1					2
$R_1R_2R_3R_4R_5$										1		2		3
$R_1R_2R_4R_5R_6$		1		1										2
$R_1R_3R_4R_5R_6$									1					1
$R_2R_4R_5R_6R_8$									1					1
$R_1R_2R_3R_4R_5R_6$				1		1								2
$R_2R_3R_4R_5R_6R_8$				1										1
$R_1R_2R_3R_4R_5R_6R_7R_8$		1	1											2

Appendix Table 3d

The distribution of mildew resistance factors whithin plant lines collected from the North of Stirling.

Resistance factors per plant \ SITES	CRAIL	PERTH	ABERDEEN	PITLOCHRY	KINGUSSIE	INVERNESS	LAIRG	PORTREE	MALLAIG	FT. WILLIAM	CRAINLARICH	OBAN	TOTAL
	NO OF PLANTS PER SITE												
r_0	2	0	5	1	5	5	3	5	7	7	2	3	45
R_1								1				1	2
R_2	3											1	4
R_3									2		1	1	4
R_4			1			1		2					4
R_5	1						1						2
R_6				2	1	1							4
R_1R_2						1		1					2
R_1R_3		1											1
R_1R_5												1	1
R_2R_4										1			1
R_2R_5					1					1			2
R_2R_6						1							1
R_3R_4					1								1
R_3R_5											2		2
R_4R_5				1									1
$R_1R_2R_4$											1		1
$R_1R_3R_5$											1		1
$R_1R_4R_5$	1	2						1					4
$R_1R_5R_6$		1											1
$R_2R_3R_4$						1					1		2
$R_2R_4R_5$										1			1
$R_2R_4R_6$				1									1
$R_2R_5R_7$				1									1

Appendix Table 3d cont.

Resistance factors per plant	CRAIL	PERTH	ABERDEEN	PITLOCHRY	KINGUSSIE	INVERNESS	LAIRG	PORTREE	MALLAIG	FORT WILLIAM	CRAINLARICH	OBAN	TOTAL
					no of plants per site								
$R_3R_4R_5$					1								1
$R_3R_4R_5$			1										1
$R_4R_5R_7$			1										1
$R_1R_2R_3R_4$												1	1
$R_1R_2R_5R_6$				1									1
$R_1R_4R_5R_6$	3	3											6
$R_3R_4R_5R_6$			1	1									2
$R_1R_2R_3R_4R_5$					1						2		3
$R_1R_2R_4R_5R_6$		1											1
$R_1R_3R_4R_5R_6$				1									1
$R_2R_3R_4R_5R_6$			1										1
$R_2R_4R_5R_6R_8$				1									1
$R_1R_2R_3R_4R_5R_6$		1											1
$R_2R_3R_4R_5R_6R_8$		1											1

Appendix Table 3e

The distribution of mildew resistance factors within plant lines collected from the South of Stirling.

Resistance factors per plant	STIRLING	GLASGOW	AYR	FAR SAWREY	ULVERSTON	CONISTON	STRANRAER	ABINGTON	PEEBLES	HAWICK	DUMFRIES	TOTAL
	NO. OF PLANTS PER SITE											
r_0	8	7	8	4	1	6	5	4	5	0	3	51
R_1					1							1
R_2		2	1	1						1		5
R_3				2		1				1		4
R_4		1	1				1		1	1		5
R_5		1										1
R_6		2		1						4		7
R_7					1	1						2
R_8										1		1
R_1R_2							1					1
R_1R_3								1				1
R_1R_4		1										1
R_1R_6											1	1
R_2R_4				1								1
R_2R_6		2			1						1	4
R_2R_7		1						1				2
R_3R_6					1							1
R_4R_6											1	1

Appendix Table 3e cont.

Resistance factors/plant — SITES	STIRLING	GLASGOW	AYR	FAR SAWREY	ULVERSTON	CONISTON	STRANRAER	ABINGTON	PEEBLES	HAWICK	DUMFRIES	TOTAL
	NO. OF PLANTS PER SITE											
R_5R_6				1							1	2
R_6R_7										1		1
$R_1R_4R_5$								1				1
$R_2R_3R_4$					1							1
$R_2R_4R_6$							2				1	3
$R_2R_4R_7$		1										1
$R_3R_4R_5$										1		1
$R_3R_4R_6$										1		1
$R_4R_5R_6$											1	1
$R_1R_2R_4R_5$	1											1
$R_1R_2R_4R_6$										1		1
$R_1R_3R_4R_5$		1										1
$R_1R_4R_5R_6$									3			3
$R_2R_3R_4R_6$			1									1
$R_2R_4R_5R_7$											1	1
$R_3R_4R_5R_6$											1	1
$R_1R_2R_3R_5R_6$						1						1
$R_1R_2R_4R_5R_6$							1					1
$R_1R_2R_4R_6R_7$						1						1
$R_1R_2R_3R_4R_5R_6$								1		1		2

Appendix Table 3e cont.

Resistance factors/plant (SITES)	STIRLING	GLASGOW	AYR	FAR SAWREY	ULVERSTON	CONISTON	STRANRAER	ABINGTON	PEEBLES	HAWICK	DUMFRIES	TOTAL
	NO. OF PLANTS PER SITE											
$R_1R_2R_3R_4R_5R_7R_8$						1						1
$R_1R_2R_3R_4R_5R_6R_7R_8$							1					1

The association between host morphological characters and

mildew resistance factors.

No. of Plant lines	Host morphological characters			ISOLATES				
	Stem colour	Hairy/glabrous Stem	Capitulum	1	2	3	4	5
80	Red	glabrous	non-radiate	r_1	r_2	r_3	r_4	r_5
4	Red	glabrous	non-radiate	R_1	r_2	r_3	r_4	r_5
8	Red	glabrous	non-radiate	r_1	R_2	r_3	r_4	r_5
5	Red	glabrous	non-radiate	r_1	r_2	R_3	r_4	r_5
10	Red	glabrous	non-radiate	r_1	r_2	r_3	R_4	r_5
6	Red	glabrous	non-radiate	r_1	r_2	r_3	r_4	R_5
2	Red	glabrous	non-radiate	R_1	R_2	r_3	r_4	r_5
2	Red	glabrous	non-radiate	R_1	r_2	R_3	r_4	r_5
2	Red	glabrous	non-radiate	R_1	r_2	r_3	r_4	R_5
6	Red	glabrous	non-radiate	r_1	R_2	r_3	R_4	r_5
2	Red	glabrous	non-radiate	r_1	R_2	r_3	r_4	R_5
3	Red	glabrous	non-radiate	r_1	r_2	R_3	R_4	r_5

No. of Plant lines	Host morphological characters			ISOLATES				
	Stem colour	Hairy/glabrous stem	Capitulum	1	2	3	4	5
1	Red	glabrous	non-radiate	r_1	r_2	r_3	R_4	R_5
2	Red	glabrous	non-radiate	R_1	R_2	r_3	R_4	r_5
1	Red	glabrous	non-radiate	R_1	R_2	r_3	r_4	R_5
1	Red	glabrous	non-radiate	R_1	r_2	R_3	r_4	R_5
10	Red	glabrous	non-radiate	R_1	r_2	r_3	R_4	R_5
2	Red	glabrous	non-radiate	r_1	R_2	R_3	R_4	r_5
1	Red	glabrous	non-radiate	r_1	R_2	r_3	R_4	R_5
4	Red	glabrous	non-radiate	r_1	r_2	R_3	R_4	R_5
1	Red	glabrous	non-radiate	R_1	R_2	R_3	r_4	R_5
2	Red	glabrous	non-radiate	R_1	R_2	r_3	R_4	R_5
2	Red	glabrous	non-radiate	r_1	R_2	R_3	R_4	R_5
4	Red	glabrous	non-radiate	R_1	R_2	R_3	R_4	R_5
18	Green	glabrous	non-radiate	r_1	r_2	r_3	r_4	r_5

Appendix Table 4 cont.

