A STUDY OF THE EFFECTS OF POWDERY MILDEW (ERYSIPHE GRAMINIS F.SP. AVENAE) ON THE GROWTH AND DEVELOPMENT OF WILD AND CULTIVATED OATS.

By

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Dedication

This thesis has been written and dedicated to my late brother, Mr. Hadj-Ahmed Sabri, and to my late grandmother, Mrs. Fatima-Zohra Sabri. All the work and time spent during my study is completely emphasized on his ambition and her goodwill, therefore shall never be regreted upon. My great love for them will be forwarded to their memories via the efforts of my hard work carried out during the research and completion of my study at the University of Glasgow.

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Summary

This thesis reports a study of the relative tolerances of infection by the powdery mildew fungus (Erysiphe graminis f.sp. avenae) in one line of wild oat (Avena fatua) and two cultivars of cultivated oats (Avena sativa), cvs Lustre and Peniarth .

The extent to which mildew developed on the three lines was assessed at various stages of growth by measuring percentage leaf area covered, and conidial production per plant and also amounts of chitin produced per unit fresh weight of leaf tissue. At all stages of plant growth, the line of wild oat supported more fungal growth than did cv. Lustre and even more so than cv. Peniarth .

The effects of infection on growth and development of the three lines was assessed by growth analysis. In all lines, infection reduced dry matter production and the development of the different plant structures particularly the leaf. The partitioning of photosynthates to different parts of the plant was also altered. Levels of infection of between about 20 and 25 % of leaf area infected reduced dry matter production in the cultivated oats but reductions were not evident in wild oat until about 30 % of the leaf area was infected. Infection had little or no effect on the pattern of translocation in wild oat whereas it did in the cultivated oats, especially in cv. Peniarth. Reductions in green leaf area were a consequence of losses due to the enhanced senescence of the lower leaves as well as to reductions in the final sizes of the upper leaves. The reduced size of the upper leaves resulted from reduced cell division and cell expansion. All the reductions occurred to a greater extent in the cultivated oats than in wild oat .

The reduction in dry matter production was reflected in reductions in yield components in all lines. Infection reduced numbers of tillers, numbers of fertile tillers and the numbers of grains per panicle. The numbers of grains per plant, hundred grain weight and total grain yield were thus reduced although the proportion of total biomass converted to grain (harvest index) was not affected in any of the three lines. Again all the reductions were greater in the cultivated oats than in wild oat .

The reduction in dry matter production was partly due to reductions in green leaf area but reductions in the rate of photosynthesis per unit of green leaf tissue

were also apparent. The photosynthetic efficiency was measured in infected and uninfected leaf tissue and also in the adjacent uninfected tissue of the infected leaf in wild oat and the cultivated oats using a Hansatech leaf electrode and by fluorescence analysis. The photosynthetic system in the infected tissue was reduced more by low levels of infection in the cultivated oats than by higher levels of infection in the wild oat. Compensatory photosynthesis in adjacent uninfected tissue was also reduced in all plant lines but not to the same extent as photosynthesis in the infected tissue. This was apparent in cv. Lustre but particularly so in wild oat. Thus infected leaves fixed less carbon than uninfected leaves but the total available to the plant and fungus in infected plants was reduced even more since more was lost through enhanced respiration. Measurements of dark respiration showed that infection increased the respiratory rate of infected and adjacent uninfected tissue in all three lines .

The reduced rates of photosynthesis could have been due to parasite induced stomatal closure since this would reduce the amount of CO_2 diffusing to the carboxylation sites within the chloroplasts of the mesophyll. The effects of infection on stomatal diffusive resistance were measured using an automatic porometer. An increase in diffusive resistance was found in the infected leaves during the light period in all lines around 5 days after inoculation, when mycelium development became significant. This increase in diffusive resistance could reduce the diffusion of CO_2 into the leaf to the mesophyll cells and in fact, a reduction in the amount of CO_2 reaching the reaction centres in the chloroplasts was evident particularly in the cultivated oats. In contrast, a decrease in diffusive resistance, almost certainly due to greater than normal stomatal opening since mycelium development was between 1 to 2 %, was recorded during the dark period in the infected leaves of cultivated oats and wild oat. This decrease was recorded earlier in the cultivated oats than in wild oat .

Some of the reduction in photosynthesis was also probably due to reductions in chlorophylls per unit area of leaf tissue. However, these reductions were not directly related to the reductions in photosynthesis since photosynthesis per milligram of chlorophyll in both infected and adjacent uninfected leaf tissue was not affected in any of the three lines. Thus the mechanisms by which photosynthesis was

impaired in infected leaves probably involved effects on photosynthetic electron transport after the PSII Q_B binding site and thus on the thylakoid proton motive force. However, the exact site of inhibition of the photosynthetic electron transport is uncertain but inhibition may be due to photoinhibition of the Q_B binding-protein or impairment of any subsequent component of the photosynthetic electron transport system including PSI .

This study showed that all aspects of growth measured were less reduced in wild oat than in the cultivated oats. Thus wild oat appears to be more tolerant of mildew infection than are the cultivated oats. However, the differences in tolerance between wild and cultivated oats were not great enough to suggest that tolerance is likely to be a useful character for inclusion in a conventional breeding programme for crop improvement .

CHAPTER 1

INTRODUCTION

Resistant cultivars have generally been very successful in limiting crop losses caused by many fungal parasites. Their use controls the genetic composition of the host population with which the parasite population must interact, and by restricting the ability of the parasite population to grow and reproduce enables the host to continue growth longer so that its yielding capacity is less impaired .

The commonest means of controlling plant parasites by resistance breeding has been through the use of major genes. In a few cases, major gene resistance has given lasting protection. For example, potato wart disease caused by Synchytrium endobioticum has been successfully controlled for many years through the use of resistant cultivars in most countries where potatoes are grown (Burton, 1989). Cultivars of tomato possessing a race-specific resistance gene against nailhead spot, a fruit, stem and leaf spot disease caused by Alternarlia tomato have been used to give good disease control (Crill, 1977). Some of the older English and French wheat varieties showed a good resistance for many years to the yellow rust caused by Puccinia striiformis until they were superseded by higher yielding varieties. For example, Little Joss, a winter wheat variety bred at Cambridge by Biffen (1907) was reported to be as resistant to yellow rust in the 1950's as when it was first bred. Innes (1974) reported that bacterial blight of cotton in the Sudan caused by Xanthomonas malvacearum was greatly reduced, and even almost eliminated by the use of resistant cultivars.

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 rust have been found to be effective for a few years during which new parasite races to which the resistance was not effective became established (Ausemus, 1943 ; Van Der Plank, 1968). Furthermore, major gene resistance to powdery mildew in barley and to leaf mould in tomatoes has not usually given lasting control of disease. Thus the causal organisms Erysiphe graminis and Fulvia fulva have been able to develop new variants with virulence genes corresponding to the resistance genes used in the host. The transient nature of major gene resistance has been in many cases due to

the tremendous variability of fungal parasites which enable them to overcome certain types of resistance, when the selection pressure in their favour is great enough (Russell, 1978).

In contrast to major gene resistance, which is generally non durable and race-specific, resistance controlled by polygenes tends to be durable and non-race-specific. Unfortunately polygenically controlled resistance rarely provides complete resistance. However, even levels of partial resistance can give useful disease control particularly when supported by other methods of control. For example, in the case of late blight of potato, partially resistant varieties are used in conjunction with fungicides. With some diseases, such as maize rust and sugar beet downy mildew, many varieties can express enough field resistance for additional control measures to be unnecessary (Russell, 1978).

In some crops adequate levels of partial resistance to important pathogens are not available, for example, in wheat and barley to the causal organism of take-all disease, Gaeumannomyces graminis, (Scott, 1981), in broad bean to the causal organisms of chocolate spot, Botrytis fabae and Botrytis cinerea (Harrison, 1988) and in potato to the causal organism of dry rot, Fusarium coeruleum (Leach and Webb, 1980). In such instances tolerance might be used in conjunction with existing levels of resistance as a breeding objective for the development of cultivars which produce an acceptable yield even when the crop is infected.

1.1. Tolerance

Reviewing existing knowledge of tolerance is hampered by the fact that the term has been loosely used from early times to describe different aspects of the host-parasite interaction. In some instances, tolerance is considered to be a form of resistance. Slow-rusting is frequently considered to be an example of tolerance (Caldwell, 1958; Mussel, 1980). The term has also often been applied to plants which are known to be infected by a wilt fungus (Bell and Presley, 1969) or a virus (Bawden,

1964) yet show few symptoms. In most cases the degree of infection was not determined and in some cases the plants may have contained limited parasite growth. Thus the term may have been applied to cases of partial resistance rather than true tolerance. Others, while recognizing that tolerance is a phenomenon distinct from resistance, classify it along with resistance for convenience (Browning *et al.*, 1977).

Some confusion between tolerance and resistance is inevitable because of a lack of knowledge of some aspects of the host-parasite interaction. The Shorter Oxford English Dictionary defines tolerance as *the action or practice of enduring or sustaining hardship or pain.* The operative words are *enduring* and *sustaining* which mean *to put up with* or *to bear with patience.* Some misuse of the term has probably come from the failure of certain definitions to encompass mechanisms of tolerance based on the relative ability of a plant to tolerate the physical presence of a developing parasite or its metabolic products (tolerance of the parasite), and the relative ability of a plant to tolerate any damage a parasite may cause (tolerance of disease). Both Schafer (1971) and Gaunt (1981) in fact refer to the possibility of tolerance of the pathogen as distinct from tolerance of disease and Schafer (1971) offers a definition of tolerance, which encompasses both, 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 or crops.*

The term tolerance should only be used in relation to properties or features of the host which enable it to endure or sustain given levels of parasite development (tolerance of the parasite), or disease (tolerance of disease), or both, and not in relation to properties or features which prevent or resist the development of the parasite or disease. Tolerance of the parasite, tolerance of disease and resistance to the parasite are likely to be determined by different sets of biochemical and physiological systems in the host, and so both may be present in any host. Thus a host should, in theory, be able to restrict the development of a parasite by resistance mechanisms but tolerate those levels of parasitic infection and disease which do occur (Clarke, 1984 and 1986) .

Tolerance of the parasite is measured by relating the amount of parasite biomass to total disease whereas tolerance of disease is measured by relating the amount

of disease produced, to the performance of the plant in terms of growth or yield (Clarke, 1986).

1.1.1. Physiological basis of tolerance

Parasites may stress or damage their host by various mechanisms during infection. Firstly, they may divert host metabolites to support their own growth and development. Secondly, they may produce metabolites which either directly or indirectly affect normal functioning of the cells and tissues of the host. Thirdly, they may disrupt the structure and integrity of host tissues. Some of this damage may be unavoidable in the sense that it is bound to occur when the parasite infects its host and completes its life cycle. Other damage may not be essential for parasite development and such damage could be considered avoidable (Clarke, 1984).

Unfortunately the potential value of tolerance cannot as yet be assessed because little is known about the nature of tolerance or the degree of tolerance that may be achieved.

1.2. The effects of infection on host growth and development

Most of the studies referred to in the following reviews have not related physiological changes in the infected plant to the extent of infection. However such studies have been included because they do indicate the kinds of effects that may occur in the infected plant as a result of the activities of the parasite.

The work of Harrison and Isaac (1969) is an early example of the application of growth analysis to plant pathological problems. They found that during the first 5 to 6 weeks of growth, potato plants infected with Verticillium albo-atrum or V. dahliae were morphologically similar to the control plants. However, during this period, the growth rate of the plants was affected as was the distribution of dry matter between the various organs. It was also found that Verticillium infection impaired the photosynthetic efficiency of the leaf area which was itself greatly reduced as the result

of defoliation and stunting effects. In contrast to uninfected plants, infected ones had lower specific leaf areas, higher leaf weight ratios and leaf area ratios and lower relative growth rates and unit leaf rates .

A number of studies have been carried out in which growth analytical techniques have been aaplied to mildew infections. Thus Last (1962) reported that although total dry weight of Erysiphe graminis f.sp. hordei infected barley (Hordeum vulgare) plants continued to increase throughout the period of investigation, control plants developed 59 % more dry matter than infected plants. Infected plants had a shorter main axis, produced fewer tillers per plant and developed a smaller leaf area per main axis and per tiller than did control plants. However, the size of the root system was reduced more than that of the shoot and, consequently, the dry weight ratio of root/total plant decreased. Infection thus affected the balance between the absorbing and the assimilating systems by decreasing the average dry weight of roots per unit leaf area. The efficiency of unit leaf area was also reduced by about 27 %. As a result of these alterations, fewer and smaller ears were produced by infected than uninfected plants. Similarly, Fric (1975) showed greater reductions in root than shoot dry weight caused by mildew infection in barley plants. He also found that infection reduced the number of tillers and their extension growth. The straw height of infected plants was 24 % less than that of control plants. The loss in total leaf area paralleled the loss in shoot dry weight and both were attributed to a decrease in the number of leaves and tillers per plant. The mean unit leaf rate of infected plants was reduced by about 27 %. Ear development and yield per plant were also affected; the total yield per plant was 32 % less in the infected plants than in the controls .

Walters and Ayres (1981) also showed that the dry weights of root and shoot, the root/shoot ratio and the relative growth rate of barley plants infected with E. graminis f.sp. hordei were significantly reduced in infected plants when compared with controls .

The effects on growth will be affected by the growth stage at which infection occurs. When mildew infection occurs late in plant development, reductions in grain size were the only effect (Brooks, 1972). Early infection, however, reduced the

number of fertile tillers, grain size and the number of grains per ear. Mildew infection of barley plants was found to result in reductions in the number of fertile tillers, the number of grains per ear and thousand grain weight, especially when plants were infected early in their development (Griffiths et al., 1975 ; Ayres and Zadoks, 1979) .

Carver and Griffiths (1981) investigated the relationship between green leaf area and grain yield of barley plants infected with E. graminis f.sp. hordei. Infection affected neither number of leaves produced nor the number of spikelet primordia differentiated, on the main axis. The only obvious effects of mildew were reductions in green leaf area, the number of fertile tillers and grain yield. The reduction in grain yield (number of ears per plant and grains per ear) was a consequence of reductions in green leaf area pre-anthesis because of the importance of this latter in grain filling. They concluded that the damage produced by barley mildew infection was essentially confined to the period pre-anthesis. Roderick and Jones (1988) investigated the effects of E. graminis f.sp. avenae on yield components of eight cultivars of oats. They found that losses in grain yield were accounted for mainly by reductions in numbers of fertile panicles and thousand grain weights. The proportion of grain yield to total biomass, or harvest index, was also reduced .

None of the research cited so far indicated that cereals, or indeed any crop, has tolerance of powdery mildew infection because in no case were comparisons made between cultivars nor were the amount of parasite or disease measured. However some studies on interactions between the common annual weed Senecio vulgaris and Erysiphe fischeri (Ben-Kalio and Clarke, 1979; Harry and Clarke, 1992) indicate that S. vulgaris possesses greater levels of tolerance of mildew infection than may be present in many crop plants. Thus even heavy levels of infection, in which between 75 % and 100 % of total leaf area were colonized, did not affect chlorophyll levels or the rate of dry matter production per unit area of leaf, and nor did they affect the distribution of photosynthates around the plant. However, total plant growth and the yield of fruits were reduced, apparently as a result of a reduction in the expansion of leaf tissue. The responses of S. vulgaris are in stark contrast to those of cultivated wheat or barley infected by E. graminis, as described earlier. A comparative study of the effects of E.

graminis on the diffusive resistance of the leaves of the weed grass Elymus repens and of barley showed that about 25 % of the leaf area of the grass required to be infected before there was a significant change in diffusive resistance compared to the controls, whereas a significant change was found with barley when less than 10 % of the leaf area was colonized (unpublished results). Both these studies clearly indicate that the wild plants are much more tolerant of powdery mildew infection than cultivated cereals are (Clarke, 1986) .

Doodson *et al.* (1964), in a detailed quantitative study of the effects of Puccinia striiformis on the growth of wheat (Triticum aestivum), reported a reduction in plant height by about 26 % and reductions in the size of leaves and number of tillers. Ear emergence and anthesis were delayed by about 14 days. In fully infected plants, the number of grains per ear was reduced by up to 42 % and the mean dry weight per grain by about 34 %. There was also a very striking reduction in root development when compared with other parts of the infected plant. The mean root dry weight of fully infected plants was reduced by about 78 %. This was probably related to the reduction in the amount of translocates moving to the roots from the aerial parts in infected plants, and also to the fact that the uninfected plants produced two to three times as many tillers as infected plants, each of which produced its own root system. Owera *et al.*, (1981) showed that P. hordei infection of the first leaf of barley resulted in plants with about 20 % less dry weight than controls 16 days after inoculation. The overall decrease in weight was paralleled by an increase in weight of the infected leaf. The amount of translocates 16 days after inoculation was reduced by about 63 % as a result of reduced photosynthesis in, and export from, the infected leaf. The infection did not alter the root/shoot ratio. Whether this was due to the small amount of fungal growth (a small sink) or to other effects during the course of infection was not investigated .

Mather and Hansing (1960) found that infection of wheat plants with loose smut, Ustilago tritici, caused total dry weight to be reduced by 33 %, root dry weight by 32 %, main axis height by 13 % and tiller number by 11 % in infected plants. Billett and Burnett (1978) examined the effects of U. maydis infection on the growth of maize (Zea mays) plants. They found that infection resulted in an increase in main axis dry

weight from 3 days after inoculation until early fungus sporulation, and then it decreased. The initial increase was paralleled by a decrease in dry weight of other developing plant parts. As infection progressed, both plant dry weight and leaf area were reduced. Leaf expansion was delayed and final leaf size reduced. Infected areas of the leaf blades had became chlorotic 3 to 5 days after inoculation, having lost 60 % of their chlorophyll. The roots suffered a proportionately larger reduction in dry weight than the shoot.

In all the examples cited above, the root / shoot dry weight ratio of infected plants was generally decreased as a result of infection or was unaffected. In some host-pathogen combinations, however, this ratio has been found to increase. For example, infection of cabbage plants with Plasmodiophora brassica was found to cause the root / shoot ratio to increase as the result of the diversion of metabolites to the developing clubroot gall at the expense of shoot development. However, as with the other cases reported earlier, the decrease in total leaf area closely paralleled that of total plant dry weight. From the 35th day after inoculation onwards, infected plants had smaller and thinner leaves than those of control plants. Leaf expansion was also delayed, one new leaf unfolding every 9 days on infected plants against one every 4 days on control plants. The dry weight of root per unit area of leaf was decreased and unit leaf rate was consistently lowered, on the average by about 15 % of the control value (Macfarlane and Last, 1959).

It appears from all these studies that the infected regions of the plant increase in dry weight at the expense of other plant parts, and in cereals infected with powdery mildews and rusts especially, at the expense of the roots and tillers. The extent to which fungal material accounted for the localised increase in dry weight was usually undetermined.

For most of the examples cited and for others, the various investigators have shown the effects of parasitic activities on the host growth and development. They have also studied the consequences of such activities on the normal functioning of the cells and tissues of the host (see section 1.3. in this chapter). Unfortunately, neither these nor any of the other studies with crop plants have measured the amount of fungal

growth on the host, and so the extent to which different levels of infection affect the host response is not known .

1.3. Impairment of plant functions

In this section the impairment of plant functions is discussed in broad terms with relevant examples .

1.3.1. Effects on carbon fixation

Infection of plant tissues by biotrophic organisms generally causes a reduction in the rate of photosynthesis .

Edwards (1970) found a biphasic inhibition of photosynthesis in barley infected with E. graminis f.sp. hordei. The first phase of inhibition occurred prior to or during the early stages of sporulation by the fungus, with the second phase occurring around 6 days after inoculation when fungus sporulation on the tissues reached a maximum. Ayres (1976) showed that in pea (Pisum sativum) infected with E. pisi, photosynthesis was reduced within 24 h. of inoculation and had decreased to less than one third of that in healthy plants by the seventh day after inoculation. Sugar beet (Beta vulgaris) leaves infected with E. polygoni showed declining rates of photosynthesis relative to controls with reductions of 35, 70 and 75% being observed at 9, 16 and 22 days after inoculation respectively. The quantum efficiency of photosynthesis was also reduced by 17 and 22 % at 14 and 18 days after inoculation, respectively (Gordon and Duniway, 1982a) .

Similar reductions have been observed with rust infections. Mitchell (1979) showed that when the first leaf of wheat was infected with P. graminis f.sp. tritici, rates of CO_2 assimilation and chlorophyll content declined progressively from 3 days after inoculation. Barley plants infected with brown rust, P. hordei, at the first leaf stage showed no reduction in photosynthesis until 9 days after inoculation after which photosynthesis declined to about half the control level (Owera *et al.*, 1981) .

During the early stages of infection, the rate of photosynthesis often rises before the subsequent fall. Scott and Smillie (1966) showed that infection of leaves of barley with E. graminis f.sp. hordei resulted in a slight increase in photosynthesis 48 h. after inoculation, but subsequently there was a progressive decrease in the leaves in comparison with the controls. Similarly Allen (1942) reported stimulation of photosynthesis soon after infection of wheat leaves with E. graminis f.sp. tritici . Hewitt and Ayres (1975) showed in oak (Quercus robur) seedlings infected with powdery mildew (Microsphaera alphitoides) an initial increase in photosynthesis 24 h. after inoculation followed by a rapid decline on the second day after inoculation .

Similar increases have been observed in wheat (Triticum aestivum) leaves infected with Puccinia graminis f.sp. tritici and bean (Phaseolus vulgaris) leaves infected with Uromyces phaseoli (Livne, 1964). Subsequently, photosynthetic activity and carbohydrate accumulation in infected leaves declined to one-third and one-half of that of control leaves respectively .

Although infection reduces total photosynthesis in the whole leaf, rates of photosynthesis can remain near the values in uninfected leaves or even be increased in certain regions of infected leaves. For example, green-islands are a characteristic feature of biotrophic infections and become apparent during the later stages of infection, when areas in and around fungal pustules remain green, while the remainder of the leaf senesces. Both increases and decreases in photosynthesis have been reported in green-islands in rust and mildew infected leaves. The rate of photosynthesis was examined in whole leaves of barley infected with P. hordei and within pustule areas, from presporulation to green-island formation. The rate of photosynthesis and the quantum efficiency of O_2 evolution declined when measurements were made on a whole leaf basis as the infection progressed. However, the rate of photosynthesis per unit chlorophyll was increased in rusted leaves. When measurements were made within pustule areas, photosynthesis per unit chlorophyll was also increased in comparison to that in similar regions on uninfected plants (Scholes and Farrar, 1986). In contrast, photosynthesis in green-islands and surrounding senescing tissue of barley leaves infected with E. graminis f.sp. hordei was reduced by 14 % (when expressed on a unit

area basis) or 32 % (when expressed on a chlorophyll basis). The quantum efficiency of photosynthesis and the chlorophyll a : b ratio were also reduced. However the senescing tissue was unable to photosynthesize due to a considerable reduction in total chlorophyll (Coghlan and Walters, 1992). Roberts and Walters (1988) also found that rates of photosynthesis, expressed on both a chlorophyll and a leaf area basis, were considerably reduced in pustule areas on leek (Allium porrum) leaves infected with P. allii. Similarly, Scholes and Farrar (1985) showed a reduction in photosynthesis per unit area and per unit chlorophyll within pustule areas of Uromyces muscari infection of bluebell (Hyacinthoides non-scripta) leaves .

Photosynthesis has been reported to be stimulated in the healthy leaves of infected plants. For example Ayres (1981) showed a stimulation of photosynthesis in healthy fourth leaves of pea when the lower three leaves of the same plant were infected with E. pisi. Wang (1960), using wheat leaves infected with P. graminis f.sp. tritici reported higher CO_2 uptake by healthy regions of inoculated leaves than by infected areas. Livne (1964) found that heavy infections of primary unifoliate bean leaves with U. phaseoli stimulated rates of $^{14}CO_2$ fixation in uninfected trifoliate leaves of the same plant by more than 50 %, while So and Thrower (1976) found that very light rust infections of second leaves of Vigna sesquipedalis slightly stimulated $^{14}CO_2$ fixation by uninfected third leaves. Thus it appears that the pathogen can affect photosynthetic CO_2 fixation in uninfected tissues of infected plants. Photosynthesis may be stimulated by the increased removal of photoassimilates from their site of synthesis as the result of the establishment of a sink for nutrient accumulation by the fungus at its infection site .

Photosynthesis is in part a diffusion process where the flux of CO_2 into a leaf is driven by the concentration gradient of the gas between the external air and the carboxylation sites. Models of diffusion pathway have been applied to the analysis of photosynthesis in several host-pathogen systems (see chapter 2, section 2.8.2.2.). The mechanisms by which photosynthesis may be inhibited are numerous. Reductions in photosynthesis have been attributed to altered rates of CO_2 diffusion into the leaf through stomata (Majernik, 1971; Ayres, 1976; Gordon and Duniway, 1982b). Reductions may also be due to direct effects of the pathogen at the biochemical or

chloroplast level. For example, reductions might be due to inhibition of the light reactions of photosynthesis (Montalbini and Buchanan, 1974; Buchanan *et al.*, 1981), or destruction of chloroplasts within developing lesions (Ahmad *et al.*, 1983) .

The reduction in photosynthesis in pea leaves infected with E. pisi from the third day after inoculation was partly attributed to an increase in the stomatal resistance to CO_2 diffusion into the leaf (Ayres, 1976). Small increases in stomatal resistance have also been reported in mildewed barley 3 days after inoculation (Ayres, 1979) and mildewed oak 6 days after inoculation (Hewitt and Ayres, 1975). In contrast, a small decrease in stomatal resistance was observed during the early stages of infection of bean leaves by Uromyces appendiculatus (Sempio *et al.*, 1966). In more recent work, barley plants (Hordeum distichum) infected with P. hordei showed a reduction in photosynthesis and an increase in the internal CO_2 concentration which was due to increased dark respiration and photorespiration (Owera *et al.*, 1981). Analysis of photosynthesis as a diffusion process showed an increased resistance to CO_2 flux due to increased mesophyll resistance. Gordon and Duniway (1982b) also showed a reduction of 50 % in stomatal conductance and an increase in mesophyll resistance in sugar beet leaves infected with E. polygoni. Increases in mesophyll resistance were correlated with reductions in the activity and concentration of ribulose-1,5-bisphosphate (RuBP) carboxylase, and also a reduction in total soluble protein .

Much of the earlier and even more recent work involved possible relationships between chlorophyll loss due to infection and the subsequent reduction in photosynthesis. A characteristic symptom of mildew and rust infections is an increased chlorosis of infected leaves due to chlorophyll breakdown. Ahmad *et al.* (1983) concluded that the reduction in photosynthesis in barley leaves infected with P. hordei was principally due to a decrease in the number of functional chloroplasts. In more recent work, Scholes and Farrar (1985) have shown that U. muscari rust infection of bluebell (Hyacinthoides non-scripta) resulted in reductions in chloroplast numbers per unit area, chloroplast volume, chlorophyll concentration and the ratio of chlorophyll a:b, within individual pustules. This work showed that, in rusted bluebell, chlorophyll is lost from individual chloroplasts. Electron micrographs showed that in rust and

powdery mildew infections, the grana and intergranal lamellae of chloroplasts become progressively more disorganised as infection progressed (Coffey *et al.*, 1972; Heath, 1974).

In some cases, reductions in chlorophyll concentrations are not closely correlated with changes in rates of photosynthesis. Allen (1942) showed that in wheat infected with E. graminis f.sp. tritici, photosynthesis per unit chlorophyll declined below the control levels one week after inoculation. These results were confirmed in a latter study on barley by Scott and Smillie (1963). However, Hewitt (1976) showed that in oak leaves infected with M. alphitoides, photosynthesis declined before total chlorophyll levels were reduced by infection, and noted that the chlorophyll a:b ratio decreased only at the latter stages of infection. In contrast, Waygood *et al.* (1974) found that photosynthesis per milligram of chlorophyll was actually 50 % higher in wheat leaves infected with P. graminis f.sp. tritici than in healthy leaves. Recently, Owera *et al.*, (1981) showed that although chlorophyll levels decreased in barley leaves infected with P. hordei, photosynthesis per unit of chlorophyll increased slightly.