No. of Plant lines	Host morphological characters: Stem colour	Hairy/glabrous stem	Capitulum	ISOLATES: 1	2	3	4	5
1	Green	glabrous	non-radiate	R_1	r_2	r_3	r_4	r_5
4	Green	glabrous	non-radiate	r_1	R_2	r_3	r_4	r_5
1	Green	glabrous	non-radiate	r_1	r_2	R_3	r_4	r_5
2	Green	glabrous	non-radiate	r_1	r_2	r_3	R_4	r_5
1	Green	glabrous	non-radiate	R_1	R_2	r_3	r_4	r_5
1	Green	glabrous	non-radiate	R_1	r_2	r_3	R_4	r_5
1	Green	glabrous	non-radiate	R_1	r_2	r_3	r_4	R_5
1	Green	glabrous	non-radiate	r_1	R_2	r_3	r_4	R_5
1	Green	glabrous	non-radiate	r_1	r_2	R_3	R_4	r_5
1	Green	glabrous	non-radiate	r_1	r_2	r_3	R_4	R_5
4	Green	glabrous	non-radiate	R_1	r_2	r_3	R_4	R_5
1	Green	glabrous	non-radiate	r_1	R_2	r_3	R_4	R_5

No. of Plant lines	Host morphological characters			ISOLATES				
	Stem colour	Hairy/Glabrous stem	Capitulum	1	2	3	4	5
1	Green	glabrous	non-radiate	r_1	r_2	R_3	R_4	R_5
3	Green	glabrous	non-radiate	R_1	R_2	R_3	R_4	R_5
10	Red	hairy	non-radiate	r_1	r_2	r_3	r_4	r_5
1	Red	hairy	non-radiate	r_1	R_2	r_3	r_4	r_5
1	Red	hairy	non-radiate	r_1	r_2	R_3	r_4	r_5
1	Red	hairy	non-radiate	r_1	r_2	r_3	R_4	R_5
1	Red	hairy	non-radiate	R_1	r_2	R_3	R_4	R_5
3	Red	hairy	non-radiate	R_1	R_2	R_3	R_4	R_5
9	Green	hairy	non-radiate	r_1	r_2	r_3	r_4	r_5
3	Green	hairy	non-radiate	r_1	r_2	r_3	r_4	r_5
1	Green	hairy	non-radiate	R_1	r_2	R_3	R_4	R_5
2	Red	hairy	radiate	r_1	r_2	r_3	r_4	r_5
2	Red	hairy	radiate	r_1	R_2	r_3	r_4	r_5
1	Red	hairy	radiate	r_1	R_2	r_3	R_4	r_5

No of Plant lines	Host morphological characters			ISOLATES				
	Stem Colour	Hairy/glabrous stem	Capitulum	1	2	3	4	5
1	Red	hairy	radiate	R_1	R_2	r_3	R_4	r_5
2	Red	hairy	radiate	r_1	R_2	R_3	R_4	r_5
1	Red	hairy	radiate	R_1	R_2	R_3	R_4	r_5
1	Red	hairy	radiate	R_1	R_2	r_3	R_4	R_5
2	Red	glabrous	radiate	r_1	r_2	r_3	r_4	r_5
1	Red	glabrous	radiate	R_1	r_2	r_3	r_4	r_5
1	Red	glabrous	radiate	r_1	R_2	r_3	r_4	r_5
1	Red	glabrous	radiate	r_1	r_2	r_3	r_4	R_5
1	Red	glabrous	radiate	r_1	R_2	r_3	R_4	r_5

Where R denotes resistance
r dentoes susceptability

Appendix Table 5a

The Inheritance of resistance factors in the f_1 generations of various crosses

		Morphological Characters	X	Y	3	4	5
			ISOLATES				
♂ 1f	GGhhrr	(bright yellow disc florets)	4	4	4	4	4
♀ 1d	GGHhRR	(creamy yellow ray floret corolla)	3	4	4	4	4
F_1	GGhhRr	(bright yellow ray floret corolla)	4	4	4	4	4
3_j	gghhrr		4	4	4	4	4
1f	GGhhrr		4	4	4	4	4
f_1	Gghhrr		4	4	4	4	4
1f	GGhhrr		4	4	4	4	4
3_j	gghhrr		4	4	4	4	4
F_1	Gghhrr		4	4	4	4	4
1s	gghhrr		4	0	4	4	4
2h	GGHHRR		4	4	4	4	4
F_1	GgHhRr		4	0	4	4	4
1d	GGHhRR	(creamy yellow ray floret corolla)	3	4	3	3	4
4h	GGHHrr	(bright yellow disc florets)	2	0	0	0	0
F_1	GGHHRr	(bright yellow ray floret corolla)	2	0	0	0	0
2h	GGHHRR		4	4	4	4	4
4h	GGHHrr		0	0	0	0	0
F_1	GGHHRr		0	0	0	0	0
4a	gghhrr		4	4	4	2	1
6d	GGHHRR		1	0	4	0	2-3
F_1	GgHhRr		1	0	4	0	0

Morphological Characters			X	Y	3	4	5
				ISOLATES			
♂ 8h	GGHHrr		0	0	0	0	0
♀ 1L	GGHHRR		2	0	4	2-3	4
F_1	GGHHRr		0	0	0	0	0
1L	GGHHRR		2	0	4	2-3	4
8h	GGHHrr		0	0	0	0	0
F_1	GGHHRr		0	0	0	0	0
8a	GGhhrr		4	4	4	4	4
1m	GGhhRR		3	0	3	0	3
F_1	GGhhRr		4	0	4	0	4
8f	GGhhrr		3	3	3	4	3
1o	GGHHRR		4	0	3	0	4
F_1	GGHhRr		4	0	2	0	4
10_j	GGhhrr	(bright yellow disc florets)	0	0	0	0	0
1d	GGHhRR	(creamy yellow ray floret corolla)	3	4	4	3	4
F_1	GGhhRr	(bright yellow ray floret corolla)	0	0	4	0	0
1L	GGHHRR		2	0	4	2-3	4
10_j	GGhhrr		0	0	0	0	0
F_1	GGHhRr		0	0	4	0	0
11_j	gghhrr		0	4	4	0	0
S. squalidus	GGhhRR		0	0	0	0	0
f_1	GghhRr		0	0	0	0	0
14c	GGHHRR		4	0	4	0	4
3g	ggHhrr		4	4	4	4	3
F_1	GgHHRr		4	0	4	0	4

Morphological Characters		X	Y	ISOLATES 3	4	5
♂ 17b	GGhhrr	0	0	0	0	0
♀ 1o	GGHHRR	4	0	3	0	4
F_1	GGHhRr	0	0	0	0	0
13i	GGhhrr	2	0	4	4	4
25_j	GGHHRR	0	0	3	0	0
F_1	GGHhRr	0	0	4	0	0

Where

GG = homozygous for red colour (dark red)

Gg = heterozygous for red colour (pale red)

gg = homozygous for green colour

HH = homozygous for hairiness

Hh = heterozygous for hairiness

hh = homozygous for glabrous

RR = homozygous radiate inflorescence character

Rr = heterozygous radiate inflorescence character

rr = homozygous non-radiate inflorescence character

♂ = male parent

♀ = female parent

F_1 = hybrid

o-4[1] = infection types

Appendix Table 5b

Segregation of resistance factors in the

F_2 generations

(Cross 5b : 1d x 4h)

Host Morphological Characters					
	Ray floret corolla colour	ISOLATES Y	3	4	5
GGHHrr		0	0	0	0
GGHHrr		4	4	4	4
GGHHRR	Bright yellow	2	3	4	4
GGHHRR	Creamy yellow	4	4	4	4
GGHHRr	Creamy yellow	0	0	0	0
GGHHRR	Bright yellow	4	4	4	4
GGHHRr	Bright yellow	2	0	2	0
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	2	0
GGHHRR	Creamy yellow	4	4	4	4
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	3	3	4	4
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRR	Creamy yellow	0	0	0	0
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr		0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0

Host Morphological Characters					
	Ray floret corolla colour	ISOLATES Y	3	4	5
GGHHRR	Creamy yellow	0	0	2	0
GGHHrr		0	0	0	0
GGHHRr	Creamy yellow	2	4	4	4
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	4	3	2	2
GGHHRr	Bright yellow	2	2	0	2
GGHHrr		0	2	1	2
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	2	0	2	0
GGHHrr		0	0	1	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHRR	Creamy yellow	0	2	0	2
GGHHRr	Creamy yellow	4	4	4	4
GGHHrr		4	4	0	4
GGHHRr	Creamy yellow	0	2	1	2

Host Morphological Characters		ISOLATES			
	Ray floret corolla colour	Y	3	4	5
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	2	0	2
GGHHrr		0	0	0	0
GGHHRr	Bright yellow	0	0	1	0
GGHHRr	Bright yellow	4	4	4	4
GGHHRr	Creamy yellow	0	0	0	0
GGHHrr		0	0	0	1
GGHHRr	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHrr		0	2	2	2
GGHHRr	Bright yellow	0	0	0	0
GGHHrr		2	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHRR	Creamy yellow	4	4	4	4
GGHHRr	Creamy yellow	3	4	0	4
GGHHRR	Bright yellow	0	0	0	0
GGHHRR	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0
GGHH		0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHrr		0	0	0	0
GGHH		0	0	0	0
GGHHRr	Bright yellow	0	0	2	0
GGHHrr		0	0	0	0

Host Morphological Characters		ISOLATES			
	Ray floret corolla colour	Y	3	4	5
GGHHRR	Bright yellow	0	0	0	0
GGHHRr	Creamy yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	0	0	0
GGHHRr	Bright yellow	0	2	0	2
GGHHRr	Creamy yellow		0	0	0
GGHHrr			0	2	2
GGHHrr			0	0	0
GGHHRr	Bright yellow		0	2	0
GGHHrr			0	0	0
GGHHRr	Bright yellow		2	2	3
GGHHrr			0	0	1
GGHHRR	Creamy yellow		0	0	0
GGHHRr	Bright yellow		2	2	2
GGHHRR	Bright yellow		0	0	0
GGHHrr			2	1	3
GGHHRr	Creamy yellow		0	0	0
GGHHRr	Bright yellow		0	0	0
GGHHRr	Bright yellow		0	0	0
GGHHRr	Bright yellow		0	0	0
GGHHRR	Bright yellow		0	2	1
GGHHRR	Bright yellow		1	2	2
GGHHRr	Bright yellow		0	0	0
GGHHRR	Bright yellow		0	0	0

Host Morphological Characters		ISOLATES			
	Ray floret corolla colour	Y	3	4	5
GGHHRr	Bright yellow		0	2	1
GGHHRr	Bright yellow		0	0	0
GGHHRr	Bright yellow		0	0	0
GGHHRR	Bright yellow		0	2	0
GGHHRr	Creamy yellow		0	0	0
GGHHRR	Bright yellow		4	4	0
GGHHRR	Bright yellow		0	0	0
GGHHrr			4	4	4
GGHHrr			0	0	0
GGHHRR	Bright yellow		0	0	0
GGHHRr	Bright yellow		0	0	0

Cross 5c : 1L x 8h

Host Morphological Characters	ISOLATES X	Y	3	4	5
GGHHRr	0	0	3	0	3
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	3	0	2
GGHHrr	0	0	0	0	1
GGHHRR	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHrr	4	0	3	0	4
GGHHRr	0	0	0	0	0
GGHHRr	0	0	3	0	2
GGHHRR	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRR	0	0	3	0	2
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	3	0	3
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0

Host Morphological Characters	X	Y	ISOLATES 3	4	5
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	3	0	4
GGHHrr	0	0	4	0	3
GGHHRR	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRr	0	0	2	0	2
GGHHRr	0	0	2	0	2
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	4	0	4
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHrr	2	0	3	0	4
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	4	0	4
GGHHrr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	4	0	4	0	4

Host Morphological Characters	X	Y	ISOLATES 3	4	5
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRR	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	4	0	2	0	4
GGHHRr	0	0	4	0	3
GGHHRr	0	0	0	2	0
GGHHrr	0	0	0	0	0
GGHHrr			3	0	2-(3)
GGHHrr			0	0	0
GGHHrr			3	4	4
GGHHrr			2	3	3
GGHHRr			0	0	0
GGHHrr			0	0	0
GGHHRr			0	0	0
GGHHRr			4	0	3
GGHHrr			4	0	4
GGHHRr			0	0	0
GGHHRR			0	0	0
GGHHRR			0	0	0
GGHHRr			0	0	0
GGHHRr			0	0	0

Host Morphological Characters	ISOLATES 3	4	6
GGHHRR	2	0	3
GGHHRr	2	0	4
GGHHRR	4	0	3
GGHHRR	0	0	0
GGHHRR	0	0	0
GGHHRR	0	0	0
GGHHRR	3	0	4
GGHHRR	0	0	0
GGHHRr	3	0	3
GGHHrr	0	0	0
GGHHRr	0	0	0
GGHHrr	4	4	4
GGHHRr	0	3	0
GGHHRr	3	0	3
GGHHRR	0	0	0
GGHHRr	0	0	0
GGHHRr	0	0	0
GGHHRR	4	0	4
GGHHRr	0	0	0
GGHHRR	0	0	0
GGHHRr	0	0	0
GGHHRr	0	0	0
GGHHRR	0	0	0
GGHHRR	0	0	0
GGHHrr	0	0	0
GGHHRr	0	0	0

Host Morphological Characters	ISOLATES 3	4	6
GGHHRr	0	0	0
GGHHRr	3	4	4
GGHHRr	0	0	0
GGHHrr	0	0	0
GGHHRR	0	0	0
GGHHrr	0	0	0
GGHHRR	0	0	0
GGHHRr	4	3	4
GGHHRr	0	0	0
GGHHRr	0	0	0
GGHHRr	0	0	0
GGHHRr	0	0	0
GGHHRR	2	0	2
GGHHRr	0	0	0
GGHHrr	0	0	0
GGHHrr	0	2	0

Cross 5d : 10_j x 1d

Host Morphological Characters		ISOLATES				
	Ray floret corolla colour	X	Y	3	4	5
GGhhrr		0	0	0	4	0
GGhhRr	Bright yellow	0	0	0	0	0
GGhhRR	Creamy yellow	0	0	4	2	3
GGhhrr		4	0	0	0	0
GGhhRR	Creamy yellow	0	0	0	0	0
GGhhRR	Bright yellow	0	0	2	4	0
GGhhRr	Creamy yellow	0	0	2	0	2
GGhhRr	Bright yellow	2	0	3	0	0
GGhhrr		0	0	0	0	0
GGhhRR	Bright yellow	0	0	4	0	0
GGhhRr	Creamy yellow	0	0	3	0	0
GGhhRr	Bright yellow	3	0	0	4	0
GGhhrr		4	4	2	4	3
GGhhRr	Bright yellow	0	0	3	0	0
GGhhRR	Bright yellow	0	0	4	0	3
GGhhRr	Bright yellow	0	4	3	4	0
GGhhRr	Bright yellow	2	0	0	0	0
GGhhRr	Bright yellow	2	3	4	0	0
GGhhRr	Bright yellow	0	0	0	2	0
GGhhRr	Creamy yellow	0	0	2	0	0
GGhhrr		4	2	3	4	4
GGhhRR	Creamy yellow	0	0	3	0	0
GGhhrr		0	0	0	0	0
GGhhRr	Creamy yellow	4	0	0	2	4
GGHHRR		0	0	4	0	0
GGHHRr		0	0	3	3	3
GGHHRR		0	0	0	0	0

Cross 5e : 1L x 10 $_j$

Host Morphological Characters	X	Y	3	4	5
		ISOLATES			
GGHHrr	0	0	0	0	4
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHrr	0	0	0	0	0
GGHHRr	0	0	4	0	4
GGHHRr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	2	0	2
GGHHrr	0	0	3	0	4
GGHHrr	0	0	0	0	0
GGHHRr	0	0	0	0	0
GGHHRr	0	0	4	0	2
GGHHrr	0	0	0	4	0
GGHHRR	0	0	3	0	0
GGHHRR	4	0	2	0	2
GGHHRR	0	0	4	0	0
GGHHRr	3	0	4	0	3
GGHHrr	0	0	0	0	0
GGHHRr	0	0	2	0	0
GGHHRr	2	0	3	0	0
GGHHRr	0	0	4	0	4
GGHHRR	0	0	0	0	0
GGHHRr	0	0	2	0	0
GGHHRr	0	0	3	0	4
GGHHrr	0	0	0	0	0

Cross 5f : 14c x 3g

Morphological Characters	ISOLATES Y		4
GGHHRR	2-(3)		4
ggHHRr	0		0
GgHHRr	0		0
ggHHRr	0		0
GgHHRr	0		0
GgHHRR	0		0
ggHHrr	0		4
GGHHRR	0		0
GgHHRr	4		4
ggHHRR	0		4
GgHHrr	0		0
GgHHrr	4		0
GGHHRr	0		0
ggHHRR	0		0
ggHHRR	2-(3)		0
GGHHRR	0		0
GgHHRr	4		4
GGHHRr	0		0
GGHHRr	0		0
GgHHRR	0		4
GgHHRr	4		0
GGHHrr	4		4
ggHHrr	0		0
GgHHRr	0		0
GGHHRr	0		0
GgHHRr	0		0

Morphological Characters	ISOLATES Y		4
GGHHrr	0		4
GgHHRR	0		0
GgHHRr	0		0
GGHHRr	4		4
ggHHrr	0		0
GgHHRr	0		0

APPENDIX TABLES 6A - 6E

Data on some primary values of growth of *E. fischeri* infected and non infected groundsel plants taken at weekly intervals during the growth period between the sixth and twelfth weeks after sowing.

Replicates	% Infection	Total No. of Leaves	No. of Senescent Leaves	Leaf Area (dm^2)	Fresh weights (g)			Dry weights (g)				Chlorophyll content of leaves (mg/dm^2)	Chlorophyll content (mg/g)	
					Root	Stem	Leaf	Root	Stem	Leaf	Total Plant		Leaves	Stem
INFECTED														
1	15	22	0	0.5176	0.1787	0.0900	1.4586	0.0147	0.0085	0.0962	0.1194	0.2032	0.0175	0.0682
2	19	30	0	0.6859	0.2010	0.2067	2.1910	0.0144	0.0172	0.1405	0.1721	0.2297	0.1452	0.0442
3	18	23	0	0.6454	0.2010	0.1261	1.7664	0.0141	0.0107	0.1154	0.1402	0.2133	0.0745	0.037[illegible]
4	18	34	0	0.8801	0.3974	0.2951	3.0323	0.0270	0.0245	0.2097	0.2612	0.2822	0.3448	0.005[illegible]
5	16	30	0	0.8349	0.3081	0.1781	2.3639	0.0205	0.0148	0.1611	0.1964	0.2439	0.2274	0.044[illegible]
Mean	17.2	27.8	0	0.7128	0.2572	0.1792	2.1624	0.0181	0.0151	0.1446	0.1779	0.2345	0.1619	0.0398
SE	2.04	6.4	0	0.18	0.12	0.10	0.75	0.01	0.01	0.05	0.07	0.04	0.11	0.03
UN-INFECTED														
1	0	33	0	0.8873	0.4045	0.2415	2.6900	0.0256	0.0213	0.1770	0.2239	0.2370	0.1856	0.0306
2	0	31	0	0.8792	0.3686	0.3057	2.6919	0.0242	0.0285	0.1639	0.2166	0.2258	0.1386	0.0356
3	0	28	0	0.7609	0.2290	0.1842	2.2243	0.0183	0.0161	0.1542	0.1886	0.2318	0.1571	0.0360
4	0	40	0	1.3469	0.4590	0.3741	3.7503	0.0348	0.0328	0.2593	0.3269	0.2753	0.3912	0.0526
5	0	32	0	0.9324	0.2883	0.2523	2.8640	0.0225	0.0220	0.1960	0.2405	0.2234	0.1112	0.0385
Mean	0	32.8	0	0.9613	0.3499	0.2716	2.8441	0.0251	0.0241	0.1901	0.2393	0.2387	0.1968	0.0387
SE	0	5.52	0	0.28	0.11	0.09	0.70	0.01	0.01	0.05	0.06	0.03	0.10	0.01