The mechanisms responsible for the increase in photosynthesis which have been reported within green-islands are not known. However, cytokinins and/or polyamines have been implicated in the biosynthetic activity retained in the green-islands (Scholes and Farrar, 1986; Coghlan and Walters, 1992).

Infection has been shown to have little or no effect on photophosphorylation in chloroplasts. For example, chloroplasts isolated from oat leaves infected with P. coronata (Wynn, 1963) and from barley leaves infected P. hordei (Ahmad *et al.*, 1983) showed no reduction in their capacity for photophosphorylation. Although it has been found that photophosphorylation may not be affected, there is evidence from several host-parasite systems that inhibition of the non-cyclic electron transport chain may occur. Chloroplasts isolated from E. polygoni infected sugar beet leaves (Beta vulgaris) (Magyarosy *et al.*, 1976) and U. fabae infected broad bean (Vicia faba) (Montalbini and Buchanan, 1974) showed reductions in the rate of electron transport and in the accompanying ATP formation in non-cyclic photophosphorylation (reduced electron flow from water to NADP). They also showed

that these reductions were associated with a decrease in the rate of photosynthetic CO_2 assimilation and alterations in chloroplast ultrastructure and other components of the photosynthetic apparatus. Magyarosy and Malkin (1978) have shown that mildew infection of sugar beet results in a substantial (33 %) reduction in the cytochrome content of the electron transport chain. Buchanan *et al.* (1981) found that non-cyclic electron transport was inhibited by up to 45 % in chloroplasts isolated from barley leaves infected with P. hordei . It seems likely that the inhibition of non-cyclic photophosphorylation is due to changes in certain components of the non-cyclic electron transport chain such as the cytochromes (Scholes and Farrar, 1986) .

Powdery mildew infection of barley and rust infection of wheat have also been shown to result in a substantial decrease in the activity and amount of ribulose-1,5-bisphosphate carboxylase (RuBP) which is the biochemical component of mesophyll resistance and which acts as the active CO_2-fixing enzyme in plants (Walters and Ayres, 1984). However, Gordon and Duniway (1982b) have suggested that RuBP carboxylase in sugar beet leaves infected with E. polygoni may not be responsible for limiting the flux of carbon through the reductive pentose phosphate pathway since the activities of other enzymes in that pathway are also reduced by infection. Recent work has shown that stimulation of photosynthesis in uninfected leaves of mildewed barley was due, in part, to an increase in the amount and activity of RuBP carboxylase (Walters and Ayres, 1984). However, both the stimulation in photosynthesis and the increase in the amount and activity of RuBP carboxylase were transient, lasting no more than 5 days after inoculation .

Changes in nucleic acid metabolism also occur. Barley mildew infection has been shown to result in a reduction in ribosomal RNA in chloroplasts (Dyer and Scott, 1972) and more recently, Higgins et al. (1985) have shown that infection of barley by E. graminis f.sp. hordei results in a reduction in the amounts of mRNA coding for the two sub-units of RuBP carboxylase, a consequence of which would be a loss of chloroplast proteins and a reduction in the rate of photosynthesis .

In most of the host/pathogen systems discussed, there was a decrease in photosynthesis as a result of infection. Reductions in photosynthesis appeared to be

related to the following : increased stomatal resistance, increases in mesophyll resistance due mainly to loss of chlorophyll, specific alterations in components of the non-cyclic electron transport chain such as cytochromes and reductions in RuBPcase protein content. Observations of leaf symptoms have led several investigators to suggest that the pathogen produced toxins some of which are known to act directly on photosynthesis, stomatal function, water retention and transport in leaves (Habeshaw, 1984) .

1.3.2. Effects on carbon losses through respiration

1.3.2.1. Photorespiration

Changes in photorespiration have not been widely investigated in diseased plants. In some host-parasite associations, the rate of photorespiration has been shown to decrease as a result of infection. Reduced photorespiration has been reported to occur in barley infected with E. graminis f.sp. hordei (Ayres, 1979 ; Walters and Ayres, 1984), in oak infected with M. alphitoides (Hewitt and Ayres, 1975) and in flax leaves infected with the rust Melampsora lini (Kakkar, 1966). Hewitt and Ayres (1975) found that in oak, the initial stimulation of photosynthesis following infection with M. alphitoides was attributed to the fact that a decline in photorespiration began earlier than that in photosynthesis. However, an initial increase in rates of photorespiration has been observed in pea leaves infected with E. pisi (Ayres, 1976) and in barley leaves infected with P. hordei (Owera *et al.*, 1981), before the subsequent decline. Furthermore, Mitchell (1979) showed no differences in rates of photorespiration between healthy and infected first leaves of wheat during infection by P. graminis f.sp. tritici .

Where reductions in photorespiration in infected plants were recorded, they appear to be due to reductions in the activities of associated enzymes. In wheat infected with P. graminis f.sp. tritici and oak leaves infected with M. alphitoides, glycolate oxidase activity was reduced by infection. In addition to a decrease in glycolate oxidase activity, Walters and Ayres (1984) found a decrease in the activities

of glycolate reductase and RuBP oxygenase upon infection of barley with E. graminis f.sp. hordei. The mechanism responsible for the observed increase in photorespiration in barley infected with brown rust is not known (Owera *et al.*, 1981). Reduced photorespiration observed in some host/pathogen combinations appeared to be the result of reduced activity of the enzymes involved (Walters, 1985).

1.3.2.2. Dark respiration

Another common observation of the effects of a parasite on its host is that dark respiration is affected. Increased dark respiration in infected plants means that, as infection progresses, an increasing proportion of assimilate is lost via respiratory processes. This is not unexpected since both the parasite and the host will have a high demand for energy, the fungus for growth and maintenance and the host additionally for defence, and respiration would thus be increased to provide both the energy and carbon skeletons needed for the various biosynthetic processes involved (Farrar and Rayns, 1987).

One of the earliest reports of respiratory changes in diseased plants is that of Allen and Goddard (1938) who showed that dark respiration was increased in powdery mildew infected wheat. They found that this increase was maintained when the fungus mycelium was removed from the surface of the infected leaf. The increase occurred in the mesophyll and so was clearly of host origin. These results were confirmed by Millerd and Scott (1956) who found that mildewed barley leaves respired at almost the same rate as leaves with the fungus removed. In the case of rust and other infections where the fungus develops endogenously there is much uncertainty about the scale of the contribution of the fungus to the increased respiration, due to the difficulties involved in attempting to separate host and fungus. It is generally held that the fungal contribution to increased respiration is small (Ayres, 1979). However, Owera et al. (1981) suggested that all the increased dark respiration in barley infected with P. hordei could be attributed to the fungus.

Increased respiration has been reported in barley leaves infected with E. graminis f.sp. hordei (Scott and Smillie, 1966 ; Farrar and Rayns, 1987), in wheat

leaves infected with P. graminis f.sp. tritici (Daly *et al.*, 1961 ; Mitchell, 1979), in bean leaves infected with U. phaseoli (Livne, 1964) and in oak infected with M. alphitoides (Hewitt and Ayres, 1975). Allen (1942) emphasized that in biotrophic pathogens, there is often a considerable period when the respiration rate of an infected leaf is high and this period corresponds with the period when the assimilation rate is low. The result is the depletion of the reserves of assimilates available to the host.

Few studies correlated changes in dark respiration to changes in photosynthesis. Roberts and Walters (1988) found an increase in dark respiration within pustule areas on leek (Allium porrum) leaves infected with P. allii where photosynthesis was reduced. The overall rate of dark respiration in whole barley leaves infected with P. hordei was found to be at least twice that of the controls at the time of sporulation, while the rate of photosynthesis declined. However, the rate of dark respiration within pustule areas was four times higher than controls at the time of sporulation, while the rate of photosynthesis increased (Scholes and Farrar, 1986). These studies contrast with recent studies on oat plants infected with E. graminis f.sp. avenae where Haigh *et al.* (1991) found that although infection reduced rates of photosynthesis, it had little effect on rates of dark respiration. Little change was also found in dark respiration in green-islands on barley leaves infected with E. graminis f.sp. hordei where photosynthesis was reduced (Coghlan and Walters, 1992).

Allen and Goddard (1938) suggested that the rise in respiration in mildewed wheat was due to diffusible substances produced by the fungus which moved into the mesophyll. Bushnell and Allen (1962) found an accumulation of metabolites, often in a mobile form, in the uninvaded cells adjacent to the periphery of the developing pustules and in the tissues immediately below. This observation suggests that the mildew parasite produces toxic substances which diffuse into the underlying cells and there initiate the metabolic changes which result in the increased respiration.

Allen (1953) suggested that the enhancement could be caused by a toxin which uncouples respiration from energy-requiring processes by acting on oxidative phosphorylation. If the uncoupling hypothesis is true, the synthesis of ATP would be reduced or prevented in the electron transport system, and the concentration of ADP

would increase and thus the ratio ADP/ATP would be increased. Poszar and Kiraly (1958) found that the ADP/ATP ratio was higher in wheat leaves infected with P. graminis f.sp. tritici. The uncoupling hypothesis has also been suggested as an explanation for increased O_2 uptake in oat tissue treated with victorin, the host-specific toxin of Helminthosporium victoriae (Krupka *et al.*, 1959). The uncoupling hypothesis has been investigated by several other authors who suggested that the effects of the toxin on mitochondria is secondary rather than primary .

The most likely cause of the increased respiration caused by biotrophic fungi is the enhanced operation of the oxidative pentose phosphate pathway (Daly, 1976). The pentose phosphate pathway appears to be located in the cytosol and is limited by the availability of $NADP^+$. It is possible that the increased activity of this pathway in rust and mildew infected tissues may be due to the release of $NADP^+$ into the cytosol after chloroplast breakdown. The rise in dark respiration that occurs in the latter stages of infection of barley with E. graminis f.sp. hordei may be a direct response to the change in the $NADP^+$/NADPH balance (Scott and Smillie, 1966; Dyer and Scott, 1972). That an increase in the pentose phosphate pathway is involved is supported by the finding that increased respiration appears to be associated with an increase in the activity of enzymes of this pathway in infected tissues. Scott (1965) found a 2 to 3-fold increase in the activity of such enzymes in mildew-infected barley tissues. Chakravorty and Scott (1982) suggested that the reductions in photosynthesis in rusted and mildewed plants lead to the release of control mechanisms on the two dehydrogenases resulting in increased activity of the pathway. Increased dark respiration could also be due to an activation of existing pathways, such as the citric acid cycle in wheat leaves infected with P. graminis f.sp. tritici (Kiraly and Farkas, 1957) and the electron transport chain in radish (Raphanus sativus) infected with Albugo candida (Williams and Pound, 1964). Farrar and Rayns (1987) showed that infection of barley leaves with E. graminis f.sp. hordei results in an increase in dark respiration by 80 % during fungus sporulation. About half of the increase was due to increased electron flow through the cytochrome chain and half through an alternative pathway. The mechanism of the latter was not obvious .

In other host/pathogen systems, increased host respiration was attributed to the stimulation of existing pathways. Whatever the cause of the increased respiratory activity, it results in the loss of photosynthate that could otherwise be utilized for plant growth. These results support the view that enhanced oxygen uptake is not directly a part of pathogenesis but rather a result of increased biosynthesis by which the host supplies the fungus with nutrients for its growth and sporulation. Also, biotrophic fungi create a metabolic sink which at the expense of host energy draws photoassimilates and other nutrients from surrounding tissues to the sites of infection (Shaw and Samborski, 1956).

1.3.3. Effects on translocation

A common effect of attack by biotrophic pathogens on plants is that the translocation and distribution pattern of assimilates throughout the plant is affected (Crowdy and Manners, 1971; Manners and Myers, 1973). The reductions in photosynthesis and the increases in dark respiration alter the amount of photosynthate supply to the various sinks in the infected and uninfected organs of the plant. Thus it is not suprising that movement of assimilates around the plant is affected by infection. One of the earliest attempts to quantify the flow of carbon in infected plants was that of Yarwood and Childs (1938) who measured an increase in the dry matter of bean leaves infected with U. phaseoli concomitant with overall reductions in dry weight for the entire plant. Subsequently, Yarwood and Jacobsen (1955) demonstrated the translocation of assimilates by means of radioactive tracers such as $^{14}CO_2$.

In general, infection by fungi results in a reduction in the export of assimilates from infected leaves and the increased import into infected tissues. For example, Fric (1975) found that in the primary leaf of barley infected with E. graminis f.sp. hordei, export of labelled assimilate was less, although export from the uninfected second leaf was greater 5 days after inoculation. The absolute amount of labelled assimilates reaching roots in the 24 h. after feeding was reduced by about 27 %, while the amount remaining in shoots was reduced by 20 %. Plants of three cultivars of wheat

infected with E. graminis f.sp. tritici showed a reduction in the percentage of labelled assimilates exported to roots when fed with $^{14}CO_2$ at third, fifth or flag leaf stages (Lupton and Sutherland, 1973). Continued assimilate importation into infected leaves was also demonstrated by Hewitt and Ayres (1976), working on oak seedlings infected with M. alphitoides .

Walters and Ayres (1982) concluded that reductions in the growth of primary roots of barley infected with E. graminis f.sp. hordei was due to a reduction in the specific activity of different fractions within roots (soluble, storage and structural), with reductions being greatest in root tips. These reductions in the root carbohydrate fractions were paralleled by a progressive reduction in the mitotic index of root tips. Walters and Ayres (1981) showed that the length and branching of main seminal and nodal root axes were reduced in mildew infected barley plants and this was associated with reduced solute accumulation which determines both cell division and cell expansion involved in root growth. These results were similar to those obtained by Minarcic and Pauleck (1975) .

Doodson *et al.* (1965) investigated the effects of infection by P. striiformis on the third leaf of wheat on the assimilation and translocation patterns of that leaf. They found that, 14 days after inoculation, only negligible amounts (0.4 %) of assimilates were leaving the infected leaf compared with 21 % in the corresponding leaf of control plants. However the pattern of distribution of translocates as between the various plant organs was relatively unaffected, the only effect being that the proportion moving to the roots and tillers was drastically reduced and this was correlated with greatly reduced dry weights of these organs in infected plants. The situation in which only one leaf was affected was somewhat artificial, although convenient for experimental purposes. The more usual situation, where the whole plant is affected, was investigated in later experiments by Siddiqui and Manners (1971) who found that in plants with all leaves infected, 14 days after inoculation, 31 % of assimilate was translocated out of infected leaves in comparison with 0.4 % in Doodson's experiment. Thus, they showed that the effects on the translocation pattern were greater when only

one leaf was infected, and that the proportion of ^{14}C assimilation moving to the infected from uninfected leaves was increased at the expense of the roots .

Livne and Daly (1966) found that in bean leaves infected by the rust U. appendiculatus, the amount of exported carbon dropped from 50 % to 2 %, whereas import from the next youngest leaf increased from < 1 % to 32 %. The diversion of assimilates to the rust was at the expense of the roots and newly emerging leaves. These results were similar to those obtained by Thrower and Thrower (1966) working on the translocation pattern in broad bean infected with U. fabae .

Host growth and translocation patterns were also examined in loose smut infections. Gaunt and Manners (1971) studied the effects of U. nuda infection on the growth of wheat plants. A growth analysis experiment showed that smut infection had considerable effects on host growth in many ways comparable to those produced by rust infection. Thus, they found a reduction of about 40 % in root growth, at the expense of ear formation, at an early stage of infection, 40 days after inoculation, followed by a stimulation of host stem growth during fungal sporulation, 50 days after inoculation. Subsequently, at 60 days, the infected stem ceased to grow but the roots developed further .

Coffey *et al.* (1970) investigated the translocation pattern of labelled ^{14}C in tomato plants infected with the early blight fungus, Alternaria solani and found a significant reduction in the amount of assimilates exported from the infected leaf but only at the early stages of infection. At latter stages of infection, where there was chlorosis as well as increased necrosis, export from infected leaves increased and the distribution of translocated assimilates was altered. They also found that the labelled carbon in the stem of infected plants was less than half that of the controls but the fraction in the roots was greater than that in uninfected plants. This later investigation shows that necrotrophs can alter the translocation and distribution patterns in their hosts in ways very similar to the effects produced by biotrophs .

Most studies showed a pronounced initial accumulation of carbohydrate at the sites of infection followed, in some cases, by a subsequent decline. Carbohydrate levels in primary leaves of bean infected with U. phaseoli depended on the development

and intensity of infection. For example at 5 days after inoculation, with a high inoculum giving around 87 pustules.cm-^{2}, sucrose levels in infected leaf tissue rose to 33 % of those in healthy tissue. In contrast, with low levels of infection giving around 50 pustules.cm-^{2}, starch levels in infected leaves rose to 200 % of those of healthy tissue. However, with the advent of sporulation, sugar levels declined well below those in healthy tissues (Inman, 1962). Increase in sugars in infected tissues has been reported before by other authors (Allen, 1942 ; Shaw *et al.*, 1956). Some of the distortions of leaves of Urtica parviflora characteristic of infection with Puccinia caricina, or the swollen shoots of Zizania caduciflora produced in response to the smut Ustilago esculenta infection (Whipps and Lewis, 1981) could be due to particularly high levels of accumulation of carbohydrates .

Conversely, several investigators have reported a decrease in the sugar content of rust-infected tissues. In wheat infected with P. graminis, total sugar content and especially the sucrose fraction, decreased slightly in the resistant host and greatly in the susceptible one (Krog et al., 1961). Murphy (1936) similarly found a decrease in soluble sugars in oats infected with crown rust (Puccinia coronata) .

The precise mechanism whereby infection by obligate biotrophs alters the pattern of assimilate translocation in plants is still not known. However, the phenomenon of tranlocation and distribution can be explained by the high metabolic activity of the attacked tissue and of the parasite which represent an active "sink" for the attraction of nutrients from other plant parts (Thrower, 1965). The factors controlling normal translocation are not well understood, but there is much evidence to implicate plant hormones in the control of both photosynthesis and translocation (Walters, 1985) .

1.3.4. Effects on transpiration

The rate of water vapour loss from leaves depends on the water concentration gradients within the leaf and resistances to water vapour diffusion between evaporating surfaces and the ambient air. The cuticle and stomata offer the chief resistance to water vapour diffusion from leaves. Whereas there is a relative

abundance of data concerning the effects of biotrophic pathogens on resistances to water flow through leaves, very little information is available on resistance to water flow across the root and through the stem to the leaf.

The influence of various rust and powdery mildew infections on transpiration rate has been studied. The early stages of infection of barley by E. graminis f.sp. hordei significantly reduces the transpiration rates of the leaves in the light, whereas the later stages of infection involve increases in transpiration rates (Priehradny, 1971). The reduction in transpiration is associated with a reduction in stomatal opening in the light as observed from impressions of the leaf surface (Majernik, 1965). In a later study, Majernik (1971) employed a viscous flow porometer and found that mildew inhibited both the opening and closure of stomata in barley; with stomatal aperture being lower in the light and greater in the dark relative to the controls by the fifth day after inoculation. Stomatal opening and transpiration rate were also inhibited in wheat plants within 6 h. of inoculation with E. graminis f.sp. tritici and this was attributed to a volatile product of fungal metabolism (Martin *et al.*, 1975).

In contrast, Ayres (1976) showed that in the first 48 h. after inoculation of pea leaves with E. pisi, stomatal opening in the light was greater in infected than in healthy leaves but on the third and later days after inoculation, it progressively decreased. The stomata failed to close in the dark and by the seventh day after inoculation were immobilized in a partly open position. Transpiration in infected leaves followed closely the pattern of stomatal opening, decreasing in the light and increasing in the dark. The net result was a slight reduction in total transpiration over a 24 h. period, from the third day onwards. The reduced transpiration in the light shown by infected leaves was probably the result of not only an increase in stomatal resistance to the diffusion of water vapour but also of an increase in boundary layer resistance caused by the presence of a cover of fungus mycelium over the leaf surface, including stomatal pores, because transpiration increased when the mycelium was removed but stomatal opening showed no effect (Ayres, 1976).

Several studies on powdery mildew of barley have attempted to distinguish stomatal and cuticular transpiration rates by measuring the loss in fresh

weight of leaves as a function of time after excision in air. For example, by this analysis, Paulech *et al.* (1970) found that all stages of powdery mildew infection in barley decreased stomatal transpiration but that in the later stages cuticular transpiration increased markedly. However measurements of decrease in fresh weight with time are difficult to interpret because of the lack of information on leaf water status, the contribution of external resistance and changes in leaf temperature (Ayres, 1976). Ayres (1976) showed that, in barley infected with powdery mildew, transpiration from the fungus was less than the reduction in transpiration from the leaf which was caused when development of the fungus mycelium increased the boundary layer resistance of the leaf.

Ayres and Zadoks (1979) showed that water consumption per plant of barley infected with powdery mildew was reduced because the transpiring leaf area was reduced and, also, because stomatal opening in the light was inhibited. Furthermore, incomplete stomatal closure in the dark, lead to increased rates of transpiration (some part of the increased water loss in the dark also occurred through fungal mycelium) and lowered water potentials in infected plants. The lowering of cell water potential inhibited growth by inhibiting cell expansion, the latter depending on the attainment of high turgor in cells during the dark period (Boyer, 1976).

The transpirational behaviour of bean plants infected with U. phaseoli showed during the early stages of infection a decrease in transpiration rate with a partial inhibition of stomatal opening in the light. After fungal sporulation, the epidermis and cuticle of infected leaves were damaged and transpiration rate per unit leaf area increased. The lack of stomatal control over leaf water loss through the damaged cuticle led to very severe wilting (Duniway and Durbin, 1971). Paul and Ayres (1984) have shown that, after sporulation, groundsel plants infected with the rust, Puccinia lagenophorae, transpired more than did uninfected controls, and, in addition rust infection resulted in an increased transpiration ratio (mg water lost per mg of CO_2 fixed), indicating a much decreased water use efficiency.

Records on fungal pathogens causing increased stomatal opening in the light are rare. Infection of potato by the blight fungus, Phytophthora infestans, induced

stomata to open more widely than normal in the light and to fail to close in the dark, resulting in increased transpiration (Farrell et al., 1969). Since stomatal opening depends upon the guard cells having a higher turgor pressure than the epidermal cells, they attributed the increase in stomatal opening to an increased osmotic value in affected guard cells. Similar results were obtained in barley leaves infected with leaf blotch, Rhynchosporium secalis (Ayres, 1972). Cruickshank and Rider (1961) have shown that infection of tobacco leaves with the downy mildew fungus, Peronospora tabacina, results in a small increase in transpiration in the light, and a doubling of transpiration in the dark in the presporulation phase of the fungus. Although no stomatal measurements were made, it appeared that the pathogen produced an effect on stomata very similar to that induced by P. infestans. Upon the onset of the post-sporulation phase, the transpiration rate decreased which was probably due to blockage of stomatal pores by sporangiophores (Cruickshank and Rider, 1961) .

1.3.5. Effects on growth regulatory substances

Parasite metabolites such as toxins, growth regulatory substances and enzymes will be essential to the development of a parasitic relationship if their production is essential for the establishment of that relationship. These metabolites could be responsible for the changes in metabolism, in transport systems and in growth and development of the host which occur during infection .

Some parasites, by producing growth regulators or by stimulating host tissues induce growth abnormalities such as galls and tumours. They cause damage in part by disrupting the normal arrangements of the cells so that the tissues no longer function normally. For example, the tissues of crown gall induced in a wide range of host plants by Agrobacterium tumefaciens, of root galls in brassicas induced by Plasmodiophora brassicae, of maize smut galls induced by Ustilago maydis and of cocoa swollen shoot induced by a virus (CSSV) have been shown to contain abnormally high amounts of growth regulatory substances such as auxins and cytokinins .

Daly and Inman (1958) found that infection of safflower hypocotyls by the fungus Puccinia carthami resulted in a fourteen-fold increase in auxin level compared to uninfected controls. Pozsar and Kiraly (1966) found that extracts from bean leaves infected with U. phaseoli showed increased cytokinin activity when compared to uninfected tissues. In rice, infection by Gibberella fujikuroi (Fusarium moniliforme) causes an elongation of the internodes of the stem, a condition originally described as the bakanae or foolish seedling disease. This increased growth has been attributed to high amounts of gibberellins (Yabuta and Yayashi, 1939). In these examples, the abnormal growth utilises nutrients which would otherwise have been available for the development of normal structures and they are therefore acting as metabolic sinks .

Despite the abundance of literature on plant growth regulators, the precise role of these compounds in plant-pathogen interactions is still unclear. This is perhaps not surprising since, in many cases, the physiological effects of individual growth regulators interact, and normal plant growth and development is determined by the integrated action of several types of hormone, rather than as the result of changes in concentrations of a single compound (Walters, 1985) .

1.3.6. Conclusions

In this section we have highlighted some of the ways, in addition to its feeding activities, that a parasite can affect the physiological and biochemical processes of the host, ultimately leading to alterations in its growth and development. The major damage caused by parasites is probably a combination of the effects of the diversion of host metabolites to form their structure, and to the activity of the metabolites secreted into the host during the course of the infection .

1.4. Evidence for tolerance in crop and wild plants

From the account so far it is obvious that no detailed study of the type required to establish the relative levels of overall tolerance in a series of cultivars has been done. That is not to say that there is no evidence for the occurrence of tolerance in some form, and in fact evidence can be cited both for tolerance of the parasite and tolerance of disease in certain crop plants (Clarke, 1986). A clear example of tolerance of disease is shown by barley in relation to take-all caused by Gaeumannomyces graminis. Barley is affected much less than wheat by given levels of infection, and its tolerance appears to be due to its greater capacity than wheat to produce adventitious nodal roots to compensate for root tissue destroyed by the infection (Scott, 1981). The greater tolerance of some brassica crops, of clubroot disease (Plasmodiophora brassicae), appears to be associated with the development of large root systems which enable them to tolerate much greater root damage than the less tolerant forms (Crute, 1986).

There is certainly good observational evidence (Tarr, 1972) and some experimental evidence (Ben-Kalio and Clarke, 1979) that degrees of tolerance, both of the parasite and of disease, occur in wild plants. For example, studies on interactions between Senecio vulgaris and Erysiphe fischeri (Ben-kalio and Clarke, 1979) indicate that the host possesses a substantial level tolerance of the mildew. Thus studies of interactions between wild plants and their parasites are likely to be the most fruitful source of information about the levels of tolerance that may occur and of the potential value of tolerance as a breeding aim for controlling yield losses in crop plants.

Studies on crop plants alone are unlikely to provide many clues because most crop plants are the products of years of selection for resistance, and any tolerance their wild ancestors may have possessed is likely to be considerably diminished (Clarke, 1986). However, apart from studies of Ben-Kalio and Clarke (1979) and Harry and Clarke (1992) on S. vulgaris, nothing is known of the capacity of wild plants to tolerate infections and studies on other wild plant pathosystems are essential. The host-parasite

system which has been selected for study here is the oat Avena fatua / E. graminis f.sp. avenae combination for the following reasons .

Of all wild grasses common in Britain, A. fatua is the one most closely related to a cultivated cereal, A. sativa. Both species are hexaploid (2n = 42) and in fact probably constitute a biological species in that they can form fertile hybrids (Jones, 1976). A. fatua is usually distinguished in the field from cultivated forms by the greater height and vigor as well as by its whitish straw and chaff at maturity. Freshly collected seeds exhibit a characteristic dormancy which can be lost by storage under dry conditions. It has, in general, a similar morphology to A. sativa, except some differences in the inflorescence structure (Jones, 1976). It has also a short life-cycle like the cultivated oats and therefore experimental work can be completed in a relatively short period. Furthermore, A. fatua is inbreeding which means that populations of genetically uniform plants can be obtained. It is commonly infected with the same species of E. graminis that attacks the cultivated oats .