Appendix Table 6B, 2nd Harvest (Seven weeks after sowing)

Replicates	% Infection	Total No. of Leaves	No. of Senescent Leaves	Leaf Area (dm^2)	Fresh weights (g)			Dry weights (g)				Chlorophyll content of leaves (mg/dm^2)	Chlorophyll conte (mg/g)	
					Root	Stem	Leaf	Root	Stem	Leaf	Total Plant		Leaves	Sten
INFECTED														
1	33	43	3	2.0977	0.6754	0.6930	5.6210	0.0587	0.0623	0.4231	0.5441	0.4112	2.0388	0.230
2	26	39	2	1.2038	0.5018	0.4118	3.2422	0.0338	0.0396	0.2403	0.3137	0.3211	1.6084	0.439
3	24	39	4	1.3297	0.8405	0.4319	3.8790	0.0510	0.0450	0.3156	0.4116	0.3826	1.6118	0.225
4	35	43	3	1.2337	0.5187	0.4646	3.2716	0.0400	0.0510	0.2862	0.3772	0.4807	2.0720	0.502
5	32	40	1	1.5703	0.8507	0.5863	4.1130	0.0600	0.0573	0.3228	0.4401	0.1643	0.7993	0.460
Mean	30	40.8	2.6	1.4870	0.6774	0.5175	4.0254	0.0487	0.0510	0.3176	0.4173	0.3520	1.6261	0.371
SE	5.90	2.55	1.42	0.46	0.21	0.15	1.20	0.01	0.01	0.08	0.11	0.15	0.64	0.16
CONTROL														
1	0	54	0	2.4422	0.8409	0.8655	6.8029	0.0583	0.0767	0.4619	0.5969	0.3807	2.0130	0.408
2	0	45	1	2.1547	1.2621	0.6907	6.1425	0.0871	0.0704	0.4841	0.6416	0.2669	1.1880	0.316
3	0	50	0	2.2307	1.1608	0.6813	6.3522	0.0840	0.0723	0.4871	0.6434	0.4158	1.9041	0.451
4	0	48	0	2.3494	1.2955	0.8983	6.5821	0.0815	0.0822	0.4406	0.6043	0.3553	1.8947	0.126
5	0	48	0	1.9901	1.1353	0.7099	6.2390	0.0715	0.0600	0.3837	0.5152	0.2324	1.2056	0.349
Mean	0	49.0	0.20	2.2334	1.1389	0.7691	6.4237	0.0765	0.0723	0.4515	0.6003	0.3302	1.6411	0.330
SE	0	4.12	0.56	0.22	0.22	0.13	0.33	0.01	0.01	0.05	0.06	0.10	0.51	0.15

Appendix Table 6C, 3rd Harvest (Eight weeks after sowing)

Replicates	% Infection	Total No. of Leaves	No. of Senescent Leaves	Leaf Area (dm^2)	Fresh weights (g)			Dry weights (g)				Chlorophyll content of leaves (mg/dm^2)	Chlorophyll conte (mg/g)	
					Root	Stem	Leaf	Root	Stem	Leaf	Total Plant		Leaves	Ste
INFECTED														
1	60	60	3	2.3670	0.8700	1.3455	6.5135	0.0693	0.1141	0.4255	0.6089	0.3331	1.8529	0.544
2	60	56	2	2.4132	0.6785	1.4995	4.7359	0.0867	0.1320	0.4593	0.6780	0.3349	1.7594	0.494
3	65	58	2	2.4227	1.1400	1.5555	6.5135	0.0844	0.1278	0.5710	0.7832	0.3826	1.6235	0.560
4	60	67	2	3.7598	1.2383	2.7059	8.2448	0.0958	0.2219	0.6694	0.9871	0.2960	1.6627	0.478
5	65	64	4	3.1153	1.2395	2.2255	7.2988	0.0992	0.1848	0.6295	0.9135	0.2456	1.2156	0.438
Mean	62.00	61.00	2.60	2.8156	1.0333	1.8664	6.6613	0.0871	0.1561	0.5509	0.7941	0.3184	1.6228	0.503
SE	3.40	5.50	1.11	0.76	0.31	0.72	1.60	0.01	0.06	0.13	0.20	0.06	0.30	0.06
CONTROL														
1	0	73	1	4.1449	1.2482	2.1200	10.5415	0.1238	0.1787	0.8535	1.1560	0.3147	1.5282	0.365
2	0	76	0	4.4769	1.7600	2.6820	11.7045	0.1270	0.2326	0.8465	1.2061	0.2538	1.3422	0.418
3	0	71	1	3.5847	1.1752	2.5704	8.1648	0.1072	0.2302	0.6323	0.9697	0.2947	1.6709	0.413
4	0	76	0	4.6682	1.9157	3.1167	11.7001	0.1596	0.2677	0.9067	1.3340	0.3143	1.6184	0.550
5	0	65	1	4.2183	1.5810	2.2060	10.6342	0.1260	0.1706	0.8081	1.1047	0.2907	1.5173	0.428
Mean	0	72.2	0.60	4.2186	1.5360	2.5390	10.5490	0.1287	0.2160	0.8094	1.1541	0.2936	1.5354	0.435
SE	0	5.66	0.68	0.51	0.40	0.50	1.80	0.02	0.05	0.13	0.17	0.03	0.16	0.09

Appendix Table 6D, 4th Harvest (Nine weeks after sowing)

Replicates	% Infection	Total No. of Leaves	No. of Senescent Leaves	Leaf Area (dm^2)	Fresh weights (g)			Dry weights (g)				Chlorophyll content of leaves (mg/dm^2)	Chlorophyll conte (mg/g)	
					Root	Stem	Leaf	Root	Stem	Leaf	Total Plant		Leaves	Ster
INFECTED														
1	75	69	5	4.2530	1.4965	3.1358	11.0527	0.1000	0.2605	0.8227	1.1832	0.3285	1.79061	0.497
2	75	84	4	4.2192	1.5326	3.5560	11.0770	0.1120	0.2981	0.8494	1.2595	0.3091	1.3739	0.481
3	70	69	7	4.2761	1.5241	2.9271	6.2308	0.1088	0.2720	0.6691	1.0499	0.3565	1.6895	0.503
4	75	73	6	3.4668	1.2424	2.5335	7.4698	0.1048	0.2357	0.6759	1.0164	0.2716	1.4171	0.578
5	70	66	6	2.8474	1.2413	2.4929	7.0979	0.0985	0.2285	0.5867	0.9137	0.2650	1.2359	0.657
Mean	73	72.20	5.60	3.8125	1.4074	2.9291	8.5856	0.1048	0.2590	0.7208	1.0845	0.3061	1.5014	0.543
SE	3.40	8.76	1.42	0.79	0.19	0.55	2.87	0.01	0.03	0.14	0.17	0.05	0.29	0.09
CONTROL														
1	0	95	2	5.5076	2.3710	5.1332	18.1442	0.1661	0.4353	1.2606	1.8620	0.3000	1.4242	0.299
2	0	86	3	4.9444	2.8062	7.3582	13.7839	0.1815	0.5856	1.1704	1.9375	0.2380	1.2127	0.459
3	0	100	1	6.1968	3.1000	8.4672	19.4780	0.2065	0.6605	1.3070	2.1740	0.1899	0.9798	0.600
4	0	96	1	6.2777	3.5915	7.6236	19.4594	0.2251	0.6229	1.2852	2.1332	0.2984	1.5846	0.409
5	0	93	2	5.7432	2.7883	8.1100	18.2546	0.1910	0.6434	1.2910	2.1254	0.2725	1.3366	0.701
Mean	0	94.20	1.80	5.7339	2.9314	7.3384	17.8240	0.1940	0.5895	1.2628	2.0464	0.2598	1.3076	0.494
SE	0	6.40	1.04	0.68	0.56	1.62	2.92	0.03	0.11	0.07	0.17	0.06	0.28	0.20

Appendix Table 6E, 5th Harvest (Ten weeks after sowing)