CHAPTER 2

GENERAL MATERIALS AND METHODS

2.1. Growth conditions

The experiments were carried out in growth rooms or greenhouses located at the Botany Department Research Laboratories, Garscube. The growth rooms were maintained at 19 ± 2°C, between 70 to 80 % RH with a 16 h photoperiod. During the light period a photon flux density (PFD) of about 55 μmol photons m^{-2} s^{-1} at bench level was provided by high pressure mercury vapour lamps. The greenhouse temperature ranged between 25-30°C and supplementary lighting was supplied by high pressure sodium lamps. The PFD at bench level was on average 83 μmol photons m^{-2} s^{-1}.

2.2. Plant material

2.2.1. Sources of wild and cultivated oats

Seeds of wild oat, *Avena fatua* L., were obtained from Herbiseed (Herbiseed Nurseries, Billingbar Park, Wokingham, England) and those of cultivated oat, *Avena sativa* L., were obtained from Plant Breeding International (PBI) Cambridge. Since the seed of wild oat was likely to be a mixture of genotypes, it was necessary to obtain a pure line for experiment. Plants were raised in the greenhouse and when the fruits were ripe, grains were collected from a single susceptible line and sown. Freshly harvested grains were treated with gibberellic acid solution at 50 ppm to break their dormancy before sowing. Progenies from these seeds were then raised, harvested in the same way and inbred for a further generation. Seeds of the inbred line were then used for experiments.

In a preliminary experiment to select the plant lines for the more detailed experiments, the reactions of the wild oat and a range of cultivars were tested by

exposing them to natural infection in the greenhouse, or by inoculating them using the detached leaf procedure. The results of the tests, given in Table 1, reveal that most of the plant lines were susceptible to mildew in some degree. The two most susceptible cultivars, Lustre and Peniarth, were selected for comparison with the inbred line of wild oat .

2.2.2. Growth of plants

Seeds of the two cultivars of oat and of wild oat were germinated on moistened filter paper in trays in an incubator at 22°C. Seeds of wild oat were placed for germination on filter paper moistened with an aqueous solution of gibberellic acid at 50 ppm (to break dormancy). Three to four days after sowing, the germinated seeds were transferred to S.A.I. (Imperial Chemical Industries) potting compost in 12.7 cm plastic pots, three seedlings per pot, at about 2 cm depth. A week later, seedlings of approximately equal vigour were transplanted either singly or 2 seedlings per pot, as required, into 12.7 cm or 15 cm plastic pots containing the same potting compost. Supplementary feeding was supplied twice weekly in the form of Phostrogen (P.O. 100, CORWEN, CLWYD, Wales) liquid feed .

For growth analysis, seedlings were transplanted into 15 cm pots containing coarse sand so that the root system could be harvested for dry weight determinations. A nutrient solution (Knop's) was added twice a week until 7 weeks after transplanting, and then once a week until the final harvest. Each pot received approximately 350 ml of the nutrient solution .

The nutrient Knop's solution made up with Analar chemicals was prepared by adding the volume of each of the five stock solutions listed in Table 2, to 500 ml of water. The volume was then made up to 1 litre .

TABLE 1 : The reaction of the wild oat and a range of cultivars to mildew infection.

Oat lines :	wild oat	Image	Pennal	Peniarth	Solva	Lustre	Rhianonn
Category: Winter oat		+	+	+	+	+	
Spring oat	+						+
Reaction to mildew: as seedling	HS	S	S	S	PR	HS	R
as adult plant	R*	R	R	R	R	R	R

S = Susceptible, R = Resistant, PR = Partial Resistance, HS = Highly susceptible .

* resistant at late adult plant stage .

TABLE 2

Stock solution	A	B	C	D	E	F
Volume (ml)	1.0	2.5	1.0	1.0	1.0	1.0

The compositions of the stock solutions were :

A 82 g $Ca(NO_3)_2$ $4H_2O$ in 100 ml solution .

B 20 g KNO_3 in 100 ml solution .

C 49 g $MgSO_4$ $7H_2O$ in 100 ml solution .

D 14 g K_2HPO_4 in 100 ml solution .

E 2.3 g commercial chelated iron salt in 100 ml solution (Sequestrene Fe^{138}) .

F A trace element mixture containing per ml :

28.6 mg H_3BO_3

13.8 mg $MnSO_4$ $4H_2O$

2.2 mg $ZnSO_4$ $7H_2O$

0.8 mg $CuSO_4$ $5H_2O$

0.9 mg H_2MoO_4

2.2.3. Design of experiments

In all the experiments, plants of the two cultivars and of wild oat were grown under the same conditions in a randomized design, with four replicates, either in growth rooms or in the greenhouse. The plants were moved around, in the growth room or greenhouse, at weekly intervals to ensure even growth .

2.3. Mildew cultures

2.3.1. Isolation method

Isolates of Erysiphe graminis f.sp. avenae used in this investigation were obtained from and maintained on greenhouse grown plants. Inoculum from these plants was used to inoculate all greenhouse experiments. For growth room experiments, plants were inoculated with a single conidial isolate of the mildew in an attempt to ensure the minimum variability in infection between plants. The isolate was obtained from greenhouse infected plants using the following procedure :

Conidia from infected plants were dusted directly onto uninfected leaf segments obtained from plants grown in a mildew-free growth room. The leaf segments were placed adaxial surface uppermost on 0.5 % water agar containing 150 ppm benzimidazole solution in 9 cm plastic Petri-dishes. The Petri-dishes were then incubated for 7 to 10 days in a growth room maintained at 21 ± 2°C with a 16 h photoperiod providing a PFD of 55 μmol photons m^{-2} s^{-1}. Single chains of conidia were picked off the colony with a fine pointed needle and gently placed on separate leaf segments of plants of the two cultivars or wild oat. An isolate originating from a single chain which gave good infection on the plant lines was then multiplied on fresh leaf segments of the different oat species for use in the growth room experiments .

2.3.2. Maintenance of the mildew isolate

2.3.2.1. On detached leaves in Petri-dishes

The single conidial isolate of the mildew was maintained on leaf segments in Petri-dishes on 0.5 % agar containing 150 ppm benzimidazole. Person *et al.* (1957), Caldwell (1960) and others were successful in culturing Erysiphe graminis on leaf segments on 50 ppm benzimidazole solution. This medium delays the senescence of detached leaf segments and has been found to be valid for studies of resistance to mildew. The Petri-dishes were incubated for 10 to 14 days. A temperature between 15 and 20°C was found to be the optimum range for incubation. Most of the leaf segments

started to produce visible colonies 5 to 7 days after inoculation and by the 10th day, susceptible plants had produced large heavily sporing colonies. Leaf segments remained green for about two weeks on the benzimidazole agar and supported abundant development of the fungus (Plate 1) .

2.3.2.2. On whole plants in an Isolation Plant Propagator

Seedlings of oat plants were raised in Propagator pots in a mildew-free growth room. When 10 days old, they were inoculated, covered and transferred to the Isolation Plant Propagator (Burkard Manufacturing Co Ltd.). The temperature of growth on the Propagator varied between 20 and 25°C. The plant pots were illuminated from above in a 16 h photoperiod with supplementary lighting provided by white fluorescent tubes, giving a PFD of 42 μmol photons m^{-2} s^{-1}. The plants were watered from below by a wick system and a positive pressure was maintained inside each plant chamber by blowing filtered air through the chambers. The mildew isolates could be maintained in this way for several weeks. After 4 to 6 weeks, a set of newly inoculated seedlings was transferred to the Isolation Plant Propagator .

Plants grown on the Propagator were used as a source of inoculum for most experiments involving a single conidial isolate because it was easier to obtain a large bulk of inoculum than using detached leaf segments .

2.4. Maintenance of mildew-free control plants

The fungicides used to maintain mildew-free plants in greenhouse experiments were 'Patrol' (Fenpropidin spray formulation applied at 0.1 %) or 'Benlate' (Benomyl spray formulation applied at 0.05 %) .

Fenpropidin (Fig. 1a) is a systemic fungicide based on piperidine which inhibits ergosterol biosynthesis, one of the essential components of the membranes of most fungal groups. This fungicide is phytotoxic when applied at high concentrations. The effects of phytotoxicity are usually expressed as chlorosis or tissue breakdown in

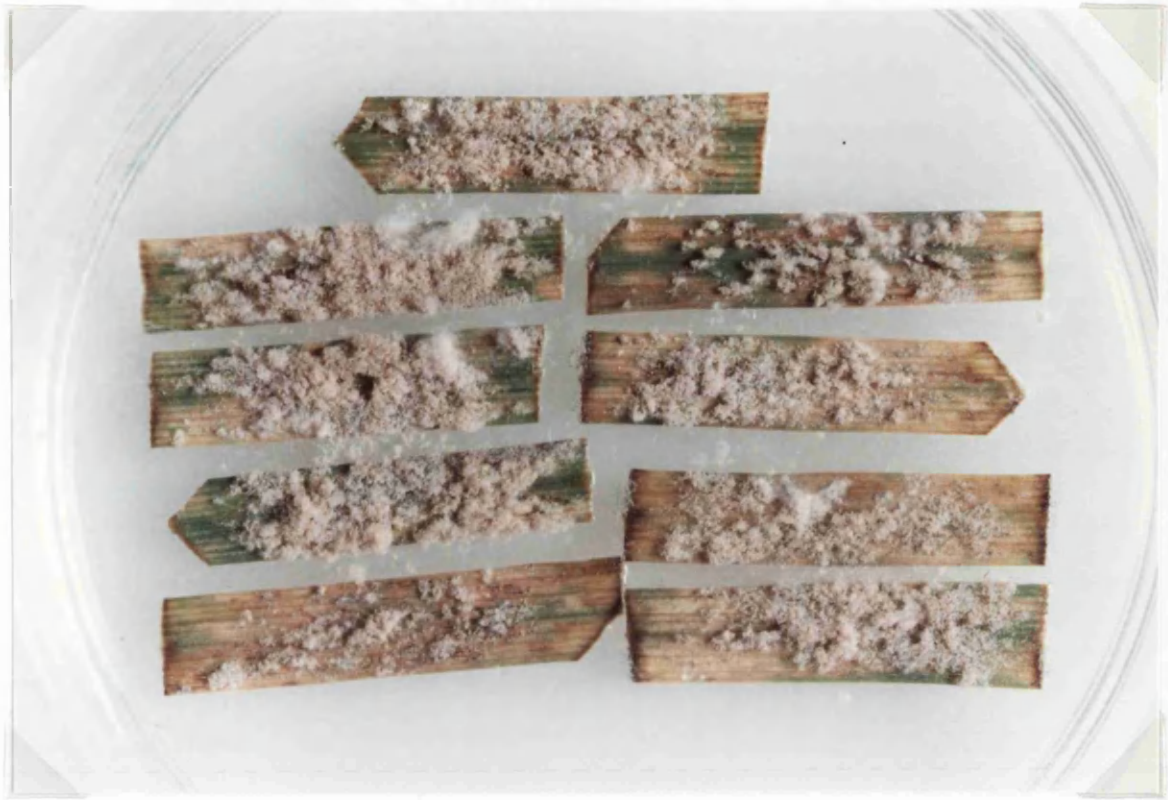
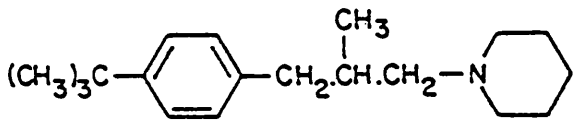


PLATE 1 : Maintenance of mildew isolates on leaf segments in Petri-dishes on 0.5 % agar containing 150 ppm benzimidazole (15 days after inoculation).

a) Fenpropidin  Patrol

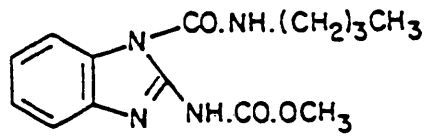
b) Benomyl  Benlate

Fig. 1 : Chemical structure of the fungicides.

leaves. In this investigation, concentrations of Patrol above 0.1 % were found to be harmful to the growth of plants .

Benomyl (methyl-1-(butylcarbamoyl)-2-benzimidazole-carbamate) (Fig. 1b) is a systemic fungicide and a member of the group known as 'MBC generators'. MBC (methyl-2-yl-benzimidazole carbamate) is the active fungitoxic molecule and it has a broad spectrum of activity. It is generally non-phytotoxic and gives good control of mildew infections. Phytotoxic effects were also expressed in plants treated with benomyl at high concentrations. Ben-Kalio (1976), in an investigation of the effects of Benlate on the growth of groundsel, showed that plants treated with the fungicide at 2 % showed symptoms of chlorosis, but not at the low concentration of 0.05 % .

Both Patrol and Benlate were applied in spray formulations. The plants to be sprayed were moved to a separate compartment of the greenhouse to ensure that spray did not drift onto the untreated plants. After spraying, they were moved back to the compartment with the untreated plants. To ensure that transfer of fungicide did not occur through root contact through the bench gravel, all the sprayed and unsprayed pots were placed on saucers. The fungicide was applied at weekly intervals from the time of transplanting since preliminary experiments have indicated that a single treatment did not protect the plant throughout the full 15 weeks growth period .

2.5. Inoculation procedures

Several methods of inoculation were used depending upon requirements :

(i) Heavily infected plants were introduced into the growth room or greenhouse and the inoculum was spread by shaking them daily for a period of one week over the experimental plants .

(ii) A small paint brush was used to transfer conidia from sporulating colonies, on leaf segments in Petri-dishes, to the leaf to be inoculated. In some experiments, the leaves were inoculated by gently brushing conidia, from sporulating colonies, over the

central region of the adaxial surface of the leaf. However, it was difficult to standardize the amount of inoculum applied by this method .

(iii) Using a spore settling tower. The mature conidia present on 10-12 days old infected plants grown in the Isolation Plant Propagator were removed by shaking the plants 24 h before inoculum was required. The spores produced during the subsequent 24 h were then used for inoculum. Inoculation was carried out 2 h before the end of the light period. The infected plants were gently shaken over the top of the tower to release a rain of spores onto leaf segments or leaves in the base of the tower. The fully expanded leaves to be inoculated were aligned horizontally, adaxial surface uppermost at the base of the tower, holding each leaf in position by attaching the distal end to a microscope slide. Conidia were shaken into the top of the tower and allowed to settle for about 10 minutes. The inoculum density, 1.75 to 2 conidia per mm^2 of leaf area, was checked in all tests by counting conidia settled on coverslips smeared with vaseline placed in the tower at the same level as the leaves. The inoculated plants were then transferred to the growth room for incubation .

2.6. Methods for assessing mildew development

2.6.1. Visual assessment of mildew development

Percentage leaf area covered by mildew colonies was determined visually on each leaf using Large and Doling's (1962) standard diagrams of mildew development (Fig. 2). The pustules on the main axis were not included in the assessment since the exposed leaf base and or stem area contributes only a small proportion of the total photosynthetic area of the plant. The leaf area was determined using a photoelectric leaf area meter (see later) and the mean percentage leaf area covered by mildew colonies on each main axis leaf was assessed. Total percentage area of leaf blade infected was calculated using the following formula :

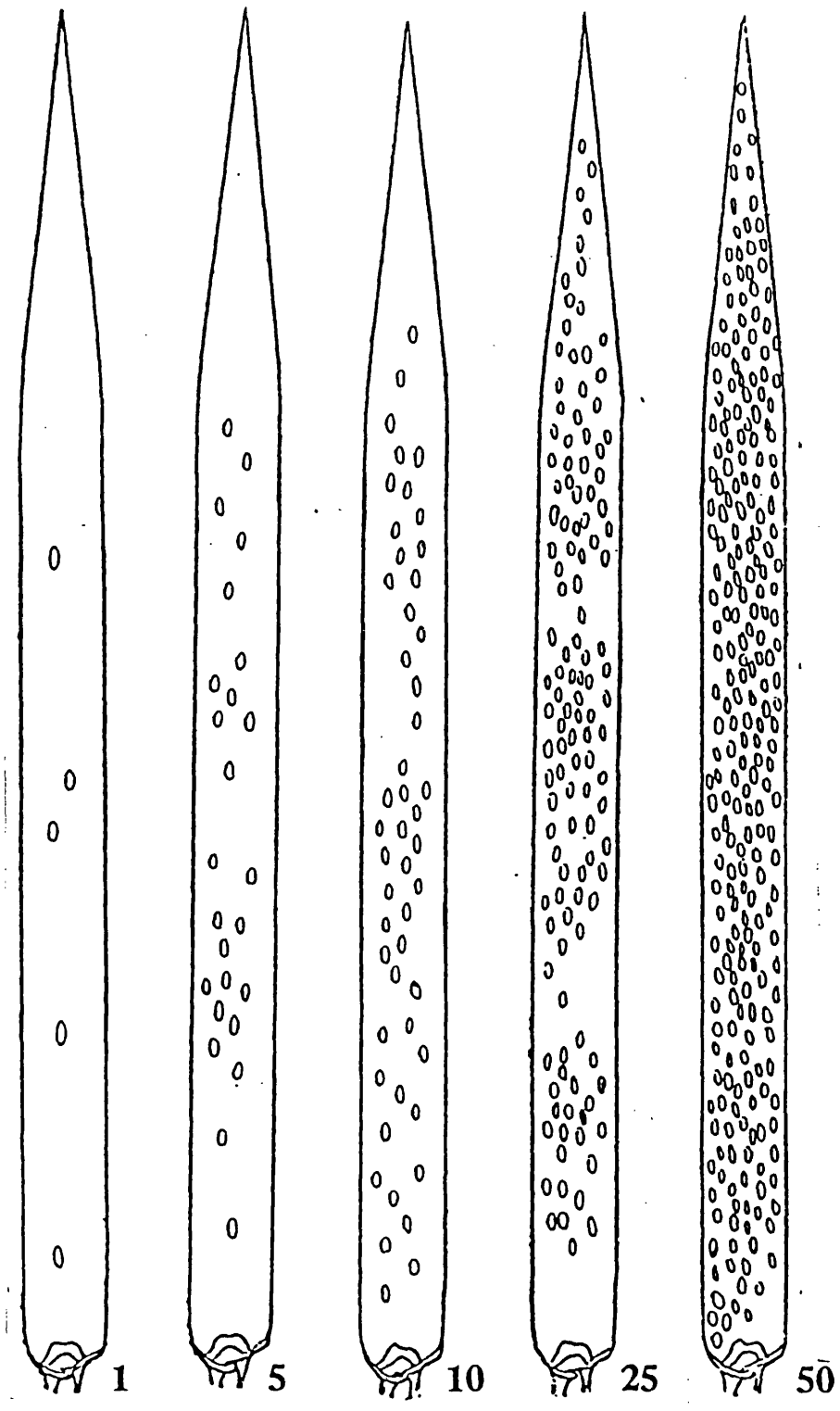


Fig. 2 : Standard diagrams used to determine percentage leaf area covered by mildew.

$$Z = \frac{y1 \cdot a1 + y2 \cdot a2 + y3 \cdot a3 + \ldots}{a_1 + a_2 + a_3 + \ldots}$$

Where y_1, y_2, y_3...n are percentage area infected of leaves 1, 2, 3...n (on the main axis) with blade areas of a_1, a_2, a_3...n .

2.6.2. Spore production

Another method of measuring fungal growth is by counting spore production per unit leaf area. Zadoks (1972b) justifies this approach on the technical ground that this method is fast, more accurate than visual estimations and non destructive .

A number of spore traps and air sampling equipment have been devised to collect spores from fungi sporulating on the surfaces of their hosts. For example, Johnson and Bowyer (1974) devised an apparatus for collecting uredospores of yellow rust from wheat leaves. Schwarzbach (1978) designed a jet spore trap, especially for the collection of mildew spores from attached living leaves. In a more direct way, spores can also be shaken from leaves or scraped from pustules and collected in a glass tube. The number of spores produced per unit leaf area can then be determined from haemocytometer counts .

The infected leaves were inserted singly into clean open-ended glass tubes 20 x 150 mm which were wide enough to insert an oat leaf without loss of spores. The leaves were then carefully cut from the plant and the tubes sealed before transfer to the laboratory. Spores were shaken from the leaves in 25 ml distilled water containing the wetting agent Tween 80 to facilitate spore dislodgement, using a vortex mixer. The area of each infected leaf was measured before it was homogenized in distilled water. The resultant suspension was agitated for half an hour on a vortex mixer. The suspension was then centrifuged at 1000 rpm for 10 min, the supernatant discarded and the pellet of spores and plant material resuspended in 5 ml distilled water .

The number of spores per unit volume in each suspension was counted using a haemocytometer. Ten counts were made per leaf sample and the mean number of spores per unit leaf area was calculated.

2.6.3. Chitin analysis

Fungal biomass can be assessed on infected plants by measuring amounts of fungal constituents such as: chitin, mannan, glucans, fungal lipids, or miscellaneous compounds such as phenols, yellow pigments, carotenoids and enzymes in infected tissues (Whipps *et al.*, 1982). The most commonly used method is based upon the colorimetric assay for hexosamines which are released on hydrolysis of chitin. The hexosamines are the basic structural units of chitin, the important hyphal wall component of most fungal groups. The assay for chitin was developed by Ride and Drysdale (1972) to estimate fungal biomass in plant tissues. It is relatively rapid and simple and has been used, with varying degrees of success,with a variety of parasitic fungi, including Fusarium spp. (Zak, 1976; Raghu Kumar and Subramanian, 1977) , Puccinia spp. (Mayama *et al.*, 1975; Whipps *et al.*, 1980) , Uromyces phaseoli (Kaminskyj and Heath, 1982) and resting spores of Plasmodiophora brassica (Thornton *et al.*, 1991) .

The assay has been criticized on the grounds that it can be confounded by galactosamine in the plant tissue and because the chitin content of mycelium varies during development (Sharma *et al.*, 1977). Whipps *et al.* (1980) noted that the assay gives only proportional values since the alkaline digestion of chitin does not proceed to completion. Nevertheless they stressed its usefulness as a quantitative method, provided that suitable account is taken of such factors. The Ride and Drysdale's method has been most frequently used for determination of fungal biomass with some modifications introduced later by several authors .

The assay used for determining the relative amount of fungal mycelium on mildewed oat leaves was slightly modified from that presented by Ride and Drysdale (1972) .

Reagents :

Acetone .

Concentrated KOH (120 g dissolved in 100 ml water) .

75 % aqueous ethanol .

40 % aqueous ethanol .

Celite suspension (The supernatant obtained by mixing 1 g celite 545 with 20 ml of 75 % ethanol and allowing to stand for 2 minutes).

5 % $NaNO_2$

5 % $KHSO_4$

12.5 % $NH_4SO_3NH_2$

0.5 % MBTH (3-methyl-2- benzothiazolinone hydrochloride) .

The solution was prepared daily .

0.5 % $FeCl_3$ (0.83 g $FeCl_3.6H_2O$ dissolved in 100 ml water) .

The solution was stored in a cool place and discarded after two days.

Assay procedure :

Samples of infected and uninfected leaf tissues of about 1 g fresh weight were weighed, cut into segments before being macerated in 10 ml of cold acetone. The slurry was centrifuged in 15 ml glass tubes at 1000 rpm for 10 minutes. After centrifugation, the supernatant was discarded and the pellet washed with 10 ml distilled water and re-centrifuged at 1000 rpm for 10 minutes. The acetone and water wash removes interfering substances which may contain hexosamines (Whipps *et al.*, 1980). The supernatant was again discarded and the pellet homogenized in 10 ml distilled water. Subsamples of 5 ml were then centrifuged and used for chitin analysis .

(1) *Hydrolysis of chitin to form chitosan*

After centrifugation, the chitin was deacetylated to form chitosan, a polymer of glucosamine, by autoclaving for 1 hour in 3 ml of KOH solution. Eight millilitres of 75 % ethanol were added to the cooled tubes and chilled on ice for 15

minutes. Then 0.9 ml of Celite suspension was layered on top of the alkali solution and the tubes were centrifuged as before. The pellet was washed once with 40 % ethanol, twice with distilled water and then suspended in distilled water to give a volume of 1.5 ml. From this stage, Ride and Drysdale recommended the use of two sensitivity levels depending on the amount of chitin present in the tissue. The first level at 0.5 ml is used for lightly infected material. The second level at 1.5 ml, for high levels of infection was used in these experiments .

(2) *Deamination and colorimetric assay*

Five percent $NaNO_2$ and 5 % $KHSO_4$ (1.5 ml each), which react to form HNO_2, were added to each tube. The solutions were shaken using a vortex mixer for 15 minutes, in order to facilitate the deamination and depolymerization of the chitosan. The tubes were then centrifuged at 1000 rpm for 2 minutes .

Two 1.5 ml samples of the supernatant were taken for the colorimetric assay. Each sample received 0.5 ml of a 12.5 % solution of ammonium sulphamate ($NH_4SO_3NH_2$) and the solutions were mixed for 5 minutes. MBTH (0.5 ml of 0.5 % solution) was added to the deaminated mixture and the tubes were heated to 100°C in a boiling water bath for 5 minutes. After cooling to laboratory temperature, 0.5 ml of 0.5 % $FeCl_3$ was added to each tube and the mixture allowed to stand for 30 minutes for the blue colour to develop before the absorbance was measured at 650 nm in a spectrophotometer .

Standard solutions prepared from known concentrations of glucosamine hydrochloride (3 to 35 µg in 1.5 ml) and water blanks were incorporated into each assay. They were treated in the same way as the final 1.5 ml samples removed for the colorimetric assay (Kaminskyj and Heath, 1982). The chitin content of diseased and healthy host tissues were thus determined using the calibration curve (Fig. 3, in the appendix) which shows a linear relationship between absorbance at 650 nm and hexosamine equivalents over the range 3 to 35 µg glucosamine .

2.7. Growth analysis of infected and uninfected plants

2.7.1. Growth analytical methods

At each harvest, the number of tillers per plant and the height of the main axis, measured from sand level to the base of the youngest leaf lamina before ear emergence but after ear emergence to the level of the fully emerged ear were recorded. The plant was then carefully removed from the sand. The root system was severed, rinsed under running tap water to remove adhering sand particles and then blotted dry .

Measurement of leaf area: A portable photoelectric leaf area meter Delta-T Devices (128 Low Road, Burwell, Cambridge CB5 OEJ, U.K.) was used for the measurement of leaf area. Yellow and brown areas were excised prior to measurement and the area of each green leaf on the main axis and total leaf area on the tillers was measured. Each leaf was measured twice and the mean calculated .

Dry weight determinations: The dry weights of the root, the main axis, leaf blades on the main axis, tillers and leaf blades on tillers of each plant were determined separately. The different plant parts were wrapped separately in pre-weighed aluminium foil envelopes and dried to constant weight at 80°C for 24 h. After cooling for about 10 minutes in a dessicator, their dry weights were determined .

Grain yield: Plants were harvested when the caryopses were ripe and the following measurements were made in addition to the number of tillers per plant and total plant dry weight .

(i) Number of ears per plant .

(ii) Number of grains per main shoot ear .

(iii) Number of grains per plant .

(iv) Hundred grain weight .

(v) Total dry weight of grain per plant .

(vi) Harvest index .

2.8. Photosynthesis and chlorophyll measurements

The instantaneous rate of photosynthetic CO_2 assimilation has generally been studied in model systems such as chloroplasts and algae. From these studies and from theoretical analyses of gas exchange behavior it is now possible to study the photochemistry of photosynthesis in intact leaves using a combination of methods, most of which are nondestructive (Sharkey, 1985) .