Replicates	% Infection	Total No. of Leaves	No. of Senescent Leaves	Leaf Area (dm^2)	Fresh weights (g)			Dry weights (g)				Chlorophyll content of leaves (mg/dm^2)	Chlorophyll conten (mg/g)	
					Root	Stem	Leaf	Root	Stem	Leaf	Total Plant		Leaves	Stem
INFECTED														
1	90	90	7	3.0670	1.5669	3.4964	8.6220	0.1050	0.3417	0.8600	1.3067	0.2849	1.0162	0.6125
2	90	88	9	2.8576	0.7931	2.7282	6.7556	0.0795	0.2420	0.7990	1.1205	0.2332	1.1917	0.6228
3	90	88	14	2.0848	0.9728	3.3648	4.8160	0.1015	0.3325	0.6456	1.0796	0.2197	1.0323	0.5557
4	90	95	18	2.0667	0.8450	2.7770	4.6715	0.0980	0.2762	0.5899	0.9641	0.2711	1.3001	0.4565
5	90	92	10	2.6962	1.1211	3.7677	6.3203	0.1010	0.3526	0.7925	1.2461	0.2048	1.0369	0.5590
Mean	90	90.60	11.60	2.5545	1.0598	3.2268	6.2371	0.0970	0.3090	0.7374	1.1434	0.2427	1.1154	0.5613
SE	0	3.69	5.46	0.57	0.39	0.57	2.01	0.01	0.06	0.14	0.17	0.04	0.16	0.08
CONTROL														
1	0	132	3	6.2556	1.8235	7.3000	17.5904	0.1923	0.6988	1.5880	2.4791	0.2563	1.0096	0.5293
2	0	107	0	5.9923	2.0353	6.8224	14.8616	0.1762	0.6345	1.2527	2.0634	0.2598	1.0557	0.4854
3	0	106	3	5.2388	1.6520	5.5815	15.8173	0.1429	0.4828	1.3854	2.0111	0.2784	1.0526	0.3842
4	0	120	3	5.7926	2.1427	7.0037	14.8532	0.1830	0.6169	1.2832	2.0831	0.2205	0.9954	0.4464
5	0	122	5	5.7131	1.1724	6.0610	15.0699	0.1465	0.6076	1.4465	2.2006	0.2394	0.9456	0.5009
Mean	0	117.40	2.80	5.7985	1.7652	6.5537	15.6385	0.1682	0.6081	1.3912	2.1675	0.2509	1.0118	0.4692
SE	0	13.60	2.22	0.47	0.48	0.88	1.44	0.03	0.10	0.17	0.23	0.03	0.06	0.07

APPENDIX TABLES 7A - 7E

Data on the effects of infection on the development of individual leaves the main axis (A) and on the development of leaves on individual latera branches (B), taken at weekly intervals during the growth period betwe the eighth and twelfth weeks after sowing.

Appendix Table 7A, 1st Harvest (8 weeks after sowing)

A. MAIN AXIS (INFECTED PLANTS)

Leaf No. on main axis	1st Plant		2nd Plant		3rd Plant		Mean % Infection	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection			
2	* -		0.0016	0	-		0.0000	0.0005	0.002
3	** -		0.0156	25	-		8.33	0.0052	0.022
4	0.0337	25	0.0314	33	0.0302	50	36.00	0.0318	0.004
5	0.0377	90	0.0633	50	0.0454	70	70.00	0.0488	0.033
6	0.0476	50	0.0978	75	0.0943	35	53.33	0.0799	0.070
7	0.0902	10	0.1350	95	0.1089	85	63.33	0.1114	0.056
8	0.1042	95	0.1349	50	0.1132	75	73.33	0.1174	0.039
9	0.1150	30	0.1544	20	0.1455	50	33.33	0.1383	0.051
10	0.1379	25	0.1609	25	0.1248	10	20.00	0.1412	0.045
11	0.1105	10	0.1049	5	0.1234	2	5.67	0.1129	0.023
12	0.1072	5	0.1008	2	0.0917	2	3.00	0.0999	0.019
13	0.1058	5	0.0648	2	0.0507	0	2.33	0.0738	0.071
14	0.0898	2	0.0387	0	0.0237	0	0.67	0.0573	0.080
15	0.0987	2	0.0183	0	0.0124	0	0.67	0.0431	0.120
16	0.0500	2	0.0034	0	0.0048	0	0.67	0.0194	0.066
17	0.0107	0	0.0029	0	0.0011	0	0.00	0.0049	0.013
18	0.0064	0	0.0009	0	0.0010	0	0.00	0.0028	0.008
19	0.0021	0	0.0005	0	0.0005	0	0.00	0.0010	0.002
20	0.0011	0	0.0003	0	0.0004	0	0.00	0.0006	0.001
21	0.007	0	0	0	0	0	0.00	0.0002	0.001

* Senesced

** Each reading is a mean of three measurements

B. LATERAL BRANCHES (INFECTED PLANTS)

Branch No.	1st Plant			2nd Plant			3rd Plant			Mean % Infection	Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection					
3	0	0	0	5	0.0416	25	6	0.0377	10	11.67	3.67	7.98	0.0264	0.057
4	6	0.00607	25	7	0.1307	33	6	0.1110	10	22.67	6.33	1.33	0.0826	0.166
5	6	0.0805	30	6	0.1262	10	5	0.0369	1	13.67	5.67	1.43	0.0812	0.111
6	6	0.0751	25	5	0.0795	4	5	0.0467	0	9.67	5.33	1.43	0.0671	0.044
7	4	0.0230	2	3	0.0172	0	2	0.0016	0	0.67	3.00	2.48	0.0139	0.027
8	2	0.0142	0	2	0.0007	0	2	0.0009	0	0.00	2.00	0.00	0.0053	0.019
9	2	0.0012	0	2	0.0008	0	2	0.0004	0	0.00	2.00	0.00	0.0008	0.001
10	2	0.0004	0	2	0.0006	0	0	0	0	0.00	1.33	2.87	0.0003	0.0007
11	2	0.0004	0	0	0	0	0	0	0	0.00	0.67	2.87	0.0001	0.0006

A. MAIN AXIS (CONTROL)

Leaf No. on main axis	1st Plant	2nd Plant	3rd Plant	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	Individual leaf area (dm^2)	Individual leaf area (dm^2)		
1	-	0.0021	-	0.0007	0.003
2	0.0021	0.0019	-	0.0013	0.003
3	0.0186	0.0192	0.0165	0.0181	0.003
4	0.0317	0.0307	0.0323	0.0316	0.002
5	0.0559	0.0677	0.0500	0.0579	0.022
6	0.1150	0.1166	0.0889	0.1068	0.039
7	0.1337	0.1430	0.1360	0.1376	0.012
8	0.1413	0.1721	0.1537	0.1557	0.038
9	0.1456	0.1697	0.1420	0.1524	0.037
10	0.1543	0.1555	0.1737	0.1612	0.027
11	0.1583	0.1443	0.1414	0.1480	0.022
12	0.1410	0.1344	0.1212	0.1322	0.025
13	0.1087	0.1151	0.1092	0.1110	0.009
14	0.0836	0.0932	0.0730	0.0833	0.025
15	0.0433	0.0474	0.0486	0.0464	0.007
16	0.0197	0.0238	0.0183	0.0206	0.007
17	0.0051	0.0067	0.0053	0.0057	0.002
18	0.0031	0.0030	0.0031	0.0031	0.000
19	0.0008	0.0008	0.0009	0.0008	0.000
20	0.0006	0.0006	0.0003	0.0005	0.000
21	0.0003	0.0003	0	0.0002	0.000

B. LATERAL BRANCHES (CONTROL)

Branch No.	1st Plant		2nd Plant		3rd Plant		Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)				
1	1	0.0013	1	0.0027	0	0	0.67	1.43	0.0013	0.003
2	7	0.1052	7	0.1081	6	0.0677	6.67	1.43	0.0937	0.056
3	7	0.1397	7	0.1486	6	0.0929	6.67	1.43	0.1271	0.074
4	7	0.1056	6	0.1751	6	0.1326	6.33	1.43	0.1378	0.087
5	5	0.1250	6	0.1435	3	0.0874	4.67	3.79	0.1186	0.071
6	4	0.0394	4	0.0398	2	0.0238	3.33	2.87	0.0343	0.023
7	2	0.0089	2	0.0147	2	0.0137	2.00	0.0	0.0124	0.008
8	2	0.0037	2	0.0012	2	0.0038	2.00	0.0	0.0029	0.004
9	2	0.0005	2	0.0007	2	0.0006	2.00	0.0	0.0006	0.0002
10	2	0.0004	2	0.0004	2	0.0003	2.00	0.0	0.0004	0.0001
11	2	0.0003	1	0.0004	0	0	1.00	2.48	0.0002	0.0005

Appendix Table 7B, 2nd Harvest (9 weeks after sowing)

A. MAIN AXIS (INFECTED PLANTS)

Leaf No. on main axis	1st Plant		2nd Plant		Mean % Infection	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection			
4	-		0.0384	10	5.0	0.0192	0.244
5	-		-	-	-	-	-
6	0.1093	75	0.0754	60	67.50	0.0923	0.215
7	0.1387	90	0.1541	75	82.50	0.1464	0.098
8	0.1534	50	0.1467	90	70.00	0.1500	0.043
9	0.1523	75	0.1869	80	77.50	0.1696	0.220
10	0.1533	80	0.1860	75	77.50	0.1696	0.208
11	0.1466	40	0.1595	90	65.00	0.1530	0.082
12	0.1285	50	0.1460	90	70.00	0.1372	0.111
13	0.1226	40	0.1274	90	65.00	0.1250	0.030
14	0.1167	20	0.1244	75	47.50	0.1205	0.049
15	0.0994	12	0.1207	50	31.00	0.1100	0.135
16	0.0535	3	0.0761	25	14.00	0.0648	0.144
17	0.0348	0	0.0415	5	2.50	0.0381	0.043
18	0.0192	0	0.0268	2	1.00	0.0230	0.048
19	0.0011	0	0.0249	0	0.00	0.0130	0.151
20	0.0006	0	0.0054	0	0.00	0.0030	0.030
21	0.0003	0	0.0010	0	0.00	0.0006	0.004
22	0.0001	0	0.0009	0	0.00	0.0005	0.005