2.8.1. Photosynthesis in intact leaves: physiology and rate limitations

In photosynthesis, light energy (hν) is absorbed and used to drive the reduction of carbon dioxide to carbohydrate, $(CH_2O)_n$. The source of electrons for photosynthetic electron transport is the oxidation of water with the concomitant release of oxygen. The rate of liberation of molecular oxygen gives a direct measure of photosynthetic electron transport (Walker and Hill, 1967). The overall process is represented by the equation :

$$nCO_2 + nH_2O \xrightarrow{h\nu} (CH_2O)_n + nO_2$$

The limitations to the rate of photosynthesis can be divided among 3 general classes : (1) the supply and utilization of CO_2 , (2) the supply and utilization of light and (3) the supply and utilization of phosphate in the photosynthetic carbon reduction cycle (C-3 cycle) (Sharkey, 1985). These limitations are discussed in the following sections .

2.8.1.1. The supply and utilization of CO_2

The efficient CO_2 supply to the carboxylation sites within the leaf depends on the concentration of CO_2 in the surrounding atmosphere and photosynthesis increases with increased CO_2 concentration. Once inside the chloroplast, CO_2 is enzymatically combined with ribulose-1,5-bisphosphate (RuBP) to form two molecules of 3-phosphoglycerate (PGA), the first product of photosynthesis. The enzyme involved is ribulose-1,5-biphosphate carboxylase/oxygenase usually abbreviated to Rubisco. The

CO_2 supply to the carboxylation sites within the leaf depends upon the amount or activity of rubisco (Sharkey, 1985).

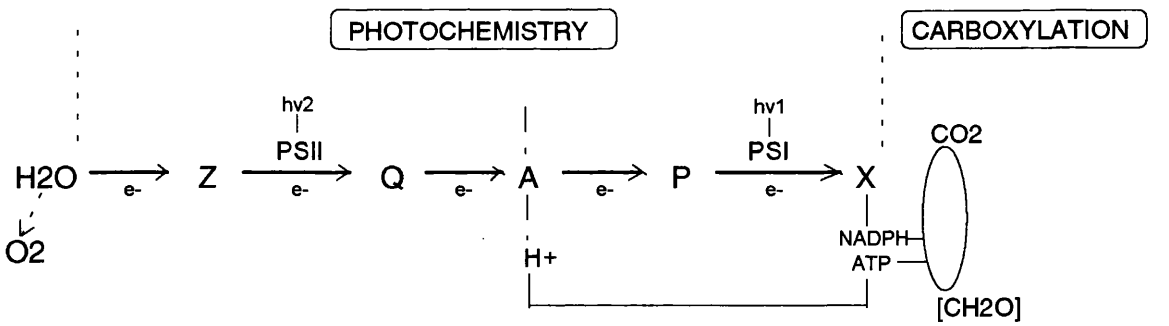
2.8.1.2. The supply and utilization of light

The absorption of light by chlorophyll causes an electron to become more electronegative. This energy can be lost as heat, or as light (fluorescence), or the electron can leave the chlorophyll molecule and reduce a neighbouring molecule.

(1) *Photosynthetic electron transport*

The electron transport chain in photosynthesis is believed to involve two photochemical reaction centres in series PSI and PSII. These centres are excited by antenna chlorophyll complexes which can absorb light and transfer that energy to reaction centre chlorophyll and a light-harvesting chlorophyll-protein complex (LHC) which can increase the effective light absorption in the photosystem reaction centre.

The following simple model of the electron transport chain in photosynthesis is given to serve as a frame of reference for later discussion.



In this model, Z and Q are the primary electron donor and acceptor of light reaction centre PSII, $h\nu_2$ and $h\nu_1$ are quanta absorbed in light reaction centres PSI and PSII, A is an intersystem intermediates (e.g. plastoquinone, PQ, $Cytb_6/f$ complex etc.), P and X are primary electron donor and acceptor of light reaction centre PSI and e- are electrons flowing from PSII to PSI. The NADPH and ATP generated are consumed in the reduction of CO_2 to sugars in the carbon assimilation cycle (carboxylation) (Munday and Govindjee, 1969).

(2) *Phosphorylation*

Photosynthetic phosphorylation is the production of ATP. Protons are pumped from outside the thylakoids during electron transport. For every electron transported to $NADP^+$ one proton is liberated inside the thylakoid during the oxidation of water and one proton is translocated across the thylakoid membrane by the plastoquinone. The resulting pH gradient across the thylakoid is the principal component of the proton motive force (pmf). When the pmf is sufficient, the outward passage of three internal protons is required for the synthesis of ATP from ADP and P_i. If either ADP or P_i is not in sufficient supply the pH gradient will build up and inhibit photosynthetic electron transport .

2.8.1.3. The supply and utilization of phosphate

The ATP and NADPH generated by photosynthesis electron transport are used in the C-3 cycle to convert 3-phosphoglycerate (PGA) to triose phosphate in the presence of enzymes. The enzymes required for the thirteen steps of the carbon cycle are present in photosynthetic tissues. Deficiencies in these enzymatic activities result in a reduction in the rate of photosynthesis .

2.8.2. Photosynthesis measurement

2.8.2.1. Principle of Oxygen evolution measurement

The measurement of oxygen evolution was carried out using a Hansatech LD2 Leaf Disc Electrode Unit (Hansatech Ltd., Paxman Road, Hardwick Industrial Estate, King's Lynn, Norfolk, U.K.) .

The O_2 electrode: The O_2 which accumulates in the gas-phase during photosynthesis was detected by a disc located in the lower half of the middle section of the apparatus. The disc was a conventional Clark-type $Pt/Ag/AgCl_2$ electrode (Delieu and Walker, 1981). It consists of an electrochemical cell containing a platinum cathode

and a silver anode immersed in a solution of saturated KCl. The electrodes were protected by a thin wick (cigarette paper) and an oxygen permeable polythene membrane, of approximately 2 cm^2 each, from a reaction chamber containing the sample leaf .

The leaf chamber: The leaf chamber is located on top of the electrode and the platinum cathode is exposed to the atmosphere in the leaf chamber through a small hole in its floor. The leaf chamber and the electrode disc are both encased in a massive anodized aluminium case through which water from a thermostatically-controlled water bath was passed in order to maintain a steady temperature (± 0.01°C). The chamber itself is cylindrical (5 ml internal volume when empty) and accommodated small pieces of leaf of 10 cm^2 area on a perforated stainless steel disc, with an unperforated centre to prevent light falling directly onto the cathode. The upper section of the chamber is the perspex floor of the upper water jacket, through which the chamber can be illuminated. When the chamber is closed, the upper section is fixed in position by two clips. O-rings are used to give an air-tight seal. Two taps, fitted to the outside of the leaf chamber, enable the chamber to be connected to the external atmosphere. These taps are used in the calibration procedure when the oxygen partial pressure is altered by inserting or removing a volume of air .

The CO_2 concentration of the sealed atmosphere of the leaf chamber is maintained at approximately 1 % (v/v) by moistening capillary matting on the floor of the chamber with sodium hydrogen carbonate ($NaHCO_3$) buffer (200 µl of 1M solution). The leaf tissue is protected from the alkaline buffer by a second stainless steel disc and a foam rubber disc within the chamber. A probe fitted with its own perspex window is inserted into the chamber and allows chlorophyll fluorescence detection by a photodiode .

For all measurements of oxygen evolution from oat leaf segments, the whole unit was kept at approximately 23°C and illumination was provided by a 24°, 50W Dichroic quartz halogen spot lamp (WOTAN). The PFD measured at the position normally occupied by the leaf material was about 828 µmol photons m^{-2} s^{-1}. The incident light intensity was altered using Balzar neutral density filters placed on the top

of the window. Nine filters, ranging from 1 % to 75 %, were used and the percentage transmission expressed in μmoles photons m^{-2} s^{-1} .

Calibration: Using a gas-tight syringe, a volume of air or N_2 was inserted through one tap while the other one was kept closed. The electrical output of the O_2 sensor indicated on a recorder as voltage, rose to a steady state when a volume of air was injected and fell when the chamber was flushed with N_2. The difference between the electrical output of the electrode in air and N_2 is a measure of the partial pressure of O_2 in the internal atmosphere. Thus within the limits imposed by the potentiating circuit, the deflection (in millivolts) corresponded to the number of moles of O_2 at T °C .
The calibration was carried out either before or after the leaf material was placed in the leaf chamber .

Photosynthesis measurements: For photosynthesis measurements, the steady state of O_2 evolution was determined. This was attained when a straight line was recorded for at least 7 minutes. The dark respiration (Rd) was calculated after placing the leaf material in the dark for about 15 minutes and the net photosynthesis (Pn) also estimated by illuminating the leaf material for 6 to 8 minutes at each PFD .

2.8.2.2. Diffusion pathway of photosynthesis

Before fixation, CO_2 must first diffuse through the leaf boundary layer, the stomata and the mesophyll layer (comprising intercellular spaces, cell wall and intracellular fluid). The process of diffusion can be described by an electrical analogue where the fluxes Pn and Rd, driven by concentration gradients, pass through resistors representing steps in the pathway. The resistance chain represented in Fig. 4 is a convenient simplification of the process of CO_2 diffusion which forms the basis of the photosynthesis-light response (PLR) model (Marshall and Biscoe, 1980) .

2.8.2.3. Calculating photosynthesis rates using PLR model

Some models have been proposed to describe the response of photosynthesis to irradiance for C_3 leaves .

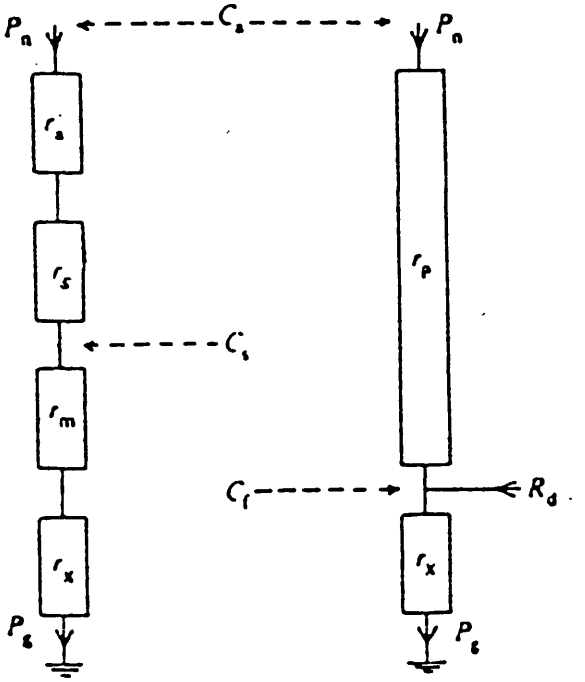


Fig. 4 : Circuit representing the diffusion pathway of CO_2 from the atmosphere to the site of fixation within the leaf. It is used in the derivation of the photosynthesis-light response model (PLR).

C_a CO_2 concentration in the atmosphere.

C_f CO_2 concentration at the site of fixation.

C_s CO_2 concentration in the intercellular spaces within the leaf.

Pg gross photosynthesis.

Pn net photosynthesis.

Rd dark respiration.

r_p physical diffusion resistance

r_a boundary layer resistance.

r_m mesophyll resistance.

r_s stomatal resistance.

r_x chemical or carboxylation resistance

Equations described the model :

$$Pg = Pn + Rd$$

$$Pn = (C_a - C_f) / r_p$$

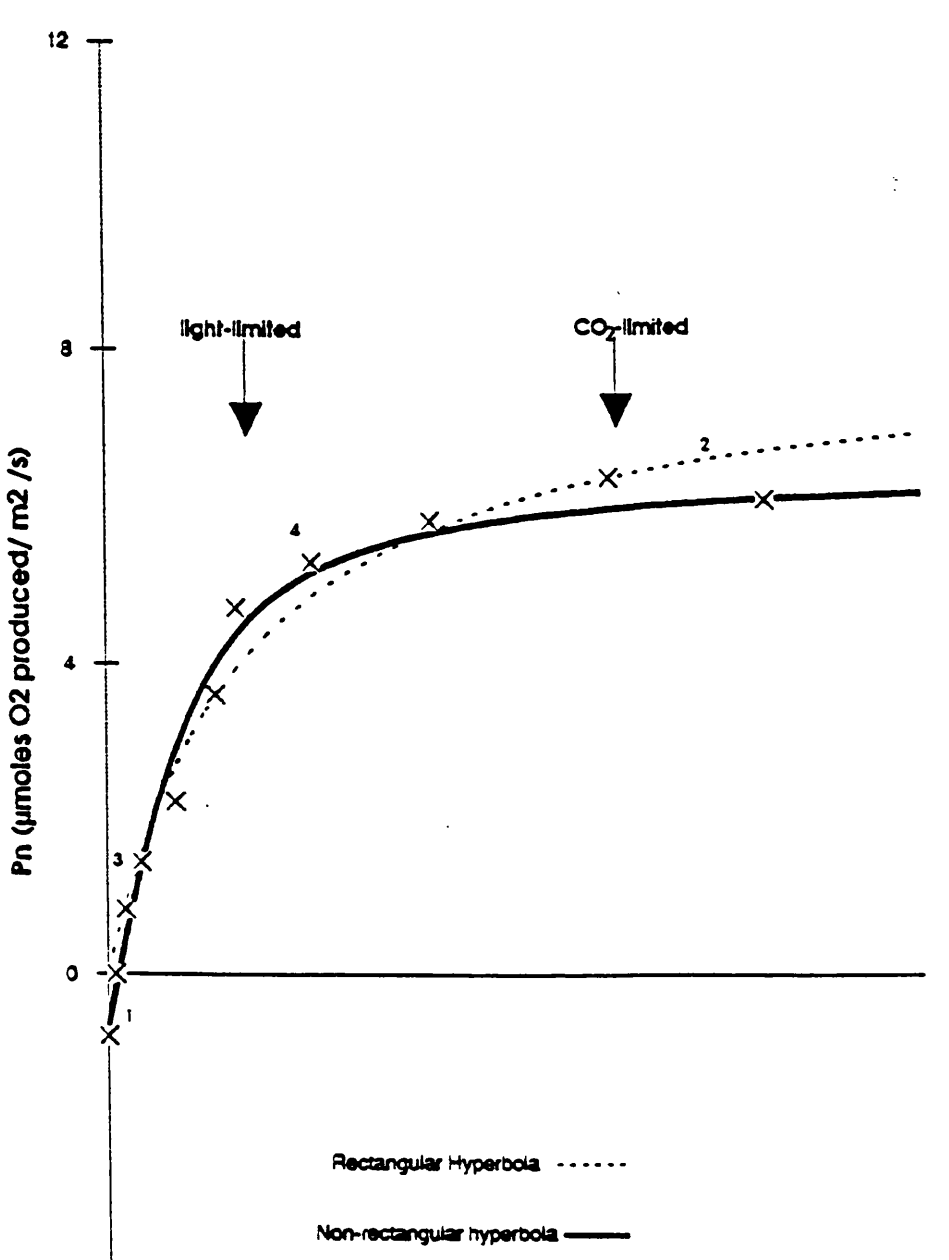


Fig. 5 : The photosynthetic parameters determined from the photosynthesis-light response curve using the model of Rabinowitch (- - - -) or that of Marshall and Biscoe (——) 1) Rd, 2) Pnmax, 3) α and 4) θ.

(1) The model of Rabinowitch

Rabinowitch (1951) proposed a model based on the biochemical reactions within the chloroplast and used various simplifying assumptions to describe these complexe reactions. The model describes the relationship between photosynthesis and irradiance in terms of a *rectangular hyperbola* (Fig. 5). The PLR curve has no sharp discontinuity between light-limited and CO_2-limited parts of the curve nor does photosynthesis saturate in bright light. The model uses 2 parameters: Pgmax and α :

$$Pg = \frac{Pgmax \,.\, \alpha I}{Pgmax + \alpha I} \qquad (1)$$

Where:

Pg = rate of gross photosynthesis (Pg = Pn + Rd).

Pgmax = maximum rate of gross photosynthesis.

α = photochemical efficiency of photosynthesis at low light intensity or quantum yield of light harvesting complexe (LHC).

I = photon flux density.

The data collected from Hansatech leaf electrode was used to calculate Pg. Plots of Pg against [I] are not linear and therefore it was not possible to measure Pgmax. However the model of Rabinowitch can be linearised by inverting the equation (1) and multiplying by [I] to give:

$$I / Pg = \frac{1}{\alpha} + \frac{1}{Pgmax} I \qquad (2)$$

A plot of [I}/Pg against [I] gives a straight line and Pgmax can be determined from the gradient [I]/Pgmax. The photosynthetic efficiency at low light intensity (α) is determined from the gradient Pg against [I]. The model (1) can also be solved by non-linear solver procedure of Microsoft Excel version 4.0.

The model (1) was modified by Thornley (1976b) for a simple enzyme-substrate reaction (Marshall and Biscoe, 1980). The gross photosynthesis depends on [I], C_f and r_x according to:

$$Pg = \frac{\alpha I \,(C_f / r_x)}{\alpha I + (Cf / rx)} \qquad (3)$$

Where :

C_f = CO_2 concentration at the site of fixation.

r_x = chemical or carboxylation resistance

C_f/C_x = Pgmax

(2) The model of Marshall and Biscoe

Referring to Fig. 4, a modification, appropriate for photosynthesis, of the model (3) was derived by Marshall and Biscoe (1980). The derived model (4) combines a simplified description of the biochemical reactions occuring within the chloroplast with the physical diffusion of CO_2 from the atmosphere. The model is a *non-rectangular hyberbola* and uses 4 parameters: Rd, Pmax, α and θ (fig. 5) :

$$\theta Pn^2 - (Pgmax + \alpha.I - \theta Rd)Pn + \alpha.I(Pgmax - (1-\theta)Rd) - Rd.Pgmax = 0 \quad (4)$$

where a is still the initial slope at low light intensity and q is the ratio of physical to total resistance to diffusion of CO_2 (r_p/r_p+r_x) which describes the degree of curvature at the shoulder of the PLR curve (Fig.5). The maximum rate of net photosynthesis, Pnmax, is calculated from the equation :

$$Pnmax = Pgmax - (1 - \theta)Rd \quad (5)$$

The model as it appears in equation (4) is in quadratic form and can be rewritten :

$$y = aPn^2 + bPn + c = 0 \quad (6)$$

Where :

$a = \theta$

$b = - (Pgmax + \alpha.I - \theta Rd)$

$c = \alpha.I (Pgmax - (1 - \theta)Rd) - Rd.Pgmax$

When y = 0, the solution or root of the equation (6) is :

$$x = \frac{-b \pm \sqrt{b^2 - 4a.c}}{2a}$$

The data was fed to the computer and analysed using a conjugate method in solver programme of Microsoft Excel version 4.0. Constants a, b and c were evaluated for any particular level of irradiance, given parameters Pmax, Rd, α and θ and equation (6) solved for Pn .

The model of Marshall and Biscoe is shown to be a significant improvement on *the rectangular hyperbola* (1). It provides good fits to the

measurements made during the course of this investigation, and gives consistent and reliable estimates of the 4 parameters (Marshall and Biscoe, 1980).

2.8.3. Measurement of chlorophyll content

The chlorophyll content of oat leaf segments was determined spectrophotometrically by the method of Mackenny (1941). The same leaf segments used for photosynthesis measurement were used for chlorophyll analysis.

Procedure :

The leaf segments were placed in a 25 ml tube and 25 ml methanol was added.

The tube was then wrapped in aluminium foil and placed in a heated water bath at 60°C for 30 to 40 minutes.

After heating, the tube was removed from the bath, cooled to room temperature and centrifuged at 70 g for 5 minutes to remove large fragments.

The supernatent was placed in a 25 ml volumetric flask and made up to volume with methanol.

The absorbance of the solution was read against methanol at 665 and 650 nm in a spectrophotometer. The amount of chlorophyll in the solution was calculated using the following formulae :

$$\mu g \text{ Chl a} \cdot ml^{-1} = 16.5 (A_{665}) - 8.3 (A_{650}).$$

$$\mu g \text{ Chl b} \cdot ml^{-1} = 33.8 (A_{650}) - 12.5 (A_{665}).$$

From these values, the ratio Chl a / Chl b and total chlorophyll could be calculated.

2.9. Fluorescence

Measurement of chlorophyll fluorescence quenching induced in photosynthetic systems by exposure to light can provide qualitative information on the organization and functioning of the photosynthetic apparatus (Genty *et al.*, 1989). The

changes in rate of oxygen evolution are accompanied by fluctuations in the yield of chlorophyll fluorescence. The problem is to relate these fluctuations to the mechanism of photosynthesis .

2.9.1. Fluorescence induction from intact leaves

Most fluorescence is emitted by chlorophyll a in PSII reaction centre. The factor controlling chlorophyll fluorescence intensity is the redox state of Q, the electron acceptor in the PSII reaction centre (see section 2.8.1.2.). Thus if a leaf is kept for a few minutes in the dark (or in low light) and then illuminated with bright light, chlorophyll fluorescence rises, in fractions of a second, to O and then to an initial peak P (fast change), then falls to a level S before rising again to a second peak M and finally declines to a steady state T in a process lasting several minutes (slow change) (Fig. 6a) (Quick and Horton, 1984 and Walker, 1987). The fast fluorescence change from O to P is considered to indicate the reduction of electron acceptors between photosystems PSII and PSI, and probably represents the change in electron transport rate through Q primarily from the water splitting complex to the pool of intersystem intermediates (see section 2.8.1.2.) (Papageorgiou, 1975; Lavorel and Eitienne, 1977; Miranda *et al*, 1981). Reoxidation of Q makes an important contribution to the slow decline in fluorescence from P to T, which is not determined solely by the redox state of Q but is also ultimately linked in with the onset of photosynthetic carbon assimilation (Quick and Horton, 1984) .

Thus the interpretation of fluorescence induction curves is complicated by the existence of several photochemical and non-photochemical components of fluorescence quenching. The major quenching components are attributable to the following : photochemical q-quenching (qQ), due to the redox state of the PS II reaction centre (Q_B acceptor), and non-photochemical energy-quenching (qE) which is closely related to the build up of the proton gradient (pH) which develops across the thylakoid membrane (Horton, 1983) (Fig. 6b) .

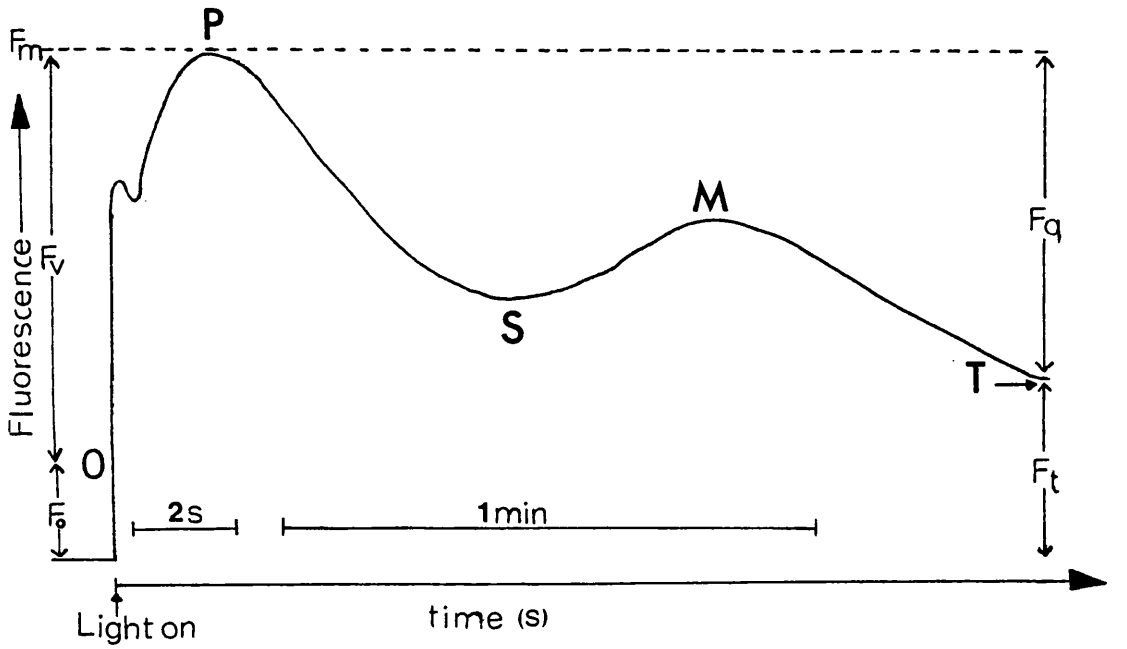


Fig. 6a : The kinetics of *in vivo* fluorescence induction. The light is turned on at zero time following pre-darkening of the leaf for 15 minutes. The symbols O-P-S-M-T are listed to describe the various transients of the induction curve. Fo, initial level of fluorescence, attained within a few milliseconds of the onset of illumination. Fm, maximal fluorescence during induction period, attained at P; Fv, variable fluorescence [=(Fm-Fo) / Fo]; F_t, steady state level of fluorescence attained at T; Fq, fluorescence quenching [=(Fm-F_t) / Fo) .

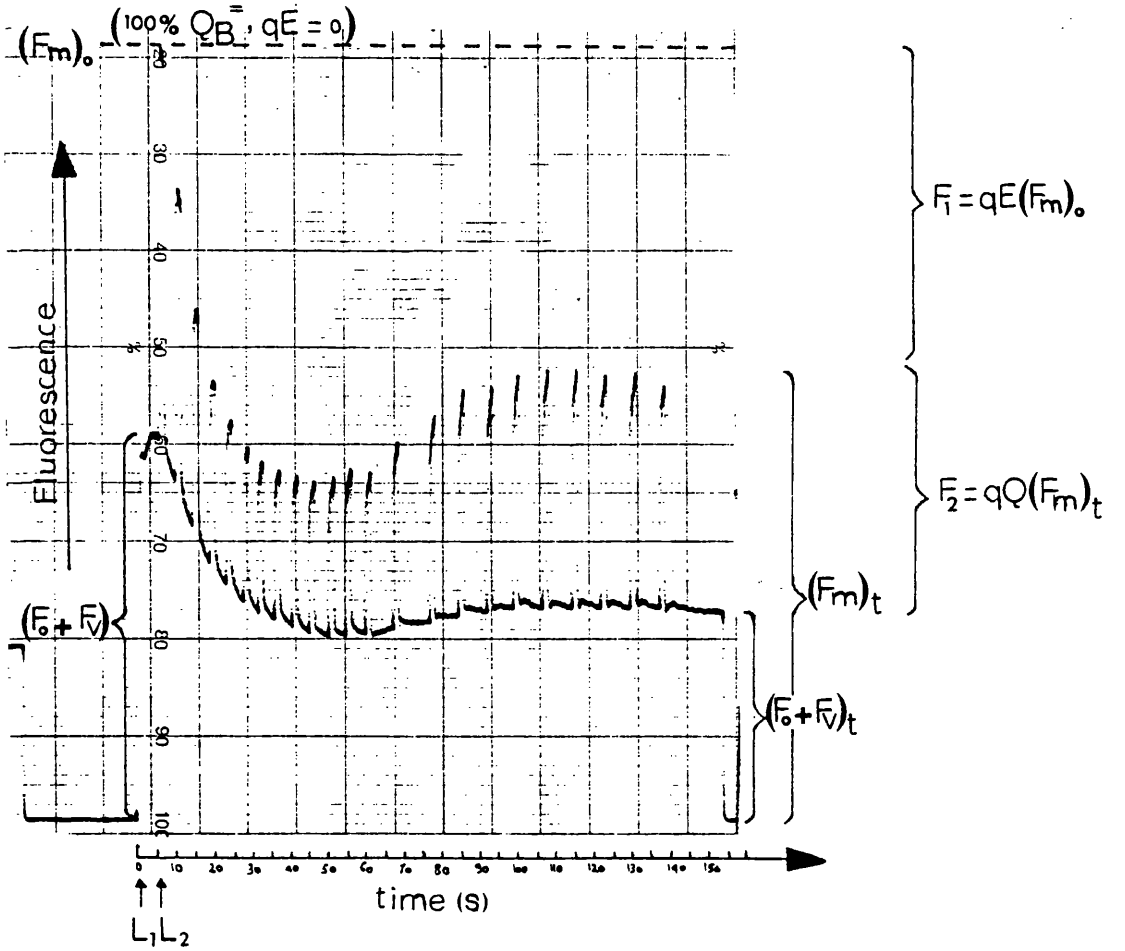


Fig. 6b : Fluorescence quenching coefficients and terminology of fluorescence parameters using light-doubling method.

2.9.2. Fluorescence induction measurement

Modulated fluorescence techniques allow the chlorophyll fluorescence components, qQ and qE, to be resolved and quantified. An alternative approach, light-doubling, first introduced by Bradbury and Baker (1981), has been used extensively on chloroplasts and leaf tissue (Miranda *et al.*, 1981; Kraus *et al.*, 1982; Dietz *et al.*, 1985; Schreiber *et al.*, 1986 and Genty *et al.*, 1989) .

2.9.2.1. Principle of fluorescence induction measurement

In vivo chlorophyll fluorescence kinetics were determined using freshly detached leaf segments (30 mm length), by a custom built modulated fluorimeter (Fig. 7). The leaf tissue was irradiated via two arms of a trifurcated glass fibre optic light guide (Ealing optics). The first arm delivered a PFD of 100 µmol photons m^{-2} s^{-1} modulated blue light (L_1) (64 Hz) onto the leaf surface. The light source was provided by a 20W quartz halogen lamp filtered through a Schott BG-1 glass filter. The irradiation was produced by an EG+G light chopper (9479 EG+G Brookdeal). The second arm delivered a PFD of 300 µmol photons m^{-2} s^{-1} of continuous blue light (L_2) on the leaf surface. The light source for this arm was provided by a 50W quartz halogen lamp filtered through a Schott BG-1 low pass blue filter. A mechanically operated optical shutter was placed in the light path to control repetitive flashes of L_2 .

The fluorescence (modulated plus continuous) was collected by the third arm of the trifurcate fibre optic and filtered through a Schott RG 665 high pass filter and a Balzar 685 nm interference filter. The transmitted light was collected by an EMI (9658 QA) photomultiplier and the signal demodulated by a Brookdeal 9503 mode-locked amplifier and recorded on a chart recorder (Fig. 7) .

This configuration allowed light-doubling experiments to be performed on intact leaf tissue (Bradbury and Baker, 1981) from which information can be drawn concerning the efficiency of the photochemical and enzymic processes of photosynthesis (Walker, 1987). Unfortunately, due to the rejection ratio of the Brookdeal 9503 amplifier, it was not possible to monitor fluorescence using a weak

modulated beam, as described by Schreiber *et al.* (1986), and so it was not possible to measure Fo with a high degree of precision .

2.9.2.2. Determining fluorescence parameters and quenching coefficients

In this investigation, it was not possible to measure the initial fluorescence rise Fo of dark adapted leaf tissue. Dark adaptation ensures that Q_B is fully oxidised before the onset of variable fluorescence. Upon irradiation of the leaf tissue with the modulated light (L_1), the variable fluorescence rises to an initial peak (Fo + Fv) or Fm (Fig. 6b). A further rise of variable fluorescence (Fo + Fv) to a maximum peak level $(Fm)_o$ occurs upon the application of continuous light (L_2), which fully reduces the electron acceptor Q_B (100 % $Q_B^=$, qE=0) (Fig. 6b). Thereafter, application of repetitive saturating light pulses which fully reduce Q_B gives smaller fluorescence peaks at any given time $(Fm)_t$, largely because of the development of qE quenching (Fig. 6b) .

The leaf tissue sample was placed in darkness for 15 minutes before fluorescence measurements were made. The continuous light was applied in short pulses of 2 seconds duration, beginning immediately after the initial rise of variable fluorescence (after about 2 seconds), and continuing at intervals of 5 seconds until 60 seconds, and then at intervals of 10 seconds until 160 seconds. The photochemical fluorescence quenching qQ and non-photochemical fluorescence quenching qE were determined, from the fluorescence induction curves, as follow :

$$qE = \frac{(Fm)_o - (Fm)_t}{(Fm)_o} = \frac{F1}{(Fm)_o}$$

$$qQ = \frac{(Fm)_t - (Fv + Fo)_t}{(Fm)_t} = \frac{F2}{(Fm)_t}$$

The fluorescence parameter qQ was determined in the same way as Genty *et al.* (1989) .

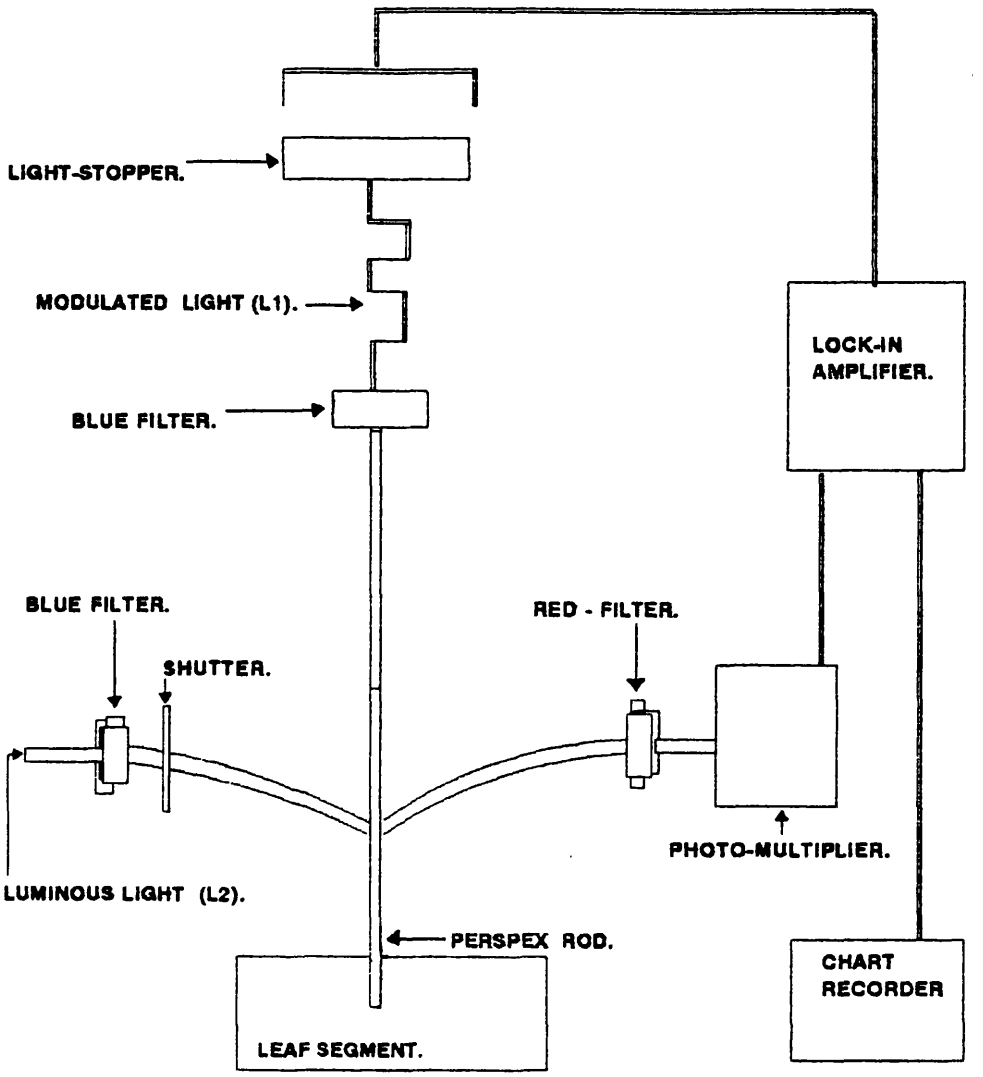


Fig. 7 : A modulated trifurcate glass fiberoptic fluorimeter.

2.10. Stomatal measurement

Attempts have been made to measure stomatal aperture ever since it was realized that gas exchange between plants and the atmosphere is regulated by the degree of opening of the stomata. For this purpose, different methods for measuring the dimensions and frequencies of stomatal appararus and the degree of opening have been used. The most obvious method is direct microscopic observation of stomata on a living leaf or portion of a leaf. Important indirect methods of observation which are used, include transpiration and leaf porometer measurements (Meidner and Mansfield, 1968 and Wilkins, 1969). Leaf porometer measurements have been broadly used to study stomatal behaviour, transpiration and CO_2 exchange in infected leaf tissues (Majernik, 1971 and Ayres, 1972 and 1976) .

2.10.1. Stomatal frequencies and distribution

Microscopic measurements were carried out to estimate the number of stomata and epidermal cells other than stomatal cells, of infected and uninfected oat leaves. Lower epidermal imprints were obtained by spreading nail varnish over equivalent positions along the leaf on both infected and uninfected leaves. These epidermal imprints were allowed to dry and then peeled off carefully. Each epidermal imprint was then mounted in distilled water on a microscope slide. The number of stomata and other epidermal cells per unit area was determined microscopically by counting the numbers per field of view (0.332 mm^2) at a magnification of x 200 .

2.10.2. Porometer measurements of leaf diffusive resistance

Measurements of leaf diffusive resistance were carried out using an automatic porometer MK 3 Delta-T- Devices (128 Low Road, Burwell, Cambridge CB5 OEJ, U.K.) .

2.10.2.1. Principle of measurement

The porometer measures the outward diffusion of water vapour through the stomata. An air tight cup, in which is situated a relative humidity sensor, is attached to a leaf for short periods of time. The sensor is fixed at a certain distance from the leaf, and water vapour emitted by the transpiring leaf diffuses out of the leaf across this distance, onto the initially dry element at rates depending chiefly on the degree of stomatal opening .

Before a reading is taken a small battery-operated pump blows an air stream, dried by passing through a column of silica gel, through the cup so that the sensor becomes dry and offers an infinite resistance to current flow. As the sensor becomes moist, its conductivity increases, and a gradually increasing current flows through the sensor. The time for the current to increase to a set level is measured automatically over a fixed interval. The rate of increase in conductivity of the sensor is directly proportional to the rate of outward diffusion of water vapour from the leaf. A thermistor is built into the leaf clamp so that the temperature of the leaf can be measured after every porometer reading. The readings obtained are then converted to diffusive resistance using a calibration curve which is usually obtained before the leaf measurements are taken and which is checked at intervals if leaf measurements are taken over a long period .

Calibration; A calibration plate containing six sets of holes of different sizes which provide six diffusion resistances of known value was used for calibration. A piece of absorbent paper (30 x 60 mm) was wetted with distilled water and placed on the flat side of the calibration plate to cover all holes. A piece of brown waterproof tape was then stuck over the paper ensuring that the edges were sealed. The plate was inserted into the sensor, engaging the position of each set of holes. At a given temperature and RH level, the counts at each position, when stabilised, were recorded. A calibration curve was obtained by plotting the six resistance values against the corresponding counts .

2.10.2.2. Leaf diffusive resistance measurements

Measurements of stomatal diffusive resistance in the light/dark period were made on attached leaves on plants. All measurements in the light were carried out in the laboratory at 12 pm. and near the window so that plants were exposed to natural daylight. The temperature in the laboratory was about 20 ± 2°C and the relative humidity about 42 ± 3 %. After measurements in the light, the plants were transferred to a darkroom for 24 h to induce stomatal closure. Measurements of stomatal diffusion resistance were then made in green light. The temperature in the darkroom was maintained at about 19 ± 2°C and the relative humidity at about 45 ± 2 % .

Constant measurements on both upper and lower surfaces of the leaf for the same sample area were difficult to obtain. Measurements were only taken in the middle and tip of the upper surface of infected and uninfected third leaf blades on each plant line .

2.11. Statistical treatment of the data

In order to make comparisons between infected and uninfected plants within each plant line and between the three lines, all the data were subjected to an analysis of variance using the GENSTAT statistical programme followed by the LSD test .

CHAPTER 3

THE EFFECTS OF MILDEW INFECTION ON THE GROWTH AND DEVELOPMENT OF WILD AND TWO LINES OF CULTIVATED OAT.

3.1. Introduction

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

A. Simple ratios

Simple ratios have been constructed (Hunt, 1978) for the determination of the distribution of dry matter along the plant axis. The more frequently used ones are as follow :

Root : Shoot ratio : This ratio indicates the relative proportion of dry matter allocated between the root and shoot during plant growth .

Leaf Weight Ratio

Leaf weight ratio (LWR) is the ratio between total leaf dry weight and total plant dry weight. In analytical terms it is an index of the leafiness of the plant on a weight basis which represents the average fraction of the plant's total stock of organic material divided between the photosynthesizing organs and the rest of the plant (Hunt, 1978 and 1990). It is derived from the formula :

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

Where : LW = Total leaf dry weight

W = Total plant dry weight

Leaf Area Ratio

Leaf area ratio (LAR) is defined as the ratio of total leaf area to whole plant dry weight. It is a morphological index of the leafiness of the plant, devised by Briggs, Kidd and West (1920b). In a broad sense, it describes the relative size of the assimilatory apparatus (Hunt, 1978 and 1990) and is calculated from the formula :

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

Where : LA = Total leaf area

Specific Leaf Area

Specific leaf area (SLA) is defined as the mean area of leaf displayed per unit of leaf weight (Hunt, 1978 and 1990). It reflects leaf density or relative thickness and varies according to the relative proportions of the assimilatory and conductive or mechanical tissues in the leaf. It is derived from the formula :

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

B. Indices of plant productivity

The Relative Growth Rate

The relative growth rate (RGR) is an index of the productive efficiency of plants in relation to total dry weight (Hunt, 1990). It represents the efficiency of the plant as a producer of new material (Blackman, 1919 and Williams, 1946)). 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 :

$$R_{2-1} = \frac{\log_e W2 - \log_e W1}{T_2 - T_1}$$

Where :

W_1 = the total plant dry weight at time T_1

W_2 = the total plant dry weight at time T_2

T_2-T_1 = the time interval between harvests

e = the base of natural logarithms

Unit Leaf Rate

The unit leaf rate (ULR) is an index of the productive efficiency of plants in relation to total leaf area (Hunt, 1990). It is a measure of increment in dry weight per unit leaf area per unit time. 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 between two harvests :

$$E_{2\text{-}1} = \frac{W2 - W1}{T_2 - T_1} \cdot \frac{\text{Log}_e\ LA2 - \text{Log}_e\ LA1}{LA_2 - LA_1}$$

Where :

LA_1 = the total leaf area at time T_1

LA_2 = the total leaf area at time T_2

This expression makes the assumption that weight and leaf area are linearly related over the period of the observations. Various other formulae are available in cases where total plant dry weight is not linearly related to leaf area (Evans, 1972) .

For the present investigation, the Williams' formula was adopted because total dry weights and leaf areas were linearly related (Fig. 8, in the appendix), and for the additional reason that the time interval between harvests, being only seven days, was relatively short .

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

LAR = SLA x LWR

RGR = ULR x LAR

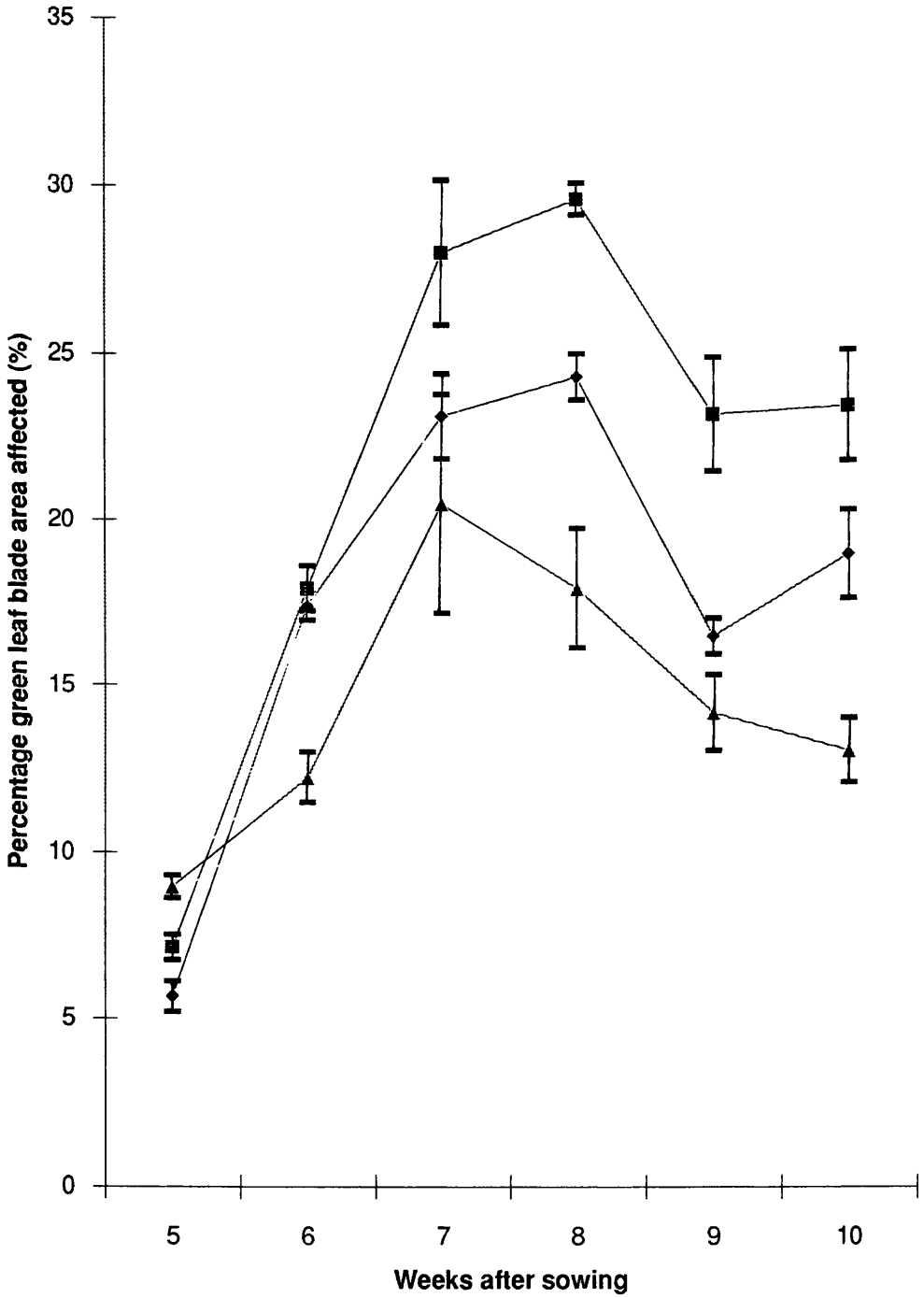


Fig. 9 : Mildew development on green leaves expanded on the main axis, in wild oat (■) and cvs Lustre (◆), Peniarth (▲).

3.2. A comparison of mildew development on cvs Lustre, Peniarth and wild oat

3.2.1. Percentage leaf area covered by mildew

Since the percentage leaf blade area covered with mildew was assessed on the plants used for the growth analysis measurements, the account of the way the plants were grown and inoculated is found in section 3.3. The assessments were made using the diagrammatic scale given in Materials and Methods (Fig. 2). The development of mildew, as mean percentage green leaf blade area affected on the main axis is given in Appendix tables 4 to 6 and the results are summarized in Fig. 9 .

Mildew infection in the dry and humid conditions of the greenhouse was much higher than that which is usually found under natural conditions in the field. Colonies of the mildew developed over all the aerial parts of the plants including both surfaces of the leaf blade, Plates 2 and 3. Mildew colonies covered a greater percentage area of the leaf in wild oat than in cvs Lustre and Peniarth. Infection was first apparent in the three lines one week after inoculation and the level increased rapidly from then on and within seven to eight weeks from sowing about 30 % of the foliage was affected in wild oat, 25 % in cv. Lustre and about 20 % in cv. Peniarth (Fig. 9). After this stage, because the lower heavily infected leaves senesced prematurely, the mean level of infection decreased due to lower levels of infection on the upper leaves especially in cvs Lustre and Peniarth. The upper leaves in all three lines appeared to express adult plant resistance (Fig. 9) .

3.2.2. Spore production on infected leaves

Spore production was measured on a different set of plants from those used for growth analysis because the measurements involved tissue destruction. For this experiment, twenty five seedlings of each line were raised singly in the growth room in 12.7 cm plastic pots containing S.A.I. potting compost. When 12 days old, the plants were transferred to the greenhouse and inoculated daily over a period of one week by

PLATE 2 :

Erysiphe graminis on the abaxial surface of a leaf of cv. Lustre (more than 75 % mildew cover).

PLATE 3 :

Erysiphe graminis on the abaxial surface of a leaf of wild oat (more than 75 % mildew cover).

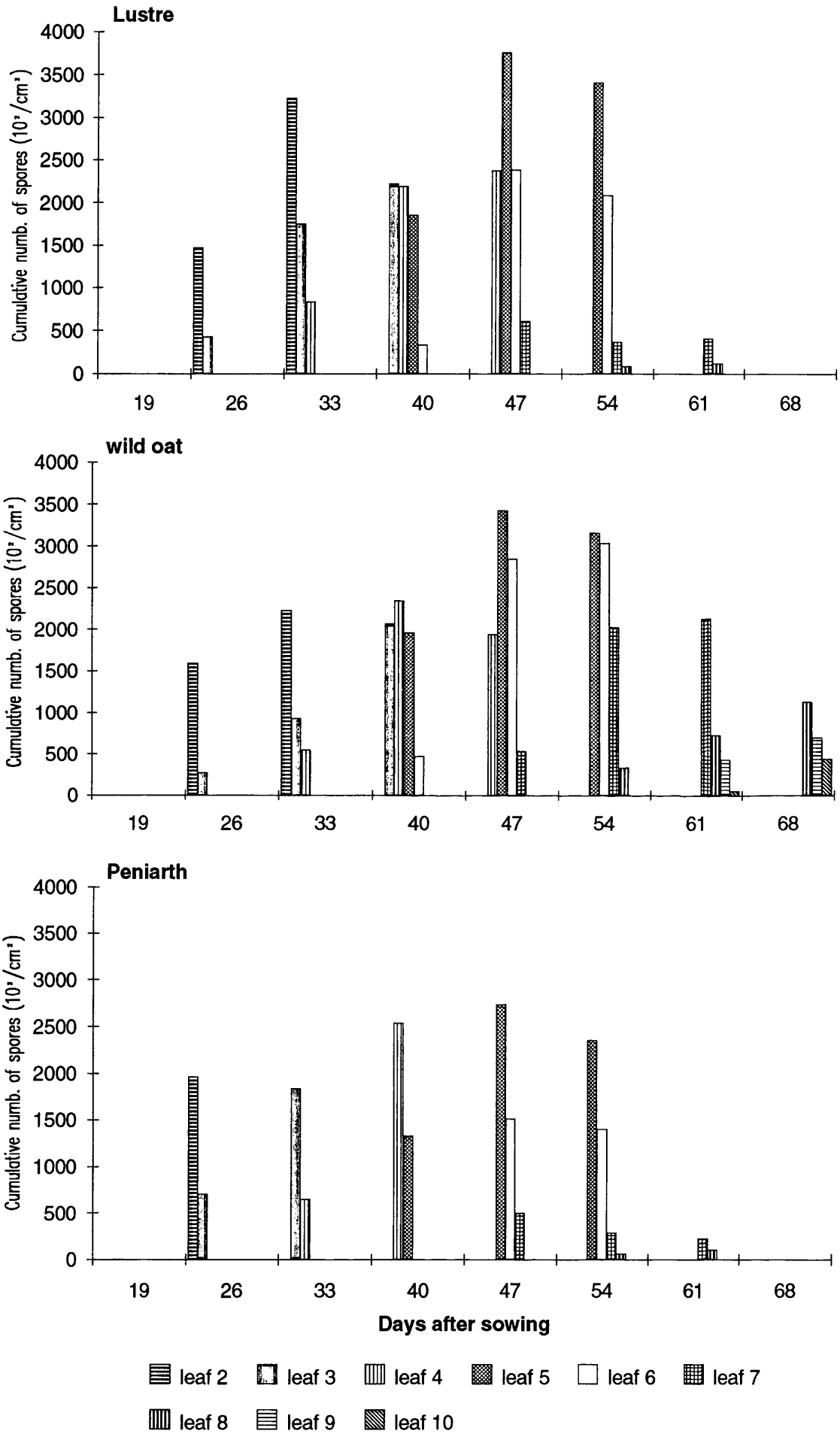


Fig. 10 : Spore production per unit area of individual leaf blades expanded on the main axis, in wild oat and cvs Lustre, Peniarth.

shaking spores from heavily infected plants over them. At each sampling time, the fully expanded leaf blades on the main axis of three plants were carefully excised and the numbers of spores present on each individual leaf blade were counted. The first sample was taken two weeks after the plants were first inoculated and subsequent samples were taken at weekly intervals. Seven counts were made in total. The results obtained for spore production per unit area of individual leaf blades along the main axis, of plants of each line, are plotted in Fig. 10 .

The cumulative number of spores calculated per unit area of each leaf on the main axis, at each sampling time, showed significant differences between leaves on each plant line and between the three plant lines. Spore production on the lower leaves of cv. Peniarth was higher than that on the corresponding leaves of cv. Lustre and wild oat, but thereafter, due to the effects of infection, the lower leaves became chlorotic and senesced more quickly on cv. Peniarth than on the other lines (Fig. 10). The youngest, uppermost, leaves of wild oat produced around 8 weeks after sowing were less infected than the older lower leaves. Thus wild oat appears to show some adult plant resistance, but at a late stage of infection. However, cvs Lustre and Peniarth showed much greater adult plant resistance since their upper leaves either did not become infected at all or were significantly less infected than the corresponding upper leaves in wild oat .

Spore production on all leaves on the main axis, at each sampling time, was calculated and the results are plotted in Fig. 11A. On both cultivars, the maximum number of spores was reached by about 47 days after sowing after which there appeared to be no further production whereas in wild oat, spore production continued to increase up to 68 days when the last sampling was made (Fig. 11A). The cumulative number of spores calculated for wild oat, at each sampling time, was significantly higher than that for cv. Peniarth whereas there were little differences between wild oat and cv. Lustre .

3.2.3. Total spore production per plant

Since mildew development appeared to follow the same pattern on the tillers as on the main axis, the total number of spores per plant could be estimated and the results are plotted graphically in Fig. 11B .