B. LATERAL BRANCHES (INFECTED PLANTS)

Branch No.	1st Plant			2nd Plant			Mean % Infection	Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection					
3	9	0.1536	75	8	0.1393	95	85	8.50	6.35	0.1465	0.091
4	8	0.1753	85	7	0.1889	50	67.5	7.50	6.35	0.1821	0.086
5	7	0.1579	85	7	0.1570	95	90.0	7.00	0.00	0.1575	0.006
6	7	0.1597	30	8	0.1502	90	60.0	7.50	6.35	0.1549	0.060
7	5	0.0651	40	4	0.0703	50	45.0	4.50	6.35	0.0677	0.033
8	3	0.0150	1	4	0.0336	50	25.5	3.50	6.35	0.0243	0.118
9	3	0.0124	0	2	0.0050	0	0.0	2.50	6.35	0.0087	0.047
10	2	0.0014	0	2	0.0040	0	0.0	2.00	0.00	0.0027	0.016
11	2	0.0002	0	1	0.0005	0	0.0	1.50	6.35	0.0003	0.002
12	0	0	0	1	0.0004	0	0.0	0.50	6.35	0.0002	0.002

A. MAIN AXIS (CONTROL)

Leaf No. on main axis	1st Plant	2nd Plant	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	Individual leaf area (dm^2)		
3	0.0186	0.0185	0.0185	0.001
4	0.0395	0.0359	0.0377	0.023
5	0.0688	0.0700	0.0694	0.008
6	0.1163	0.1192	0.1177	0.018
7	0.1484	0.1472	0.1478	0.008
8	0.1854	0.1828	0.1841	0.013
9	0.1914	0.1800	0.1857	0.072
10	0.2010	0.1952	0.1981	0.030
11	0.2079	0.1147	0.2013	0.084
12	0.2067	0.1891	0.1979	0.111
13	0.2068	0.2060	0.2064	0.004
14	0.1713	0.1714	0.1713	0.0006
15	0.1875	0.1699	0.1787	0.112
16	0.1055	0.0821	0.0938	0.121
17	0.0830	0.0797	0.0813	0.017
18	0.0898	0.0669	0.0783	0.118
19	0.0598	0.0465	0.0531	0.084
20	0.0268	0.0245	0.0256	0.015
21	0.0154	0.0127	0.0140	0.017
22	0.0044	0.0032	0.0038	0.008
23	0.0014	0	0.0007	0.009

B. LATERAL BRANCHES (CONTROL)

Branch No.	1st Plant		2nd Plant		Mean Total No. of leaves	SE	Mean Leaf area (dm^2)	SE
	Total No. of leaves	Leaf area (dm^2)	Total No. of leaves	Leaf area (dm^2)				
3	8	0.1612	9	0.1639	8.50	6.35	0.1625	0.017
4	8	0.2608	8	0.2279	8.00	0.00	0.2443	0.209
5	8	0.2640	9	0.2304	8.50	6.35	0.2472	0.213
6	9	0.2966	8	0.2395	8.50	6.35	0.2680	0.263
7	6	0.0933	6	0.0835	6.00	0.00	0.0884	0.062
8	4	0.0699	4	0.0662	4.00	0.00	0.0680	0.023
9	3	0.0095	4	0.0081	3.50	6.35	0.0088	0.009
10	3	0.0085	4	0.0091	3.50	6.35	0.0088	0.004
11	3	0.0033	2	0.0029	2.50	6.35	0.003	0.002
12	3	0.0021	2	0.0019	2.50	6.35	0.0020	0.001
13	2	0.0011	2	0.0010	2.00	0.00	0.0010	0.001
14	2	0.009	2	0.0005	2.00	0.00	0.0007	0.002
15	0	0	1	0.0005	0.50	6.35	0.0002	0.003

Appendix Table 7C, 3rd Harvest (10 weeks after sowing)

A. MAIN AXIS (INFECTED PLANTS)

Leaf No. on main axis	1st Plant		2nd Plant		3rd Plant		Mean % Infection	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection			
5	-	-	0.0545	25	-	-	8.33	0.0182	0.078
6	-	-	0.0632	25	-	-	8.33	0.0211	0.091
7	-	-	0.0964	5	-	-	1.67	0.0321	0.138
8	-	-	0.1163	10	-	-	3.33	0.0388	0.167
9	0.1463	85	0.1296	25	-	-	36.67	0.0920	0.199
10	0.1599	98	0.1527	40	0.1667	98	78.67	0.1598	0.017
11	0.1373	99	0.1533	60	0.1503	98	85.67	0.1470	0.021
12	0.1828	95	0.1569	90	0.1637	98	94.33	0.1678	0.033
13	0.1267	96	0.1748	90	0.1627	98	94.67	0.1547	0.062
14	0.1564	75	0.1500	90	0.1500	95	86.67	0.1521	0.009
15	0.1577	75	0.1538	95	0.1231	95	88.33	0.1449	0.047
16	0.1024	10	0.1340	50	0.1437	90	50.00	0.1267	0.054
17	0.0964	10	0.1001	45	0.0868	75	43.33	0.0944	0.017
18	0.0574	6	0.1176	10	0.0855	75	30.33	0.0868	0.075
19	0.0408	2	0.0644	2	0.0669	10	4.67	0.0574	0.036
20	0.0205	0	0.0537	2	0.0468	6	2.67	0.0403	0.043
21	0.0052	0	0.0118	0	0.0327	2	0.67	0.0166	0.036
22	0.0026	0	0.0050	0	0.0139	0	0.00	0.0072	0.015
23	0.0007	0	0.0027	0	0.0093	0	0.00	0.0042	0.011
24	0.0004	0	0.0007	0	0.0052	0	0.00	0.0021	0.007
25	0	0	0.0004	0	0.0006	0	0.00	0.0003	0.001
26	0	0	0.0002	0	0	0	0.00	0.0001	0.000

B. LATERAL BRANCHES (INFECTED PLANTS

Branch No.	1st Plant			2nd Plant			3rd Plant			Mean % Infection	Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection					
3	3	0.0308	98	5	0.0367	25	5	0.0475	98	73.67	4.33	2.87	0.0383	0.021
4	9	0.1736	90	9	0.0951	60	5	0.0584	98	82.67	7.67	5.73	0.1090	0.146
5	9	0.1777	95	10	0.1535	45	7	0.1041	98	79.33	8.67	3.79	0.1451	0.093
6	9	0.1761	95	8	0.1287	60	8	0.1667	98	84.33	8.33	1.43	0.1572	0.062
7	6	0.1002	85	7	0.1010	50	8	0.1133	98	77.67	7.00	2.48	0.1048	0.018
8	4	0.0235	98	5	0.0624	30	6	0.0730	98	75.33	5.00	2.48	0.0530	0.065
9	4	0.0305	50	4	0.0657	10	5	0.0042	95	51.67	4.33	1.43	0.0335	0.077
10	2	0.0053	50	4	0.0324	5	2	0.0053	95	50.00	2.67	2.87	0.0143	0.039
11	2	0.0010	2	4	0.0192	0	2	0.0012	60	20.67	2.67	2.87	0.0071	0.026
12	2	0.0010	0	2	0.0080	0	2	0.0007	50	16.67	2.00	0.00	0.0032	0.010
13	1	0.0004	0	2	0.0019	0	0	0	0	0.00	1.00	2.48	0.0006	0.003
14	0	0	0	2	0.0014	0	0	0	0	0.00	0.67	2.87	0.0005	0.002
15	0	0	0	2	0.0010	0	0	0	0	0.00	0.67	2.87	0.0003	0.001
16	0	0	0	2	0.0006	0	0	0	0	0.00	0.67	2.87	0.0002	0.001

A. MAIN AXIS (CONTROL)

Leaf No. on main axis	1st Plant	2nd Plant	3rd Plant	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	Individual leaf area (dm^2)	Individual leaf area (dm^2)		
3	0.0162	-	-	0.0054	0.023
4	0.0259	0.0350	-	0.0203	0.045
5	0.0542	0.0538	0.0609	0.0563	0.010
6	0.0491	0.1200	0.0963	0.0885	0.090
7	0.0751	0.1343	0.1254	0.1116	0.079
8	0.1541	0.1531	0.1569	0.1547	0.005
9	0.1911	0.2059	0.1887	0.1952	0.023
10	0.2045	0.1868	0.1778	0.1897	0.034
11	0.1621	0.2150	0.1989	0.1920	0.067
12	0.1925	0.2534	0.1815	0.2091	0.096
13	0.1923	0.2670	0.1966	0.2186	0.104
14	0.2042	0.2069	0.2072	0.2061	0.004
15	0.1873	0.1681	0.2059	0.1871	0.047
16	0.1788	0.1780	0.1786	0.1785	0.001
17	0.1732	0.1676	0.1742	0.1717	0.009
18	0.1594	0.1708	0.1650	0.1651	0.014
19	0.1606	0.1638	0.1492	0.1579	0.019
20	0.1135	0.0894	0.1061	0.1030	0.031
21	0.0677	0.0602	0.0628	0.0636	0.009
22	0.0543	0.0636	0.0556	0.0578	0.012
23	0.0136	0.0164	0.0126	0.0142	0.005
24	0.0086	0.0077	0.0069	0.0077	0.002