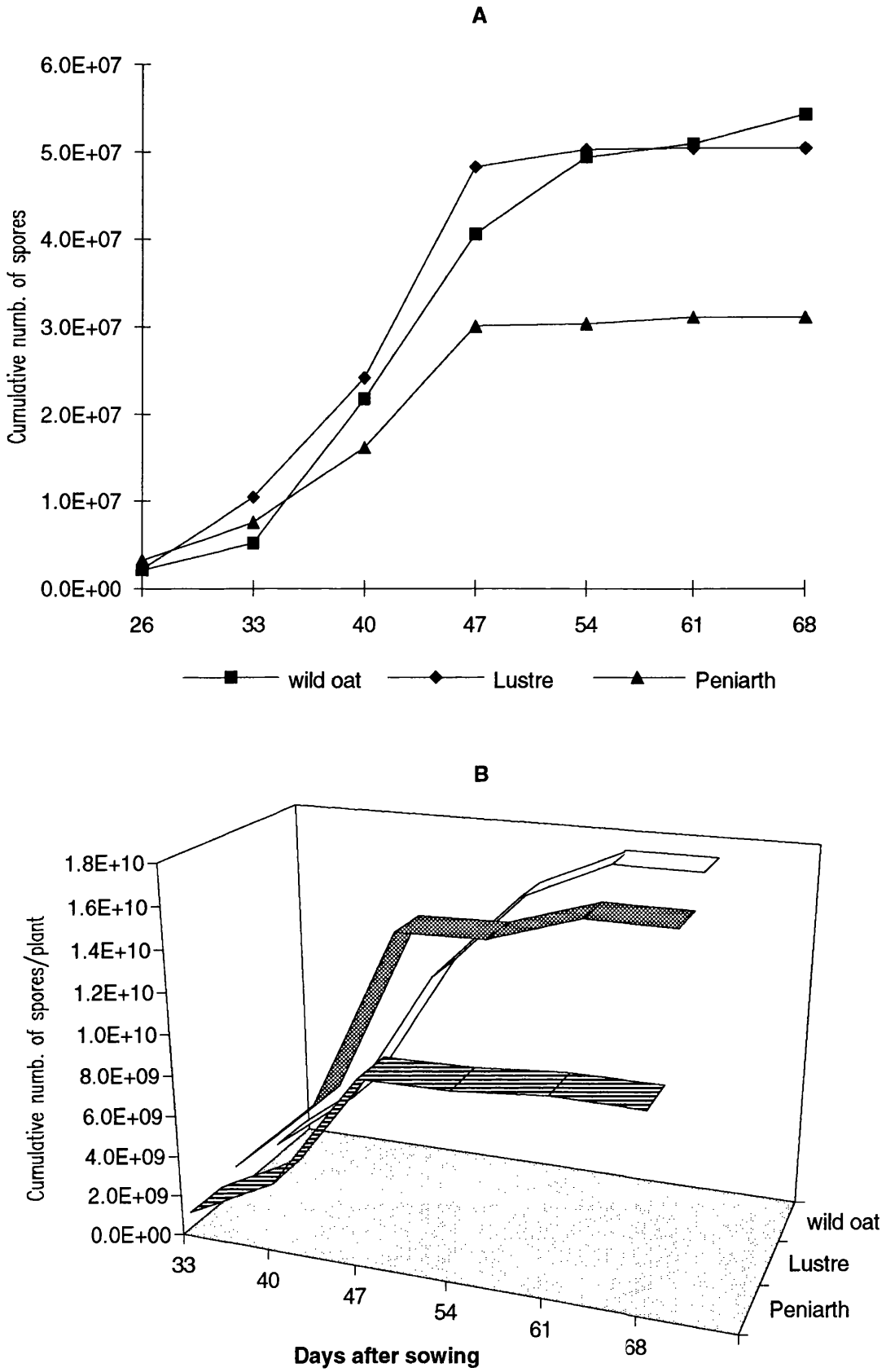


Fig. 11 : Spore production on all leaves expanded on the main axis (A) and total spore production per plant (B), in wild oat and cvs Lustre, Peniarth.
(6.0E+07 = 6×10^{7}, 1.8E+10 = 1.8×10^{10})

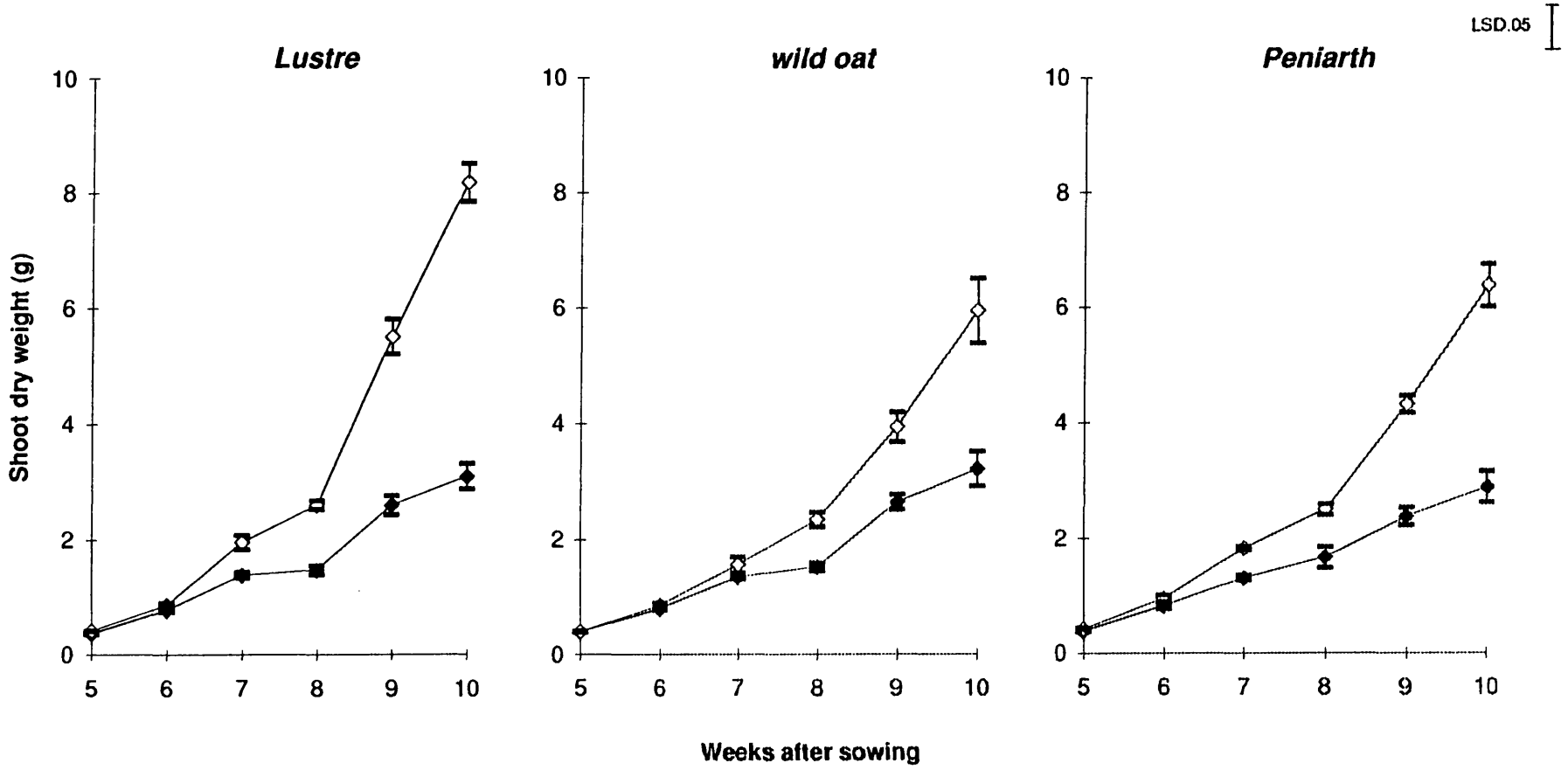


Fig. 12 : Mean shoot dry weights of Erysiphe graminis infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors

3.3. A comparison of the effects of infection on the growth and development of cvs Lustre, Peniarth and wild oat

Sixty plants of each line were raised singly in the growth room in 15 cm plastic pots containing coarse sand as described in Materials and Methods. When the third leaf blade had fully expanded, plants were transferred to the greenhouse and inoculated. Thirty plants of each oat line were inoculated daily over a period of one week. The other thirty plants were kept free from mildew by spraying with 0.1 % Patrol solution at weekly intervals. The treated and untreated plants were placed in a random arrangement on the same greenhouse bench and fertilized at weekly intervals. Four plants per treatment per line were sampled at each harvest. The first harvest was made two weeks after inoculation with subsequent harvests at weekly intervals until six harvests had been made. A seventh harvest of ten plants of each line was made when fruits were ripe for the measurement of grain yield .

The experiment was carried out twice. Since each experiment gave essentially the same results, the detailed results of one only are reported .

3.3.1. Effects of infection on dry matter production

Dry weights of individual organs and total dry weights of infected and uninfected plants, of each line, at each harvest, are given in Appendix tables 1 to 3 and the results are plotted in Figs. 12-14 .

Although shoot dry weight (Fig. 12) of infected plants was smaller than that of uninfected plants at each harvest, none of the differences became significant ($P < 0.05$) until the eighth week after sowing in the three lines, when mildew cover was about 25 % in cv. Lustre, about 18 % in cv. Peniarth and about 30 % in wild oat (Fig. 12). At this stage, the dry weight of shoots of infected plants increased significantly more slowly than in the controls, in all three lines, with the differences between infected and uninfected plants being more marked in the two cultivars than in wild oat as infection progressed (Fig. 12) .

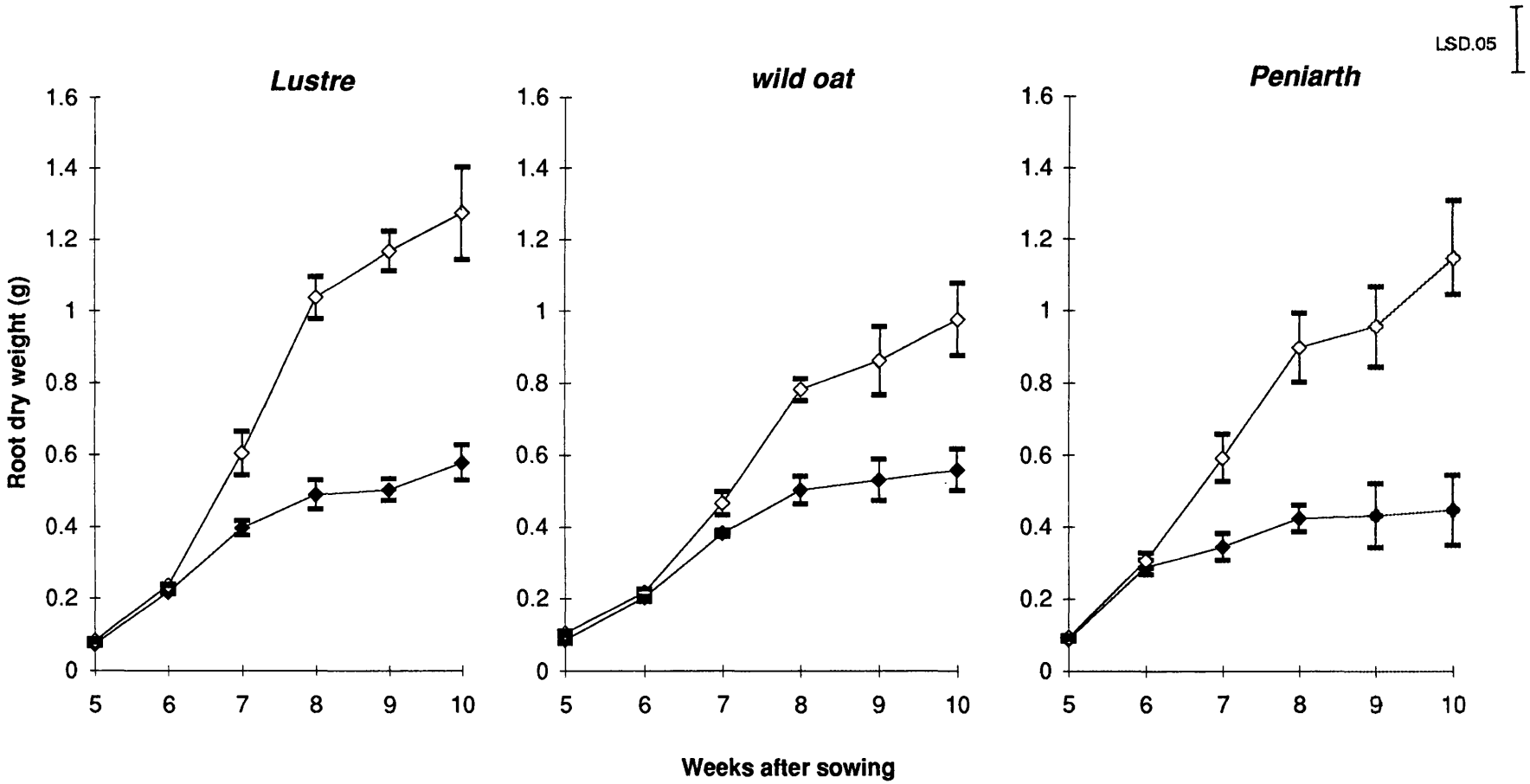


Fig. 13 : Mean root dry weights of Erysiphe graminis infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

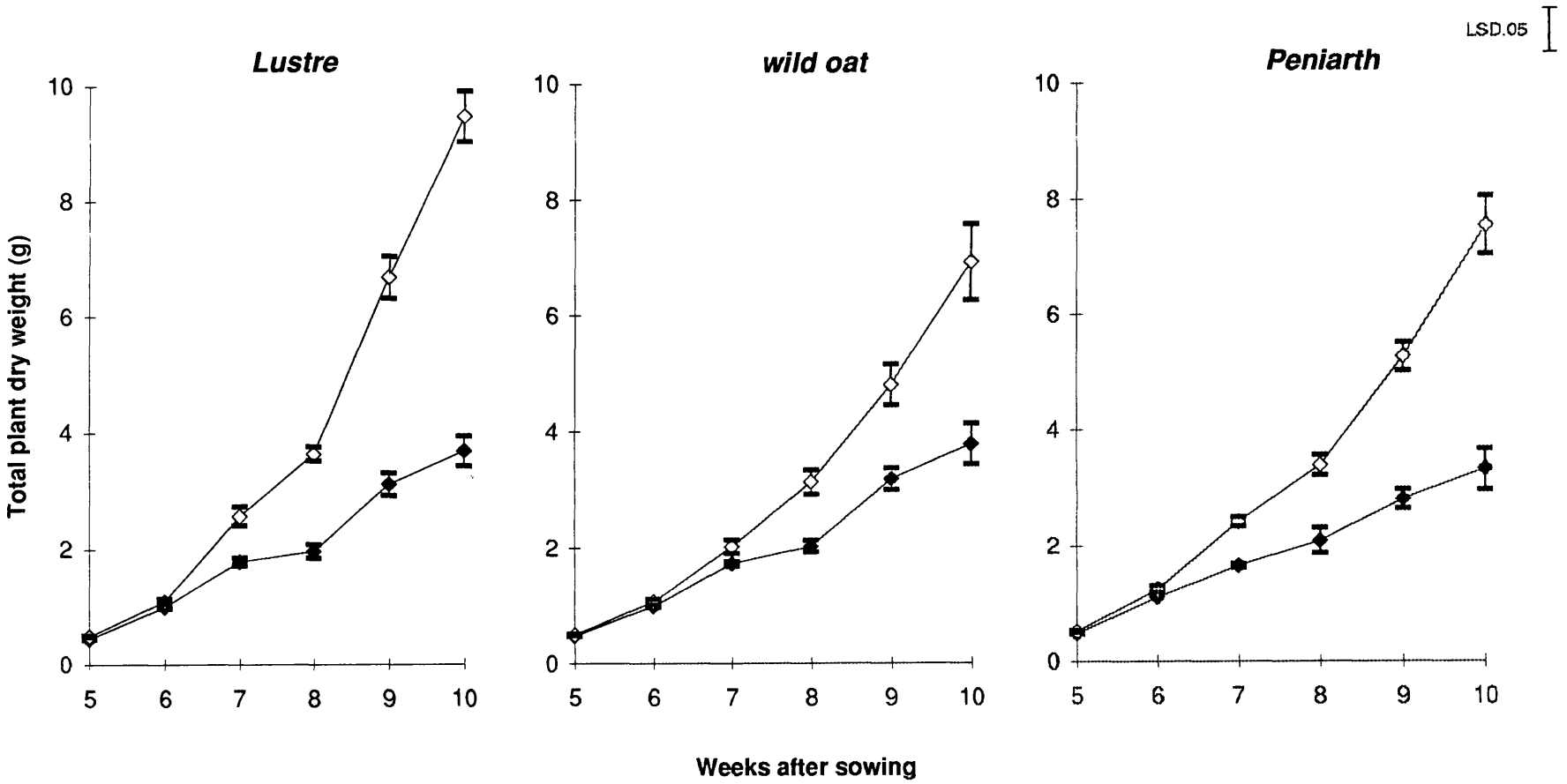


Fig. 14 : Mean total dry weights of Erysiphe graminis infected (◆) and uninfected (◇) plants of wild oat and vs Lustre, Peniarth. Vertical bars represent standard errors.

Infection also reduced root dry weight of the infected plants in all three lines (Fig. 13). Differences between infected and uninfected plants became significant ($P < 0.05$) 7 weeks after sowing in cvs Lustre and Peniarth when percentage mildew cover was about 20 % in cv. Peniarth, about 23 % in cv. Lustre but not until the eighth week after sowing in wild oat when percentage mildew cover was about 30 %, and the differences became more pronounced as infection progressed especially in cvs Lustre and Peniarth (Fig. 13) .

The total dry weights of infected plants were thus significantly reduced ($P < 0.05$) by 30 % in the two cultivars but by only 15 % in wild oat by the seventh week after sowing (Fig. 14). By the tenth week, the total dry weights of infected plants of cvs Lustre and Peniarth and wild oat showed a reduction of about 61 %, 55 % and 45 % respectively over that of uninfected plants. Thus reductions in dry weights were greater in cvs Lustre and Peniarth than in wild oat at each harvest .

3.3.2. Effects of infection on the patterns of plant development

3.3.2.1. Effects on shoot development

In both infected and uninfected plants of each line, change of the shoot apex from a vegetative to a reproductive state and thus the commencement of main axis elongation occurred around 30 days after sowing when the fifth leaf had emerged .

3.3.2.1.1. *Main axis height*

The main axis heights recorded at each harvest are given in Appendix tables 4 to 6, and plotted graphically in Fig. 15 .

The rate of elongation of the main axis of the uninfected plants, of each line, increased steadily up to the eighth week after sowing and then accelerated up to the tenth week. In the infected plants, the rate of elongation followed the same trend but the rates of extension were lower. However, the main axis of the infected plants of wild oat was taller than those of the infected plants of cvs Lustre and Peniarth at each harvest (Fig. 15). The differences between main axis heights of infected and uninfected plants

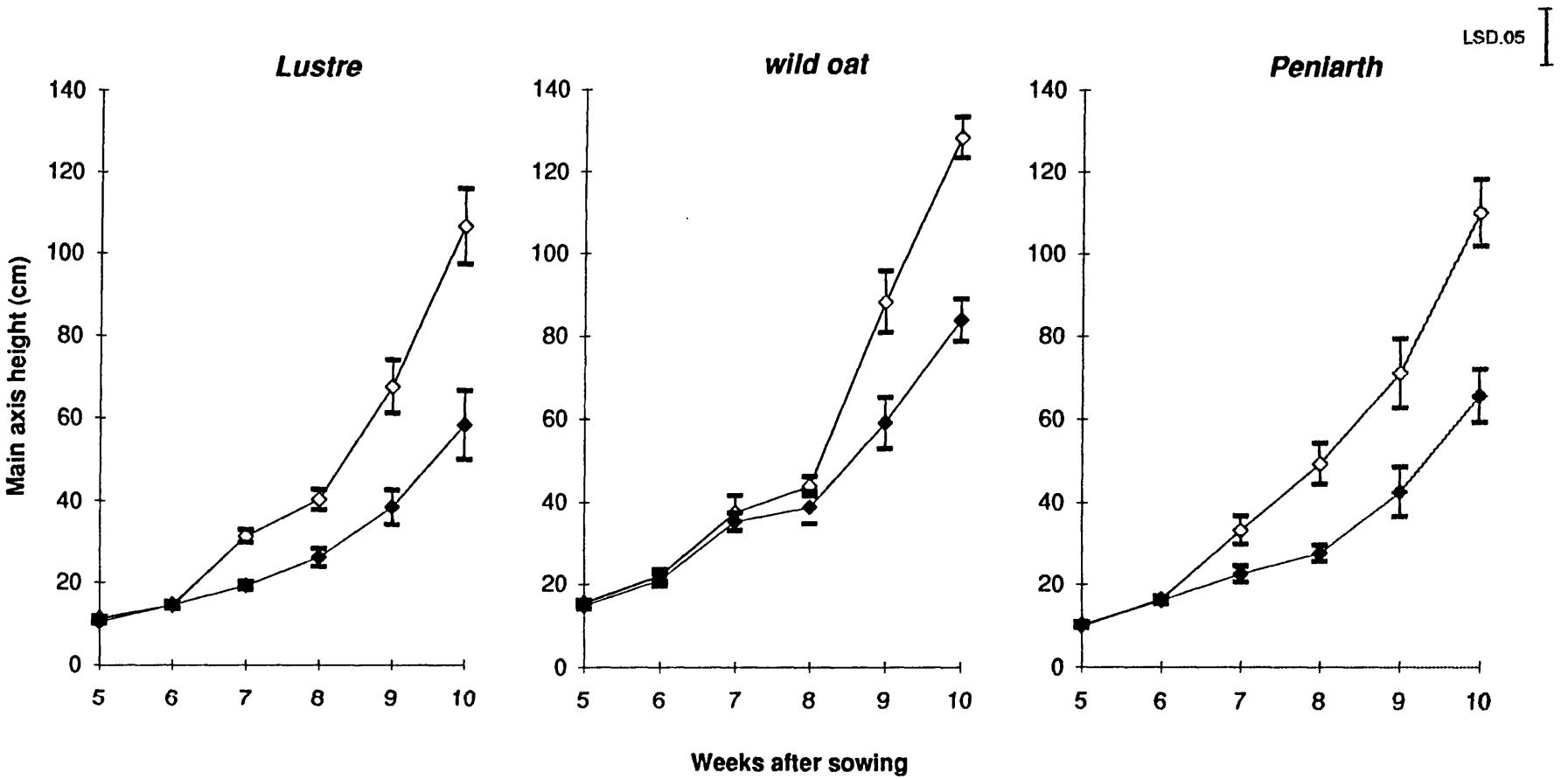


Fig. 15 : Mean main axis height of Erysiphe graminis infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

did not become significant ($P < 0.05$) until 8 weeks after sowing in cvs Lustre and Peniarth and a week later in wild oat .

3.3.2.1.2. *Number of leaves expanded on the main axis*

The mean total number of leaves (including green leaves and senescent leaves) and the mean number of senescent leaves along the main axis of infected and uninfected plants of each line, at each harvest, are recorded in Table 3 .

The infected and uninfected plants of all three lines had produced about 7 leaves along the main axis by the sixth week after sowing. At this stage percentage mildew cover was over 15 % in cv. Lustre and wild oat and over 10 % in cv. Peniarth. From the seventh week, additional leaves expanded earlier on the uninfected plants of cvs Lustre and Peniarth than on the infected plants. As a result of this, the uninfected plants of both cultivars completed leaf expansion on the main axis earlier than the infected plants and in fact, in cv. Peniarth the infected plants expanded one leaf fewer than the control plants (Table 3). There were, however, no such differences in the rate of leaf expansion on plants of wild oat at any harvest .

The first senescent leaves were observed in infected plants of cv. Peniarth five weeks after sowing, and a week later in infected plants of cv. Lustre and wild oat. The rate of senescence of infected leaves increased with infection, especially in cvs Lustre and Peniarth, and as a result of this, infected plants of wild oat had a higher number of non-senescent leaves than those of the two cultivars (Table 3) .

3.3.2.1.3. *Green leaf blade area on the main axis*

Total green leaf blade areas along the main axis of infected and uninfected plants of each line, together with areas of individual leaf baldes at each harvest, are given in Appendix tables 1 to 3 and 4 to 6 respectively. The areas of the individual green leaf blades are presented as histograms in Figs. 16A-F (in the appendix) and total leaf blade areas are plotted in Fig. 17 .

The reductions in sizes of the individual green leaf blade areas appeared to vary according to the level of infection on the leaf, the areas of leaf blades at the lower

TABLE 3

Leaf development on the main axis of Erysiphe graminis f.sp. avenae infected (I) and uninfected (C) plants of the three oat plant lines taken at weekly intervals. Each reading is a mean of four replicates ± SE.

Harvest		Total No of leaves			No of senescent leaves		
		Lustre	wild oat	Peniarth	Lustre	wild oat	Peniarth
Ist	I	5.0 ± 0.0	5.0 ± 0.0	5.0 ± 0.0	-	-	1.00 ± 0.00
	C	5.0 ± 0.0	5.0 ± 0.0	5.0 ± 0.0	-	-	-
2nd	I	7.0 ± 0.0	7.0 ± 0.0	7.0 ± 0.0	2.00 ± 0.00	2.00 ± 0.00	3.00 ± 0.00
	C	7.0 ± 0.0	7.0 ± 0.0	7.0 ± 0.0	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00
3rd	I	8.0 ± 0.0	8.0 ± 0.0	8.0 ± 0.0	4.00 ± 0.00	3.25 ± 0.22	4.25 ± 0.22
	C	9.0 ± 0.0	8.0 ± 0.0	9.0 ± 0.0	2.00 ± 0.00	1.00 ± 0.00	2.00 ± 0.00
4th	I	9.0 ± 0.0	9.0 ± 0.0	9.0 ± 0.0	5.00 ± 0.00	3.50 ± 0.25	5.25 ± 0.22
	C	10.0 ± 0.0	9.0 ± 0.0	10.0 ± 0.0	2.00 ± 0.00	1.75 ± 0.22	2.50 ± 0.25
5th	I	10.0 ± 0.0	10.0 ± 0.0	10.0 ± 0.0	5.00 ± 0.00	4.25 ± 0.22	6.00 ± 0.00
	C	11.0 ± 0.0	10.0 ± 0.0	11.0 ± 0.0	3.00 ± 0.00	2.50 ± 0.25	3.50 ± 0.25
6th	I	11.0 ± 0.0	10.0 ± 0.0	10.0 ± 0.0	6.25 ± 0.22	4.50 ± 0.25	6.00 ± 0.00
	C	11.0 ± 0.0	10.0 ± 0.0	11.0 ± 0.0	3.50 ± 0.25	3.00 ± 0.00	4.00 ± 0.00

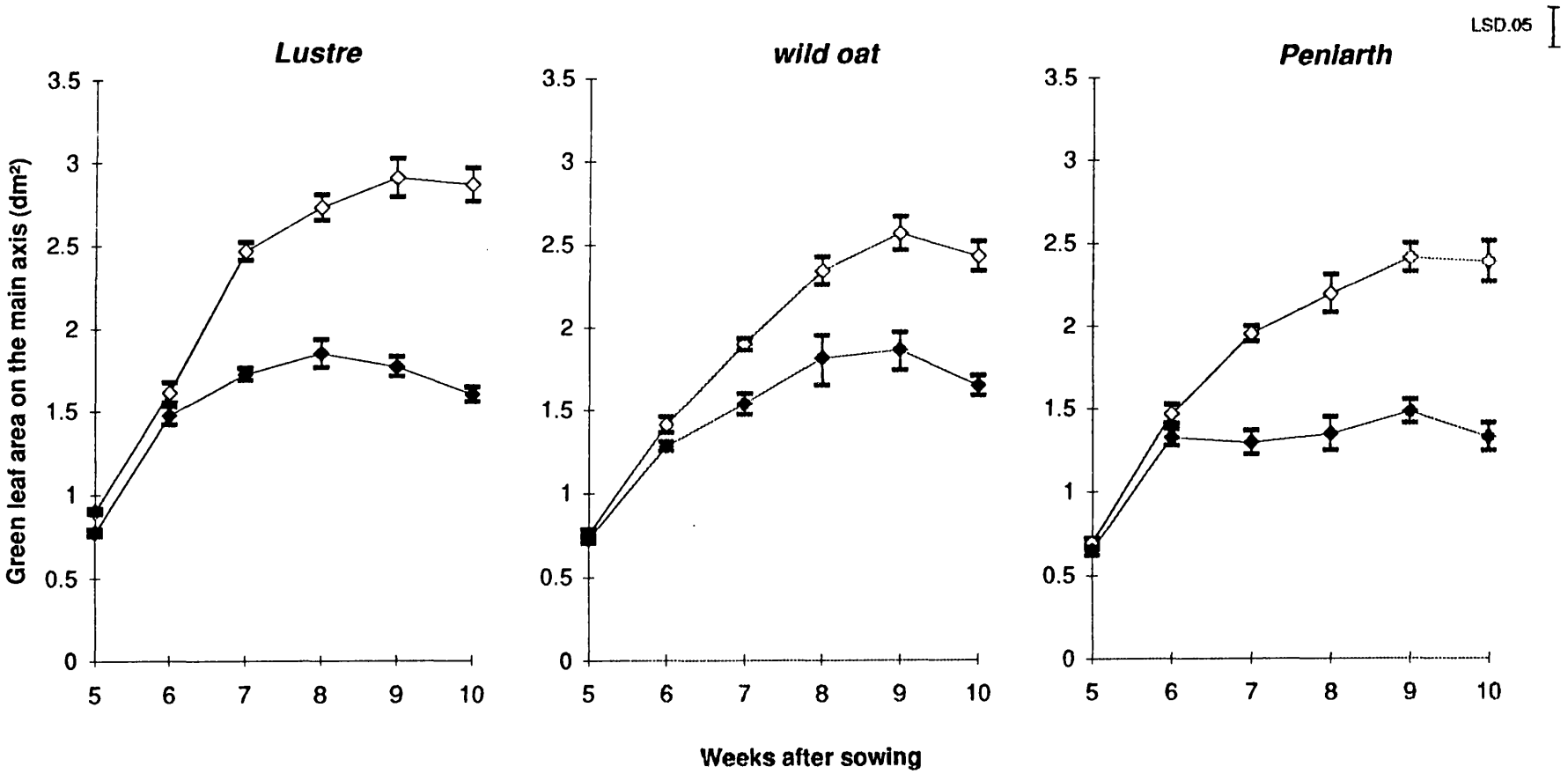


Fig. 17 : Mean total green leaf area of leaves, on the main axis, of infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Penlarth. Vertical bars represent standard errors.

positions being more heavily infected and more reduced than those of leaf blades at higher positions on the main axis in each line (Figs. 16A-F). The major difference between infected and uninfected plants of each line, at different harvests, was the smaller size of the infected green leaf blades. However, even the apparently uninfected or slightly infected upper leaf blades (about 5 % or less mildew cover) on the infected plants were significantly reduced in size .