B. LATERAL BRANCHES (CONTROL)

Branch No.	1st Plant		2nd Plant		3rd Plant		Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)				
3	8	0.1060	8	0.1286	8	0.1110	8.00	0.000	0.1152	0.029
4	9	0.2021	9	0.2088	9	0.2168	9.00	0.000	0.2092	0.018
5	11	0.3845	8	0.2625	11	0.4022	10.00	4.30	0.3497	0.189
6	11	0.3913	12	0.4481	12	0.4495	11.67	1.43	0.4296	0.082
7	10	0.2300	7	0.1972	9	0.2210	8.67	3.79	0.2161	0.042
8	8	0.1286	6	0.0787	6	0.0875	6.67	2.87	0.0983	0.066
9	7	0.1011	6	0.0596	6	0.0743	6.33	1.43	0.0783	0.052
10	7	0.1057	4	0.0402	4	0.0553	5.00	4.30	0.0671	0.085
11	6	0.0965	4	0.0176	5	0.0266	5.00	2.48	0.0469	0.107
12	4	0.0153	3	0.0017	2	0.0032	3.00	2.48	0.0067	0.018
13	4	0.0066	2	0.0009	2	0.0018	2.67	2.87	0.0031	0.007
14	4	0.0226	4	0.0096	2	0.0029	3.33	2.87	0.0117	0.025
15	2	0.0041	2	0.0011	2	0.0014	2.00	0.00	0.0022	0.004
16	2	0.0017	3	0.0014	2	0.0019	2.33	1.43	0.0017	0.001
17	2	0.0011	3	0.0027	2	0.0010	2.33	1.43	0.0016	0.002
18	2	0.0013	3	0.0014	2	0.0009	2.33	1.43	0.0012	0.001
19	2	0.0008	3	0.0050	0	0	1.67	3.79	0.0019	0.007
20	0	0	2	0.0055	0	0	0.67	2.87	0.0018	0.008
21	0	0	1	0.0006	0	0	0.33	1.43	0.0002	0.001

Appendix Table 7D, 4th Harvest (11 weeks after sowing)

A. MAIN AXIS (INFECTED PLANTS)

Leaf No. on main axis	1st Plant		2nd Plant		3rd Plant		Mean % Infection	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection			
9	0.1741	99	-	-	0.1639	85	61.33	0.1127	0.243
10	-	-	-	-	-	-	-	-	
11	-	-	-	-	0.1724	99	33.00	0.0575	0.247
12	0.2213	97	-	-	0.1786	95	64.00	0.1333	0.291
13	0.1962	98	0.1830	98	0.1901	99	98.33	0.1898	0.016
14	0.2393	96	0.2252	95	0.1600	98	96.33	0.2082	0.105
15	0.1925	98	0.1822	95	0.1606	98	97.00	0.1784	0.040
16	0.1681	98	0.1929	95	0.1456	98	97.00	0.1689	0.059
17	0.1563	90	0.1865	95	0.1084	95	93.33	0.1504	0.098
18	0.1500	90	0.1675	85	0.1306	90	88.33	0.1494	0.046
19	0.1609	95	0.1192	55	0.0679	60	70.00	0.1160	0.116
20	0.1042	95	0.0923	35	0.0502	10	46.67	0.0822	0.070
21	0.0549	85	0.0574	8	0.0414	5	32.67	0.0512	0.021
22	0.0597	80	0.0322	5	0.0208	5	30.00	0.0376	0.050
23	0.0200	55	0.0223	2	0.0102	2	19.67	0.0175	0.016
24	0.0026	5	0.0011	0	0.0018	0	1.67	0.0018	0.002

B. LATERAL BRANCHES (INFECTED PLANTS

Branch No.	1st Plant			2nd Plant			3rd Plant			Mean % Infection	Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection					
3	7	0.0963	95	7	0.0799	98	8	0.1298	90	94.33	7.33	1.43	0.1020	0.063
4	10	0.1611	98	6	0.0694	98	9	0.2070	98	98.00	8.33	5.17	0.1458	0.174
5	10	0.2558	98	9	0.1691	98	9	0.2568	90	95.33	9.33	1.43	0.2272	0.125
6	7	0.2003	98	9	0.2804	95	8	0.2069	99	97.33	8.00	2.48	0.2292	0.110
7	8	0.1461	98	9	0.2241	98	9	0.0831	99	98.33	8.67	1.43	0.1511	0.175
8	4	0.0155	99	4	0.0523	98	5	0.0774	99	98.67	4.33	1.43	0.0484	0.077
9	4	0.0291	95	4	0.0481	98	2	0.0218	95	96.00	3.33	2.87	0.0330	0.034
10	3	0.0037	40	4	0.0499	99	3	0.0222	95	78.00	3.33	1.43	0.0253	0.058
11	3	0.0068	40	2	0.0033	85	3	0.0208	95	73.33	2.67	1.43	0.0103	0.023
12	2	0.0019	2	2	0.0015	80	2	0.0024	50	44.00	2.00	0.00	0.0019	0.001
13	2	0.0014	0	2	0.0031	40	2	0.0051	10	16.67	2.00	0.00	0.0032	0.005
14	4	0.0082	10	2	0.0063	0	2	0.0013	0	4.00	2.67	2.87	0.0053	0.009
15	2	0.0023	0	2	0.0072	0	2	0.0026	0	0.67	2.00	0.00	0.0040	0.007
16	4	0.0145	5	2	0.0031	0	2	0.0026	0	1.67	2.67	2.87	0.0067	0.017
17	4	0.0221	25	2	0.0025	0	2	0.0010	0	8.33	2.67	2.87	0.0085	0.029
18	4	0.0143	10	2	0.0026	0	2	0.0026	0	3.33	2.67	2.87	0.0065	0.017
19	4	0.0368	10	2	0.0012	0	2	0.0013	0	3.33	2.67	2.87	0.0131	0.051
20	2	0.0112	10	2	0.0012	0	2	0.0007	0	3.33	2.00	0.00	0.0044	0.015
21	0	0	0	1	0.0006	0	2	0.0005	0	0	1.00	4.30	0.0004	0.001

A. MAIN AXIS (CONTROL PLANTS)

Leaf No. on main axis	1st Plant	2nd Plant	3rd Plant	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	Individual leaf area (dm^2)	Individual leaf area (dm^2)		
4	-	0.0303	0.0349	0.0217	0.047
5	0.0585	0.0476	0.0494	0.05183	0.014
6	0.0846	0.0794	0.8844	0.0841	0.011
7	0.1006	0.1023	0.1211	0.1080	0.028
8	0.1528	0.1678	0.1540	0.1582	0.021
9	0.1809	0.2070	0.1899	0.1926	0.033
10	0.1938	0.1956	0.1937	0.1944	0.003
11	0.1916	0.1983	0.2330	0.2076	0.055
12	0.2270	0.2152	0.2413	0.2278	0.032
13	0.2001	0.2386	0.2303	0.2230	0.050
14	0.2584	0.2704	0.2481	0.2590	0.028
15	0.2459	0.3247	0.2695	0.2800	0.100
16	0.1969	0.2976	0.2725	0.2557	0.130
17	0.2662	0.3208	0.2733	0.2868	0.074
18	0.2441	0.2642	0.2440	0.2508	0.029
19	0.1909	0.2054	0.1970	0.1978	0.018
20	0.1814	0.2049	0.2049	0.1971	0.034
21	0.0840	0.0984	0.1164	0.0996	0.040
22	0.0866	0.0704	0.0925	0.0832	0.028
23	0.0448	0.0364	0.0516	0.0443	0.019
24	0.0334	0.0265	0.0366	0.0322	0.013
25	0.0022	0.0030	0.0022	0.0025	0.001
26	0	0.0015	0.0004	0.0006	0.002