As a consequence of the reductions in size of the individual green leaf blades, total leaf blade areas were significantly reduced ($P < 0.05$) in each line (Fig. 17). Total green leaf blade areas of uninfected plants of each line increased throughout the period of investigation, but those of infected plants increased slowly up to the eighth week after sowing in both cv. Lustre and wild oat, but then they declined when total mildew cover was about 25 % and in cv. Lustre and about 30 % in wild oat. However, in cv. Peniarth, there was no further increase in total leaf blade area after the sixth week after sowing when only about 12 % of the foliage was affected. The differences between infected and uninfected plants of each line were not significant until the seventh week after sowing, and 3 weeks after where a greater reduction was produced, especially in cvs Lustre and Peniarth (Fig. 17) .

3.3.2.1.4. *Number of Tillers*

The numbers of tillers recorded at each harvest are given in Appendix tables 4 to 6 and the results plotted in Fig. 18 .

The first tillers were formed before the first harvest 5 weeks after sowing. In the control plants, numbers continued to increase up to about 4.50 tillers in wild oat, about 4.75 tillers in cv. Lustre and about 5.5 tillers per plant in cv. Peniarth, reached at the ninth week after sowing, but in the infected plants there was no further increase after the seventh week when there were about 2.75 tillers in both wild oat and cv. Lustre and about 3.25 tillers per plant in cv. Peniarth (Fig. 18). The LSD for tiller number per plant shows significant differences ($P < 0.05$) between infected and uninfected plants of each line. These differences were evident at the seventh week after sowing in both cvs Peniarth and Lustre, when percentage mildew cover was over 20 %

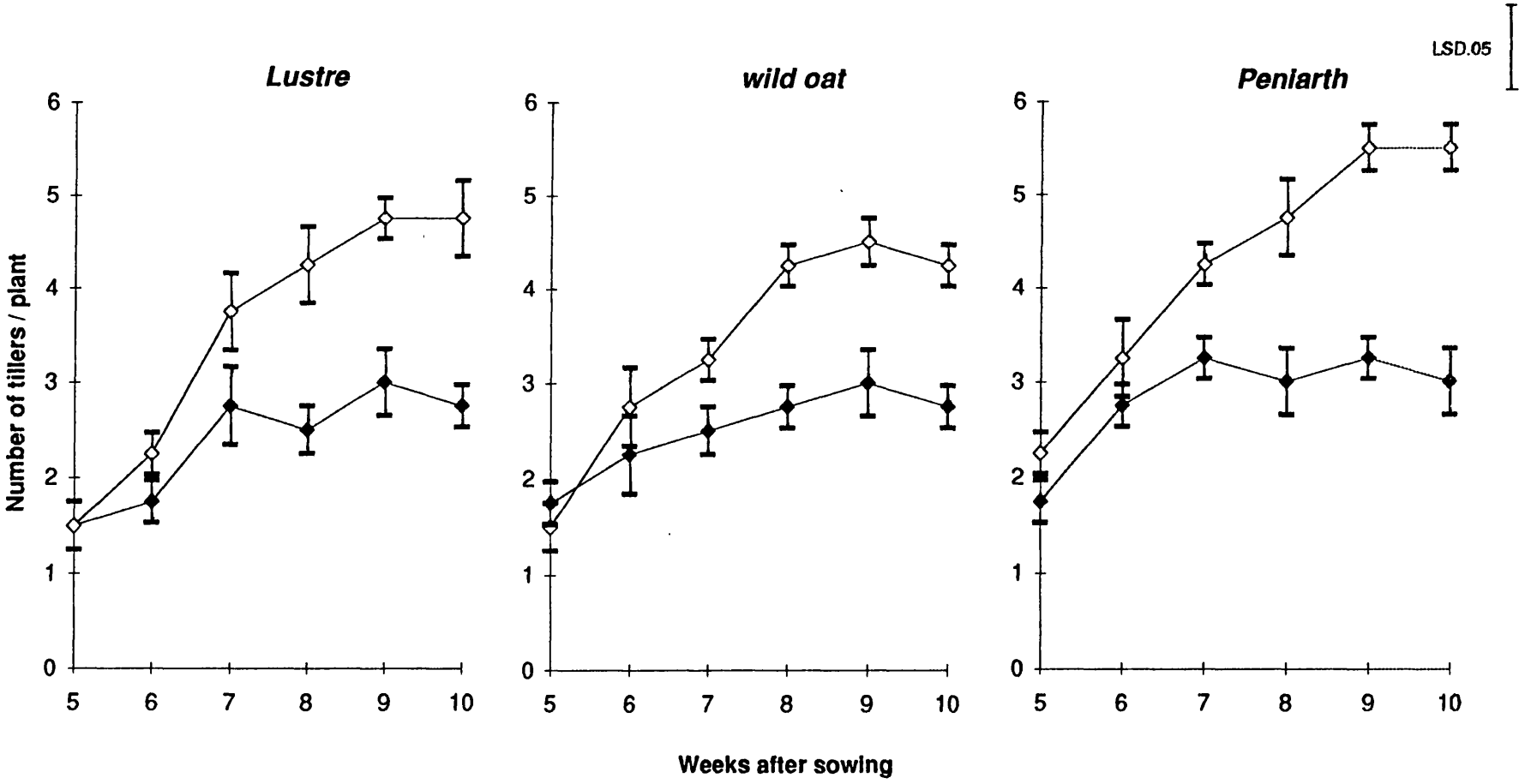


Fig. 18 : Mean number of tillers produced in infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

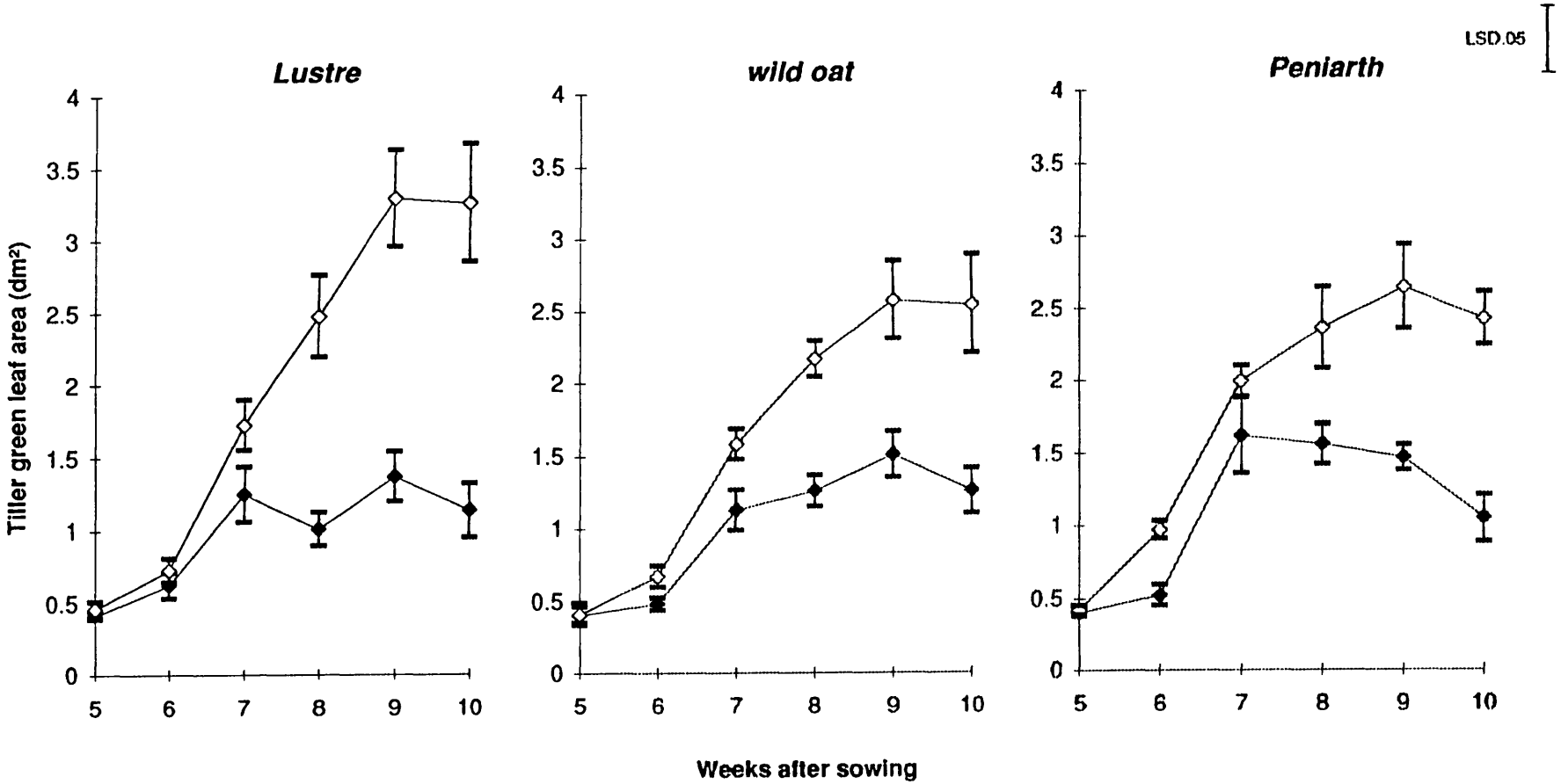


Fig. 19 : Mean total green leaf area of leaves, on tillers, of infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

and 23 % respectively, and a week later in wild oat, when percentage mildew cover was about 30 % .

3.3.2.1.5. *Green leaf blade area on tillers*

Mean total green leaf blade areas of tillers of plants at each harvest are given in Appendix tables 4 to 6 and the results are plotted graphically in Fig. 19 .

The reduction in tiller green leaf blade area on the infected plants of each oat line was due to a reduction in the number of tillers produced per plant. The reduction in tiller green leaf blade area followed the same pattern as that on the main axis, the only difference being that tiller green leaf blades were more reduced in area than were green leaf blades on the main axis. The analysis of variance shows significant differences ($P < 0.05$) between infected and uninfected plants which were apparent around the seventh week after sowing in all three lines with the differences being greatest in cv. Lustre (Fig. 19) .

3.3.2.1.6. *Cell size and cell number per leaf*

Since the reduction in green leaf blade area could be due to reductions in cell expansion and/or in cell division, attempts were made to estimate cell size and cell number per leaf. Three leaves from position 1 and two from position 3, on the main axis, showing about 30 %, 40% and 75 % mildew cover on the leaves from position 1, 50 % and 80 % mildew cover on the leaves from position 3, were excised from five infected plants of each of the two lines. Leaves from corresponding positions were excised from five uninfected plants. The area of each leaf was measured with the leaf area meter and lower epidermal imprints were obtained (see Materials and Methods, section 2.10.1.). The number of stomata (guard cells and subsidiary cells) and the epidermal cells other than stomata, per unit area were determined microscopically by counting the number per field of view (0.332 mm^2) at a magnification of x 200. Five counts per strip were made and the mean number of stomata and of other epidermal cells per unit area was calculated. A total of ten epidermal imprints per leaf were assessed. The results are given in Tables 4 to 6 and are illustrated in Plates 4 and 5 .

The results show that the number of stomata and the non stomatal cells, per unit area (0.332 mm^2) was always higher in the infected leaves than in the corresponding uninfected leaves in both cv. Peniarth and wild oat (Tables 4 and 5). The mean number of stomata per field of view was about 11.9 in infected leaves and 10.7 in uninfected leaves of cv. Peniarth, 12.0 in infected leaves and 10.5 in uninfected leaves of wild oat ; while the mean number of the other epidermal cells was about 31.6 in infected leaves and 29.2 in uninfected leaves of cv. Peniarth, 30.4 in infected leaves and 28.4 in uninfected leaves of wild oat. The analysis of variance shows significant differences ($P < 0.05$) in the number of stomata and the non stomatal epidermal cells per unit area between leaves with different mildew intensities in each line (Table 6A), but there were no significant differences between the uninfected leaves except for the number of non stomatal epidermal cells which was found to be significantly different in each line and this was probably due to differences in leaf position (Table 6B). The analysis of variance also shows significant differences ($P < 0.05$) between infected and uninfected leaves in each line (Table 6C) .

Since the ratio between numbers of stomata and epidermal cells per field of view (stomatal index) was not altered by infection in either line (Table 7a-7b), it is clear that the higher number of stomata per field of view for the infected leaves is due to the reduced expansion of the epidermal cells with the relative increase in number indicating the extent of this reduction in both lines. Counts of stomata and of the other epidermal cells per unit leaf area, together with measurements of total leaf area, were used to calculate the approximate numbers of epidermal cells per leaf (Table 8a-8b). The values found indicate that there were between 4 to 13 % fewer stomata and epidermal cells in the lower epidermis of infected than uninfected leaves of wild oat (Table 8a), and between 5 to 17 % fewer stomata and epidermal cells in the lower epidermis of infected than uninfected leaves of cv. Peniarth (Table 8b). Thus, the reduction in leaf blade area, which ranged from 8 to 23 % in wild oat and from 9 to 29 % in cv. Peniarth, was due to reduced cell division as well as reduced cell expansion .

TABLE 4

a) The effects of infection on the number of stomata (guard cells and subsidiary cells) per unit area of infected and uninfected lower epidermis of leaf tissue of wild oat.

	Infected					**Control**				
Leaf position	**1**			**3**		**1**			**3**	
Mean L.a. (cm^2)	11	12	10	26	23	12	13	13	28	30
% Infection	30	40	75	50	80	0	0	0	0	0
	Number of stomata per unit area					Number of stomata per unit area				
	10	13	11	15	17	10	11	13	14	13
	11	12	12	14	14	09	12	10	10	10
	10	10	14	11	12	09	10	10	11	13
	09	10	12	12	14	10	11	10	10	12
	11	10	10	10	13	11	12	09	09	11
	09	12	15	11	16	11	09	09	11	09
	09	08	12	17	12	08	09	09	10	10
	11	09	09	11	15	09	12	12	09	13
	09	10	14	13	12	11	10	11	10	12
	13	12	12	14	16	10	08	11	12	10
Mean	10.2	10.6	12.1	12.8	14.1	9.8	10.4	10.4	10.6	11.3
Std	1.25	1.50	1.76	2.09	1.76	0.98	1.36	1.28	1.43	1.42

b) The effects of infection on the number of the epidermal cells, other than stomata, per unit area of infected and uninfected lower epidermis of leaf tissue of wild oat.

	Infected					**Control**				
Leaf position	**1**			**3**		**1**			**3**	
Mean L.a. (cm^2)	11	12	10	26	23	12	13	13	28	30
% Infection	30	40	75	50	80	0	0	0	0	0
	Number of epidermal cells per unit area					Number of epidermal cells per unit area				
	30	31	28	29	36	27	28	30	29	30
	30	29	30	32	33	26	27	26	29	27
	29	28	33	30	31	26	26	27	31	30
	28	28	32	32	31	28	27	28	28	29
	28	27	29	29	34	30	29	26	28	26
	29	29	33	31	36	28	28	28	31	26
	28	26	31	36	32	26	27	26	29	28
	31	26	29	27	35	27	31	27	30	31
	27	28	32	30	32	28	26	28	29	27
	31	29	30	31	37	31	26	28	31	27
Mean	29.1	28.1	30.7	30.7	33.7	27.7	27.5	27.4	29.5	28.1
Std	1.30	0.94	1.68	2.28	2.10	1.62	1.50	1.20	1.12	1.70

TABLE 5

a) The effects of infection on the number of stomata (guard cells and subsidiary cells) per unit area of infected and uninfected lower epidermis of leaf tissue of cv. Peniarth.

	Infected					**Control**				
Leaf position	**1**			**3**		**1**			**3**	
Mean L.a. (cm^2)	10	10	8.5	22	18	11	12	12	25	24
% Infection	30	40	75	50	80	0	0	0	0	0
	Number of stomata per unit area					Number of stomata per unit area				
	12	15	15	10	15	10	13	14	12	13
	09	13	12	14	12	09	10	10	11	10
	11	10	12	13	14	11	12	10	12	11
	09	12	14	10	16	09	10	13	10	11
	12	11	16	12	10	10	09	09	13	12
	11	12	14	12	17	10	12	10	09	09
	10	10	10	12	11	12	10	09	10	11
	12	09	09	09	11	10	11	09	11	12
	10	10	12	14	14	10	09	11	09	10
	12	10	12	11	12	11	11	10	13	10
Mean	10.8	11.2	12.6	11.7	13.2	10.2	10.7	10.5	11.0	10.9
Std	1.17	1.72	2.06	1.62	2.23	0.87	1.27	1.63	1.41	1.14

b) The effects of infection on the number of the epidermal cells, other than stomata, per unit area of infected and uninfected lower epidermis of leaf tissue of cv. Peniarth.

	Infected					**Control**				
Leaf position	**1**			**3**		**1**			**3**	
Mean L.a. (cm^2)	10	10	8.5	22	18	11	12	12	25	24
% Infection	30	40	75	50	80	0	0	0	0	0
	Number of epidermal cells per unit area					Number of epidermal cells per unit area				
	30	30	29	30	36	30	31	31	31	32
	27	33	30	31	31	26	30	30	28	27
	28	32	33	35	34	28	34	28	32	29
	28	32	34	29	36	28	28	31	28	28
	32	30	36	33	30	29	30	27	34	31
	30	34	34	32	35	29	32	30	27	26
	27	29	31	33	29	28	30	28	28	30
	29	28	30	28	29	28	30	28	32	32
	29	35	35	32	34	26	28	29	26	27
	30	31	33	30	32	29	29	28	32	27
Mean	29.0	31.4	33.5	31.3	32.7	28.1	30.2	29.0	29.8	28.9
Std	1.48	1.56	2.66	2.00	2.61	1.22	2.01	1.34	2.56	2.12

TABLE 6

A : Analyses of variance

(1) *Number of stomata per unit area of infected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	0.160	0.160	0.05	NS
Infection	4	126.260	31.565	9.63	0.001
Plant line x Infection	4	15.940	5.985	1.83	0.1
Residual	90	295.000	3.278		
Total	99	437.360			

* $LSD_{.05}$ = 1.61

(2) *Number of the other epidermal cells per unit area of infected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	17.640	17.640	4.68	0.01
Infection	4	240.060	60.040	15.92	0.001
Plant line x Infection	4	59.660	14.915	3.96	0.01
Residual	90	339.200	3.769		
Total	99	656.560			

* $LSD_{.05}$ = 1.72

* LSD values for plant line x infection interactions.

B : Analyses of variance

(1) *Number of stomata per unit area of uninfected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	0.640	0.640	0.35	NS
Infection	4	8.960	2.240	1.21	NS
Plant line x Infection	4	6.360	1.590	0.86	NS
Residual	90	166.400	1.849		
Total	99	182.360			

* $LSD_{.05} = 0.85$

(2) *Number of the other epidermal cells per unit area of uninfected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	32.490	32.490	9.63	0.001
Infection	4	37.300	9.325	2.76	0.05
Plant line x Infection	4	27.260	6.815	2.02	0.1
Residual	90	303.699	3.374		
Total	99	400.750			

* $LSD_{.05} = 1.63$

* LSD values for plant line x infection interactions.

C : Analyses of variance

(1) *Number of stomata per unit area of infected and uninfected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	0.125	0.125	0.04	NS
Infection	1	91.125	91.125	28.34	0.001
Plant line x Infection	1	0.605	0.605	0.19	NS
Residual	196	630.139	3.215		
Total	199	721.995			

* $LSD_{.05} = 0.71$

(2) *Number of the other epidermal cells per unit area of infected and uninfected leaf tissue*

Source of variation	df	SS	MS	VR	P
Plant line	1	53.045	53.045	10.55	0.001
Infection	1	262.205	262.205	52.13	0.001
Plant line x Infection	1	0.845	0.845	0.17	NS
Residual	196	985.859	5.030		
Total	199	1301.955			

* $LSD_{.05} = 0.89$

* LSD values for plant line x infection interactions.

TABLE 7

Effects of infection on Stomatal Index in infected and uninfected lower epidermis of leaves of :

a) wild oat

Leaf position on main axis	Stomatal Index			
	Infected plants		Control plants	
	Mean	SE	Mean	SE
1	0.26	0.017	0.26	0.012
1	0.27	0.019	0.27	0.019
1	0.28	0.019	0.27	0.020
3	0.29	0.026	0.26	0.026
3	0.29	0.018	0.29	0.018

b) cv. Peniarth

Leaf position on main axis	Stomatal Index			
	Infected plants		Control plants	
	Mean	SE	Mean	SE
1	0.27	0.016	0.27	0.017
1	0.26	0.029	0.26	0.016
1	0.28	0.031	0.27	0.024
3	0.27	0.021	0.27	0.011
3	0.29	0.016	0.27	0.010

TABLE 8

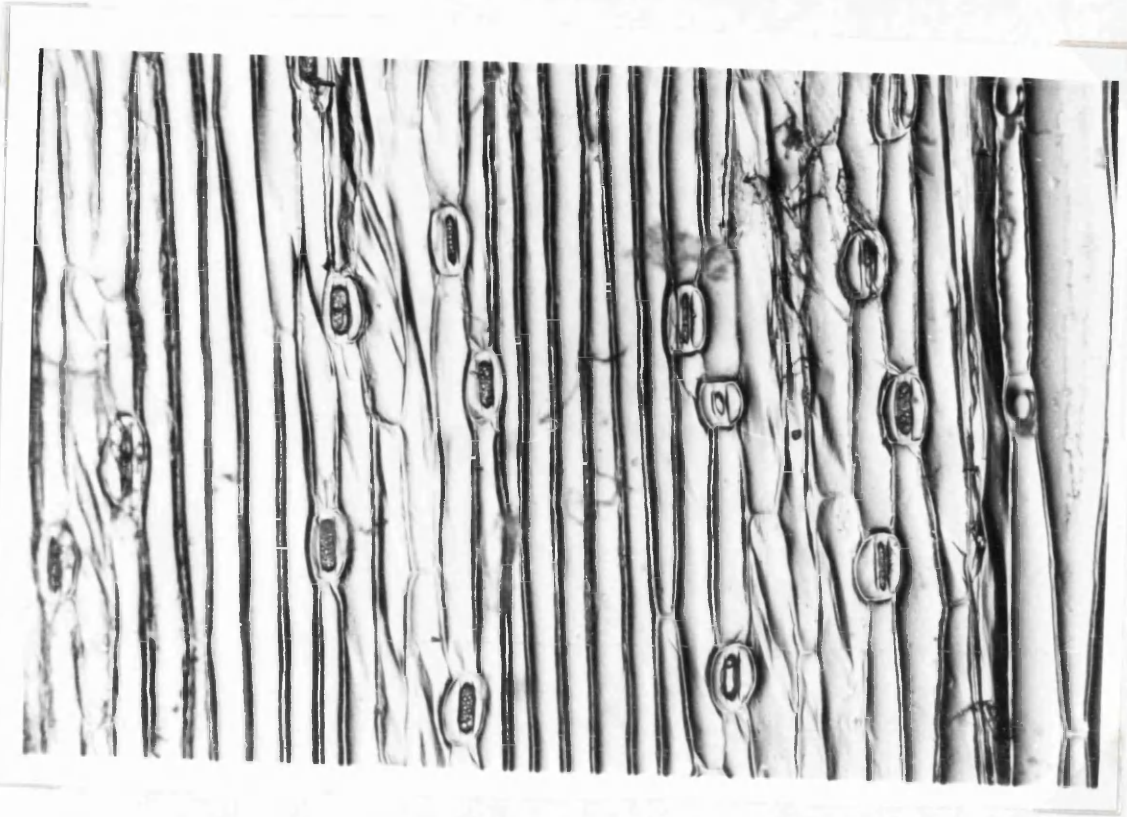
Effects of infection on total number of stomata and the other epidermal cells per lower epidermis of leaves of :

a) wild oat

Leaf position on main axis	Infected plants			Control plants			Percentage reduction	
	Leaf area (cm^2)	Number of stomata	Number of epiderm. cells	Leaf area (cm^2)	Number of stomata	Number of epiderm. cells	Leaf area	stomata + epider. cells
1	11.0	338.30	965.14	12.0	354.58	1002.22	8.33	3.90
1	12.0	383.52	1016.70	13.0	407.64	1077.90	7.69	5.74
1	10.0	364.83	925.64	13.0	407.64	1070.06	23.08	12.67
3	26.0	1003.43	2406.67	28.0	894.89	2490.49	7.15	-
3	23.0	957.80	2337.02	30.0	1022.12	2541.74	23.33	7.55