B. LATERAL BRANCHES (CONTROL PLANTS)

Branch No.	1st Plant		2nd Plant		3rd Plant		Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)				
3	6	0.0865	8	0.1143	7	0.0956	7.00	2.48	0.0988	0.035
4	9	0.2039	9	0.1965	12	0.2280	10.00	4.30	0.2095	0.041
5	11	0.3578	13	0.4079	12	0.3573	12.00	2.48	0.3743	0.072
6	13	0.4915	12	0.4346	12	0.4684	12.33	1.43	0.4648	0.071
7	11	0.3265	11	0.3351	11	0.3374	11.00	0.00	0.3330	0.014
8	7	0.0775	8	0.1305	8	0.1011	7.67	1.43	0.1030	0.066
9	9	0.1208	9	0.1006	7	0.1256	8.33	2.87	0.1157	0.033
10	7	0.1444	9	0.1543	7	0.1407	7.67	2.87	0.1465	0.017
11	5	0.0484	6	0.0627	4	0.0277	5.00	2.48	0.0463	0.043
12	5	0.0279	5	0.0381	4	0.0138	4.67	1.43	0.0266	0.030
13	3	0.0112	4	0.0324	2	0.0054	3.00	2.48	0.0163	0.035
14	3	0.0034	3	0.0521	2	0.0024	2.67	1.43	0.0193	0.070
15	4	0.0016	3	0.0675	3	0.0014	3.33	1.43	0.0235	0.095
16	3	0.0011	2	0.0033	3	0.0012	2.67	1.43	0.0019	0.003
17	3	0.0016	3	0.0043	3	0.0027	3.00	0.00	0.0029	0.003
18	3	0.0011	3	0.0032	2	0.0045	2.67	1.43	0.0029	0.004
19	3	0.0021	2	0.0016	4	0.0075	3.00	2.48	0.0037	0.008
20	3	0.0046	2	0.0016	4	0.0098	3.00	4.30	0.0053	0.010
21	2	0.0012	2	0.0009	3	0.0050	2.33	1.43	0.0024	0.006
22	2	0.0016	2	0.0010	2	0.0032	2.00	0.00	0.0019	0.003
23	0	0	2	0.0011	0	0	0.67	2.87	0.0004	0.002

Appendix Table 7E, 5th Harvest (12 weeks after sowing)

A. MAIN AXIS (INFECTED PLANTS)

Leaf No. on main axis	1st Plant		2nd Plant		3rd Plant		Mean % Infection	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection	Individual leaf area (dm^2)	% Infection			
12	0.1499	98	-	-	-	-	32.67	0.0500	0.215
13	0.1751	100	-	-	-	-	33.33	0.0584	0.251
14	0.1538	100	0.1841	100	-	-	66.67	0.1126	0.254
15	0.1925	100	0.1648	100	-	-	66.67	0.1191	0.258
16	0.1302	100	0.1590	100	0.1448	100	100.00	0.1447	0.036
17	0.1123	100	0.1413	100	0.1333	100	100.00	0.1290	0.037
18	0.1119	100	0.1327	100	0.1107	100	100.00	0.1184	0.031
19	0.0970	100	0.1023	100	0.1031	100	100.00	0.1008	0.008
20	0.0583	100	0.1311	100	0.0907	100	100.00	0.0937	0.090
21	0.0375	100	0.0824	100	0.0551	100	100.00	0.0583	0.056
22	0.0253	100	0.0547	100	0.0333	100	100.00	0.0378	0.038
23	0.0147	100	0.0282	100	0.0096	100	100.00	0.0175	0.024
24	0.0053	80	0.0063	100	0.0014	100	93.33	0.0043	0.006
25	0.0029	85	-	-	-	-	28.33	0.0010	0.004
26	0.0005	0	-	-	-	-	0.00	0.0002	0.007

B. LATERAL BRANCHES (INFECTED PLANTS)

Branch No.	1st Plant			2nd Plant			3rd Plant			Mean % Infection	Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection	Total No. of Leaves	Leaf Area (dm^2)	% Infection					
3	7	0.0079	98	6	0.0082	100	-	-	-	66.00	4.33	9.40	0.0054	0.011
4	10	0.0892	100	7	0.0366	100	10	0.1032	100	100	9.00	4.30	0.0763	0.087
5	8	0.1188	100	7	0.0882	100	10	0.1554	100	100	8.33	3.79	0.1208	0.083
6	10	0.1333	100	8	0.1383	100	10	0.1937	100	100	9.33	2.87	0.1551	0.083
7	7	0.1444	100	6	0.0730	100	8	0.1222	100	100	7.00	2.48	0.1132	0.091
8	7	0.1194	100	4	0.0607	100	6	0.0626	100	100	5.67	3.79	0.0809	0.083
9	7	0.1328	100	4	0.0356	100	5	0.0358	100	100	5.33	3.79	0.0681	0.139
10	4	0.0438	100	2	0.0111	100	6	0.0356	100	100	4.00	4.96	0.0302	0.042
11	2	0.0117	100	1	0.0065	100	3	0.0371	100	100	2.00	2.48	0.0184	0.041
12	2	0.0052	100	2	0.0006	100	3	0.0092	100	100	2.33	1.43	0.0050	0.011
13	2	0.0039	100	1	0.0004	100	2	0.0269	100	100	1.67	1.43	0.0104	0.036
14	2	0.0014	100	2	0.0011	100	2	0.0113	100	100	2.00	0.00	0.0046	0.014
15	2	0.0012	100	2	0.0026	100	2	0.0186	100	100	2.00	0.00	0.0075	0.024
16	2	0.0019	100	2	0.0033	100	2	0.0174	100	100	2.00	0.00	0.0075	0.021
17	2	0.0004	0	2	0.0099	100	2	0.0238	100	66.67	2.00	0.00	0.0114	0.029
18	0	0	0	5	0.0283	100	2	0.0158	100	66.67	2.33	6.25	0.0147	0.035
19	0	0	0	4	0.0743	100	2	0.0198	100	66.67	2.00	4.96	0.0314	0.095
20	0	0	0	3	0.0434	100	2	0.0047	100	66.67	1.67	3.79	0.0160	0.059
21	0	0	0	2	0.0034	100	2	0.0018	100	66.67	1.33	2.87	0.0017	0.004

A. MAIN AXIS (CONTROL PLANTS)

Leaf No. on main axis	1st Plant	2nd Plant	3rd Plant	Mean Individual leaf area (dm^2)	SE
	Individual leaf area (dm^2)	Individual leaf area (dm^2)	Individual leaf area (dm^2)		
7	0.1546	0.1321	0.1425	0.1431	0.028
8	0.1732	0.1680	0.1558	0.1657	0.022
9	0.2058	0.1999	0.1696	0.1918	0.045
10	0.2478	0.2105	0.2221	0.2268	0.047
11	0.2538	0.2139	0.2111	0.2263	0.059
12	0.2667	0.2339	0.2355	0.2454	0.056
13	0.2640	0.2256	0.2165	0.2354	0.063
14	0.2748	0.3092	0.2729	0.2856	0.051
15	0.2952	0.2653	0.2857	0.2820	0.038
16	0.2848	0.3189	0.2881	0.2973	0.047
17	0.2865	0.3230	0.2613	0.2903	0.077
18	0.2251	0.2690	0.2502	0.2481	0.055
19	0.2366	0.2465	0.2345	0.2392	0.016
20	0.2103	0.2199	0.2321	0.2208	0.027
21	0.1668	0.1743	0.1771	0.1727	0.013
22	0.0977	0.1502	0.1126	0.1202	0.067
23	0.0860	0.1196	0.0968	0.1008	0.043
24	0.0687	0.0906	0.0650	0.0748	0.034
25	0.0000	0.0543	0.0008	0.0184	0.077
26	0.0000	0.0044	0.000	0.0015	0.006

B. LATERAL BRANCHES (CONTROL PLANTS)

Branch No.	1st Plant		2nd Plant		3rd Plant		Mean Total No. of Leaves	SE	Mean Leaf Area (dm^2)	SE
	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)	Total No. of Leaves	Leaf Area (dm^2)				
3	9	0.1753	10	0.1869	9	0.1646	9.33	1.43	0.1756	0.028
4	11	0.2675	9	0.2297	11	0.2108	10.33	2.87	0.2360	0.072
5	13	0.5602	12	0.4530	14	0.4994	13.00	2.48	0.5042	0.133
6	14	0.3036	13	0.2228	14	0.2943	13.67	1.43	0.2736	0.110
7	12	0.3057	12	0.3401	9	0.2142	11.00	4.30	0.2867	0.161
8	8	0.1236	8	0.1606	9	0.1853	8.33	1.43	0.1565	0.077
9	7	0.1015	10	0.2051	7	0.1515	8.00	4.30	0.1527	0.129
10	9	0.1930	6	0.1198	8	0.1375	7.67	3.79	0.1501	0.095
11	6	0.0555	7	0.1055	7	0.1184	6.67	1.43	0.0931	0.082
12	5	0.0421	5	0.0474	6	0.0595	5.33	1.43	0.0497	0.022
13	4	0.0167	4	0.0200	4	0.0267	4.00	0.00	0.0211	0.013
14	4	0.0171	5	0.0471	4	0.0493	4.33	1.43	0.0378	0.045
15	5	0.0325	4	0.0155	4	0.0472	4.33	1.43	0.0317	0.039
16	5	0.0379	4	0.0176	4	0.0346	4.33	1.43	0.0300	0.027
17	5	0.0367	4	0.0298	4	0.0304	4.33	1.43	0.0323	0.009
18	5	0.0326	4	0.0276	4	0.0406	4.33	1.43	0.0336	0.016
19	5	0.0502	5	0.0399	5	0.0547	5.00	0.00	0.0483	0.019
20	4	0.0656	4	0.0207	5	0.0517	4.33	1.43	0.0460	0.057
21	2	0.0160	4	0.0238	4	0.0389	3.33	2.87	0.0262	0.029
22	0	0.0000	2	0.0111	2	0.0119	1.33	2.87	0.0077	0.016
23	0	0.0000	1	0.0023	0	0.0000	0.33	1.43	0.0008	0.003
24	0	0.0000	1	0.0020	0	0.0000	0.33	1.43	0.0007	0.003

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