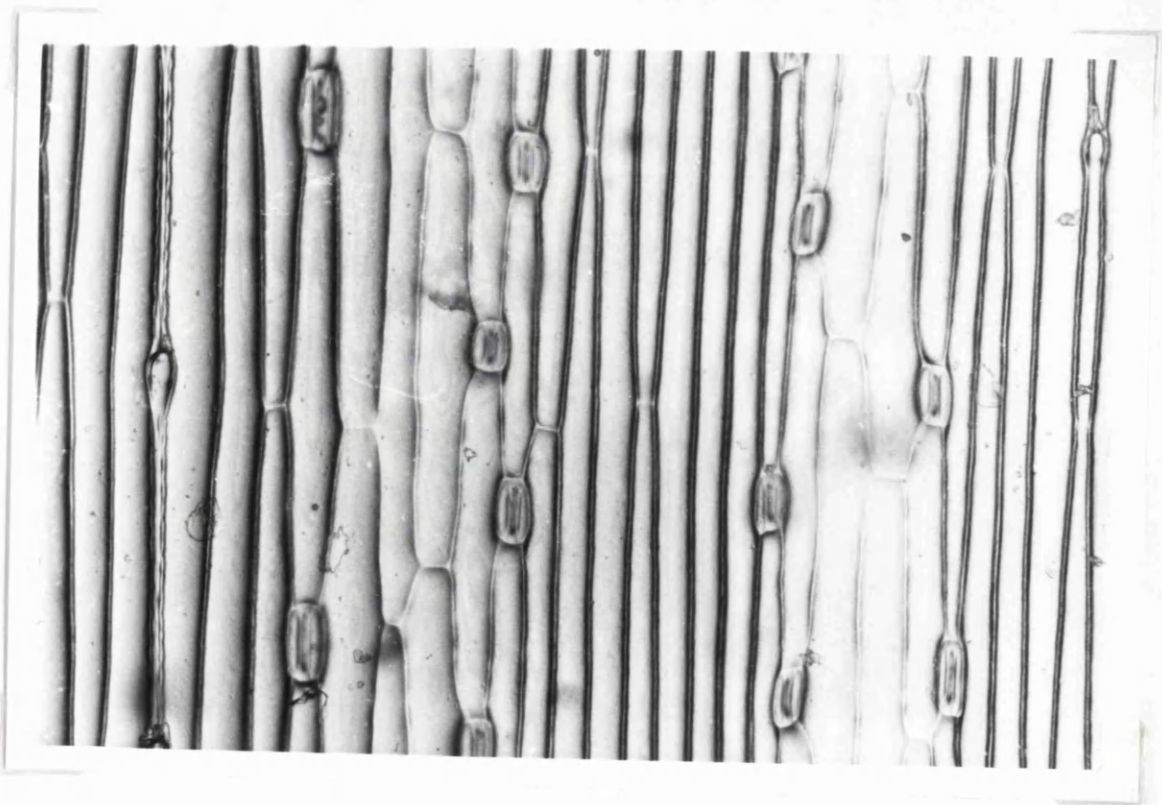
b) cv. Peniarth

Leaf position on main axis	Infected plants			Control plants			Percentage reduction	
	Leaf area (cm^2)	Number of stomata	Number of epiderm. cells	Leaf area (cm^2)	Number of stomata	Number of epiderm. cells	Leaf area	stomata + epider. cells
1	10.0	325.63	874.38	11.0	338.30	928.66	9.10	5.28
1	10.0	337.69	946.75	12.0	387.14	1092.68	16.67	13.20
1	8.5	322.92	858.56	12.0	379.90	1049.26	29.17	17.33
3	22.0	776.09	2076.21	25.0	829.16	2246.26	12.00	7.25
3	18.0	716.39	1774.70	24.0	788.75	2081.29	25.00	13.20

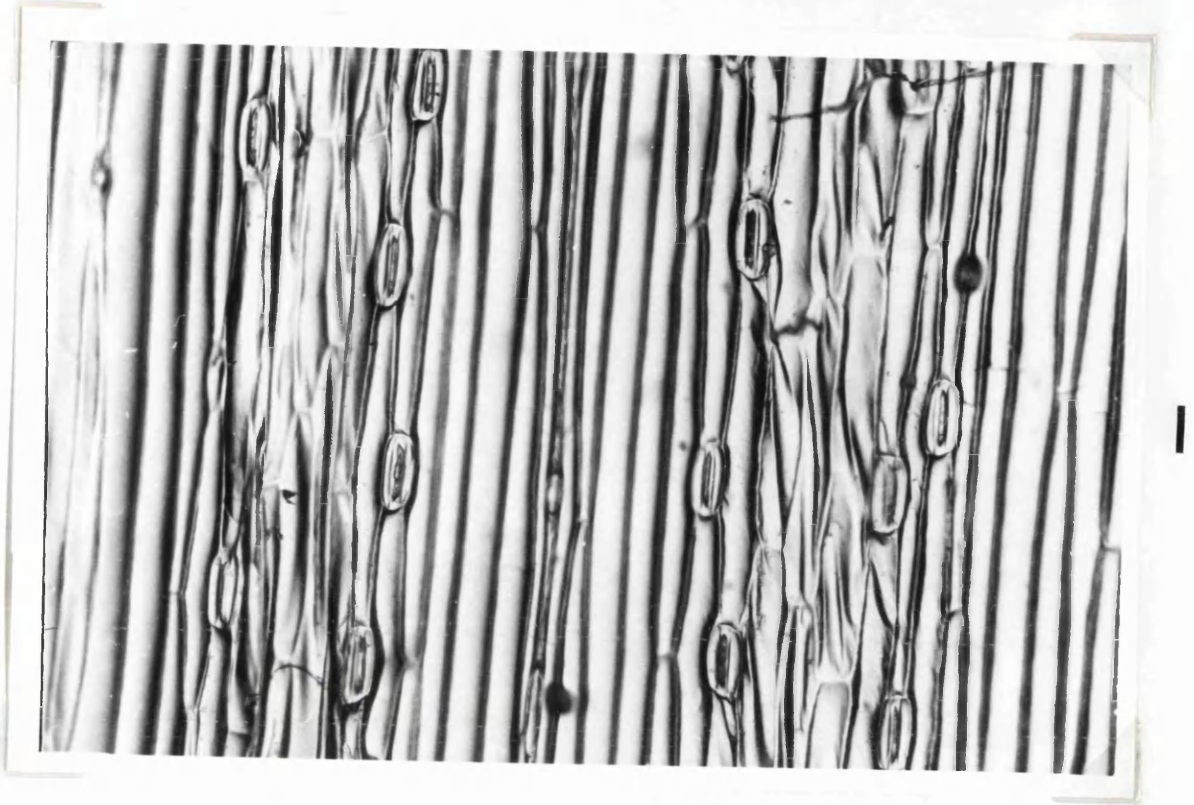


I

PLATE 4 : Photomicrographs of the lower epidermis of wild oat infected (I) and uninfected (C) with Erysiphe graminis.

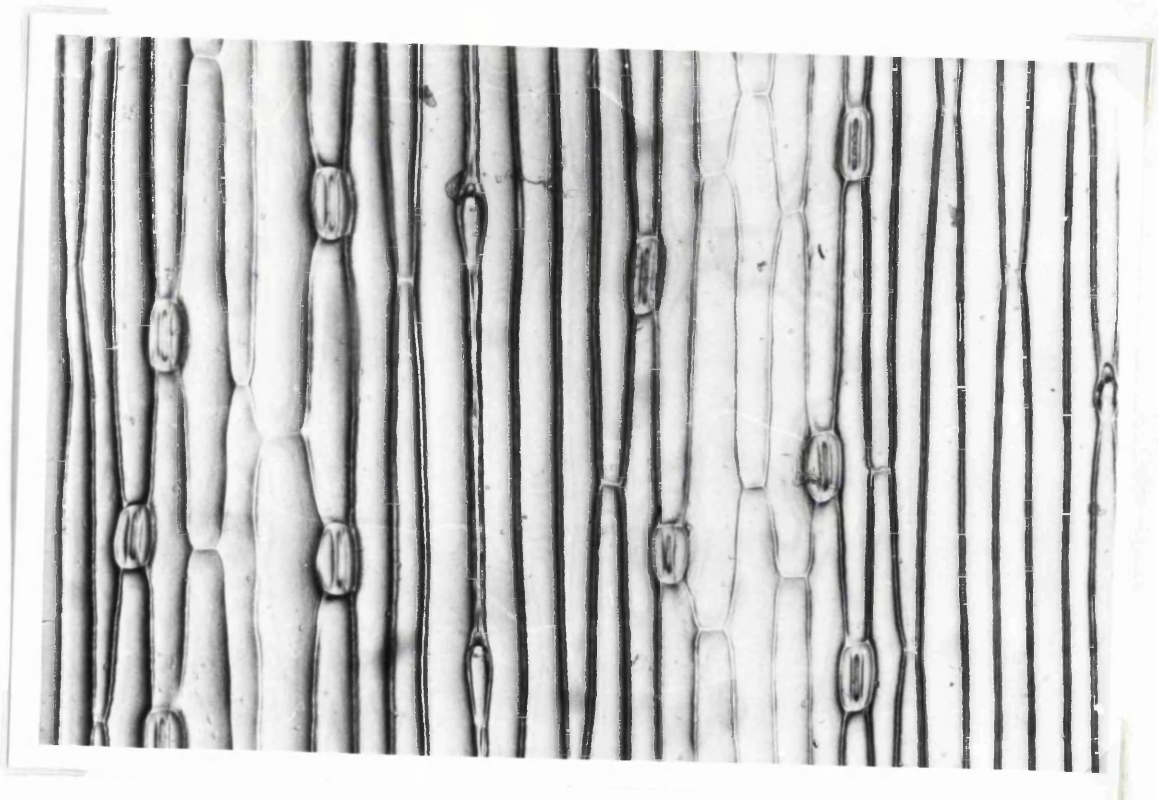


C



I

PLATE 5 : Photomicrographs of the lower epidermis of cv. Peniarth infected (I) and uninfected (C) with Erysiphe graminis.



C

3.3.2.2. Effects on root development

Infection clearly altered root growth, as shown earlier by the measurements on the accumulation of dry weight in the different organs. However, the morphological changes which probably occurred in total length and in individual root thickness as a result of infection were not examined .

3.3.2.3 Effects on reproductive structures and grain yield

The results of counts or measurements of yield components and grain yield of infected and uninfected plants of each line are given in Appendix tables 7 to 9 and they are summarized in Tables 9 and 10 .

The LSD shows that, for each oat line, the total grain yield per plant was significantly reduced ($P < 0.05$) by mildew infection. The reduction as a percentage of the uninfected controls of cv. Peniarth, cv. Lustre and wild oat being, 57 %, 55 %, and 48 % respectively. Comparisons between infected and uninfected plants in relation to yield components, showed that the reduction in grain yield was principally a consequence of fewer tillers, fewer fertile tillers, fewer grains per main axis panicle and therefore fewer grains per plant (Table 9). Infection reduced the number of tillers of infected plants but the differences between infected and uninfected plants were not significant in all three lines. However, the number of fertile tillers per plant was reduced by infection in cv. Lustre but significantly reduced ($P < 0.05$) in cv. Peniarth and wild oat (Table 10A). Infection also significantly reduced ($P < 0.05$) the number of grains per main axis panicle in cvs Lustre, Peniarth and wild oat. The number of grains per plant was therefore significantly reduced ($P < 0.05$) in all three lines (Table 10B). The infection also significantly reduced ($P < 0.05$) hundred grain weight and total grain yield in cvs Lustre and Peniarth, but also, although to a lesser extent, in wild oat ($P < 0.1$) (Table 10C). However, although the total grain yield and total plant dry weight were significantly reduced in all three lines, though to a lesser extent in wild oat, the values obtained for the harvest index of infected plants were not significantly different from those of uninfected plants for any line (Table 10D). It thus appear that the

TABLE 9

Primary values on the effects of Erysiphe graminis on yield components and final grain yield of infected and uninfected plants of the three oat lines. Each reading is a mean of ten replicates ± SE.

Grain yield & Yield components	Lustre		wild oat		Peniarth	
	Infected	Control	Infected	Control	Infected	Control
Number of tillers	3.2 ± 0.2	3.6 ± 0.3	3.5 ± 0.3	4.0 ± 0.4	3.7 ± 0.3	4.2 ± 0.4
Number of fertile tillers	2.5 ± 0.2	3.1 ± 0.2	3.0 ± 0.2	3.7 ± 0.3	2.8 ± 0.2	3.6 ± 0.3
Number of grains per panicle [1]	39.2 ± 3.1	51.5 ± 3.8	51.7 ± 3.2	64.1 ± 3.5	38.9 ± 2.8	47.8 ± 2.6
Number of grains per plant	85.2 ± 8.1	150.2 ± 4.8	97.7 ± 6.9	171.9 ± 8.1	74.5 ± 5.3	139.7 ± 4.5
Hundred grain weight (g)	1.830 ± 0.05	2.253 ± 0.09	1.176 ± 0.04	1.325 ± 0.03	1.673 ± 0.05	2.067 ± 0.07
Total grain yield (g)	1.527 ± 0.11	3.410 ± 0.22	1.171 ± 0.12	2.272 ± 0.11	1.244 ± 0.09	2.892 ± 0.15
Total plant d .wt (g)	4.873 ± 0.43	9.358 ± 0.56	4.069 ± 0.28	7.102 ± 0.37	3.914 ± 0.29	8.180 ± 0.48
Harvest Index	24.57 ± 1.70	26.81 ± 1.28	22.27 ± 1.58	24.60 ± 1.63	24.25 ± 1.06	27.31 ± 1.59

1 main axis panicle.

TABLE 10

A : Analyses of variance

(1) *Number of tillers / plant*

Source of variation	df	SS	MS	VR	P
Plant line	2	3.100	1.550	1.30	NS
Treatment	1	3.267	3.267	2.75	NS
Plant line x Treatment	2	0.033	0.017	0.02	NS
Residual	54	64.200	1.189		
Total	59	70.600			

* $LSD_{.05} = 0.98$

(2) *Number of fertile tillers / plant*

Source of variation	df	SS	MS	VR	P
Plant line	2	4.4333	2.2167	4.34	0.05
Treatment	1	8.0667	8.0667	15.78	0.001
Plant line x Treatment	2	0.2333	0.1167	0.36	NS
Residual	54	27.6000	0.5111		
Total	59	40.3333			

* $LSD_{.05} = 0.64$

* LSD values for plant line x treatment interactions.

B : Analyses of variance

(1) *Number of grains / panicle*[1]

Source of variation	df	SS	MS	VR	P
Plant line	2	2488.0	1244.0	11.10	0.001
Treatment	1	1881.6	1881.6	16.78	0.001
Plant line x Treatment	2	39.7	19.8	0.18	NS
Residual	54	6053.6	112.1		
Total	59	10462.9			

* $LSD_{.05} = 9.52$

(2) *Number of grains / plant*

Source of variation	df	SS	MS	VR	P
Plant line	2	7875.7	3937.8	9.62	0.001
Treatment	1	70520.7	70520.7	172.30	0.001
Plant line x Treatment	2	241.0	120.5	0.29	NS
Residual	54	22102.1	409.3		
Total	59	100739.6			

* $LSD_{.05} = 18.17$

1 main axis panicle

* LSD values for plant line x treatment interactions.

C : Analyses of variance

(1) *Hundred grain weight*

Source of variation	df	SS	MS	VR	P
Plant line	2	6.92497	3.46249	91.63	0.001
Treatment	1	1.55751	1.55751	41.22	0.001
Plant line x Treatment	2	0.22683	0.11342	3.00	0.05
Residual	54	2.04061	0.03779		
Total	59	10.74993			

* $LSD_{.05} = 0.17$

(2) *Total grain yield*

Source of variation	df	SS	MS	VR	P
Plant line	2	5.7459	2.8730	12.94	0.001
Treatment	1	35.4555	35.4555	159.66	0.001
Plant line x Treatment	2	1.5447	0.7723	3.48	0.05
Residual	54	11.9916	0.2221		
Total	59	54.7377			

* $LSD_{.05} = 0.42$

* LSD values for plant line x treatment interactions.

D : Analyses of variance

(1) *Total plant dry weight*

Source of variation	df	SS	MS	VR	P
Plant line	2	24.636	12.318	6.45	0.001
Treatment	1	231.465	231.465	121.18	0.001
Plant line x Treatment	2	6.132	3.066	1.61	NS
Residual	54	103.141	1.910		
Total	59	365.374			

* $LSD_{.05} = 1.24$

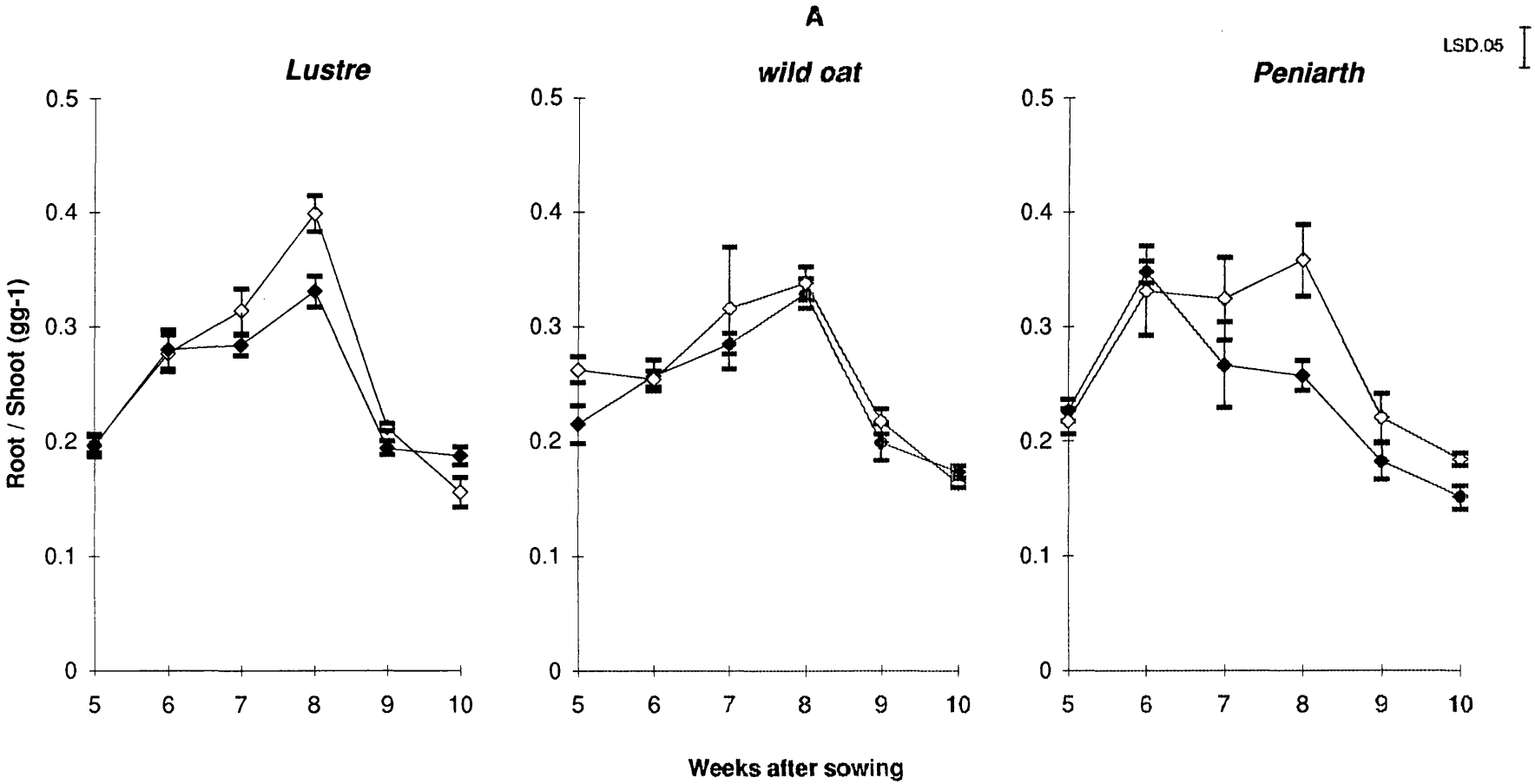
(2) *Harvest Index*

Source of variation	df	SS	MS	VR	P
Plant line	2	190.69	95.35	1.08	NS
Treatment	1	211.99	211.99	2.40	NS
Plant line x Treatment	2	2.24	1.12	0.01	NS
Residual	54	4760.49	88.16		
Total	59	5165.42			

* $LSD_{.05} = 8.43$

* LSD values for plant line x treatment interactions.

FIGURES 20A-20D : Effects of infection on (A) root/shoot ratio, (B) leaf weight ratio, (C) leaf area ratio and (D) specific leaf area of infected (◆) and uninfected (◇) plants of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.


A
LSD.05
Lustre
wild oat
Peniarth
Root / Shoot (gg-1)
0.5
0.4
0.3
0.2
0.1
0
5
6
7
8
9
10
Weeks after sowing

proportion of total biomass converted to grains was not affected in any of the plant lines.

3.3.3. Effects of infection on the distribution of dry matter

3.3.3.1. Effects on root : shoot ratio

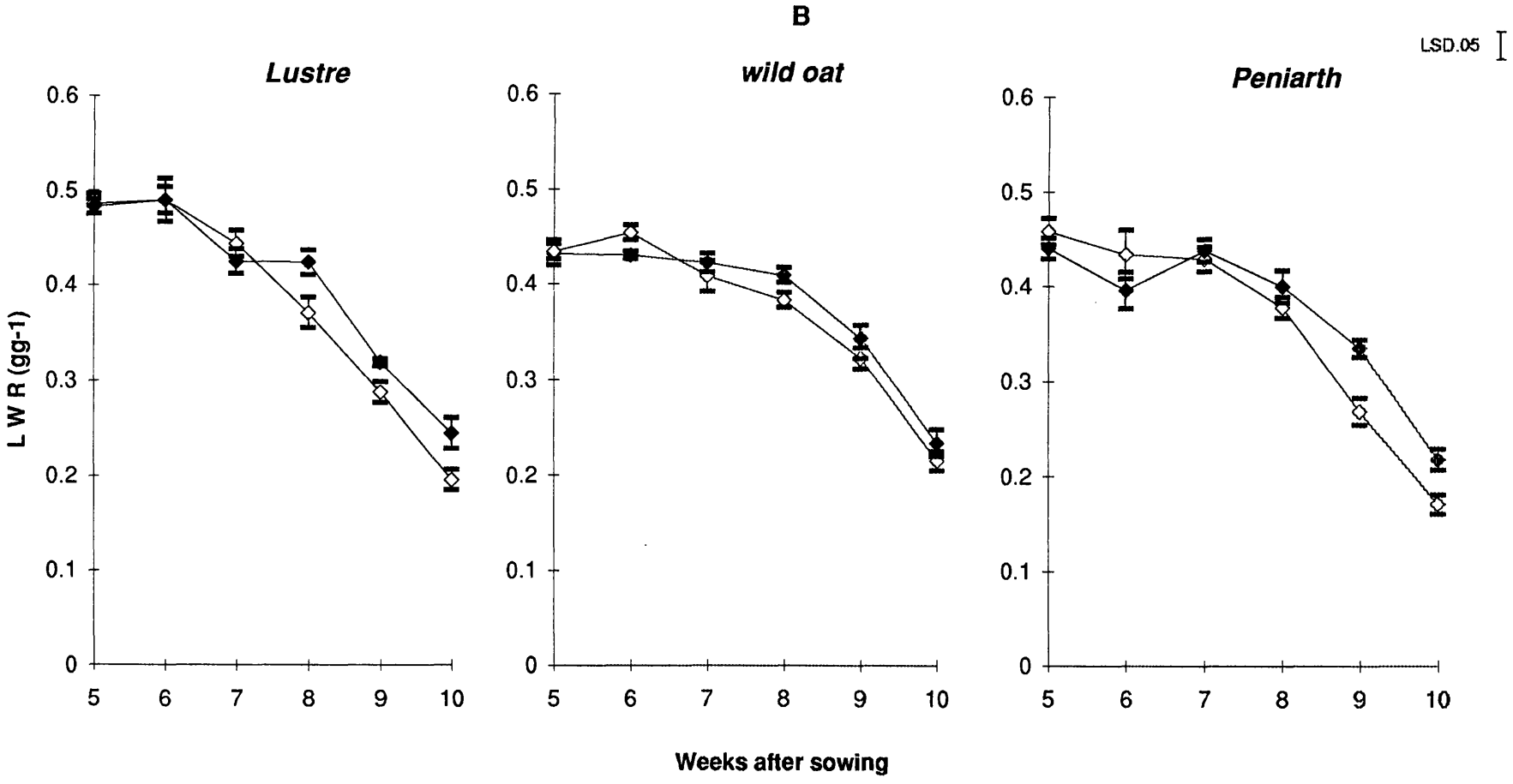
The ratios of root to shoot dry weight of infected and uninfected plants of each line at each harvest are plotted in Fig. 20A .

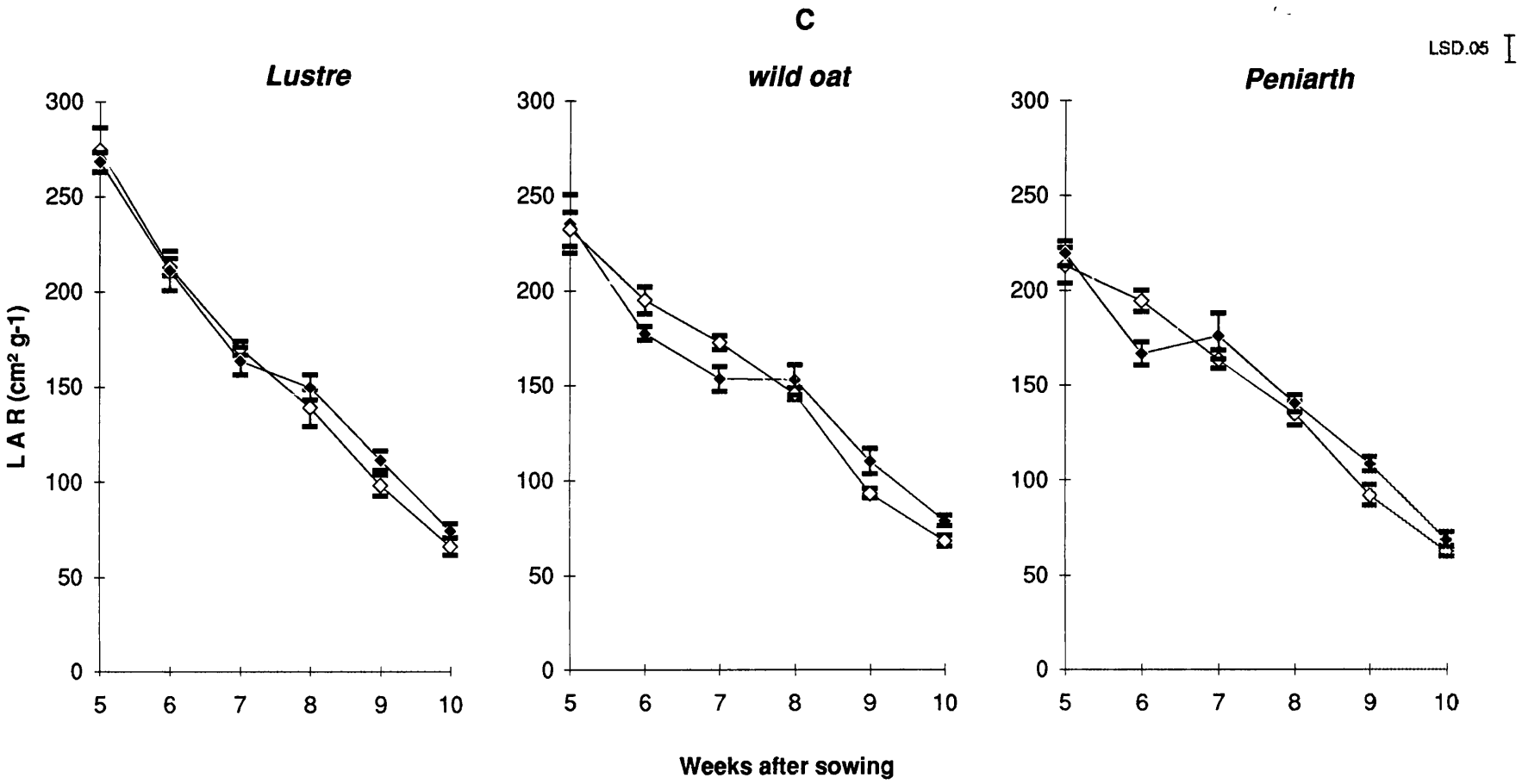
The analysis of variance shows that the proportion of total dry matter contained in the roots of infected plants was less than that of uninfected plants in cv. Lustre and wild oat but significantly less ($P < 0.05$) in cv. Peniarth (Fig. 20A). The differences in the ratios between infected and uninfected plants of the cv. Peniarth were first evident at about the same time as reductions in dry matter production, seven weeks after sowing when total mildew cover was about 20 %, whereas these differences did not become significant even when total mildew cover was about 23 % in cv. Lustre or about 28 % in wild oat (Fig. 20A).

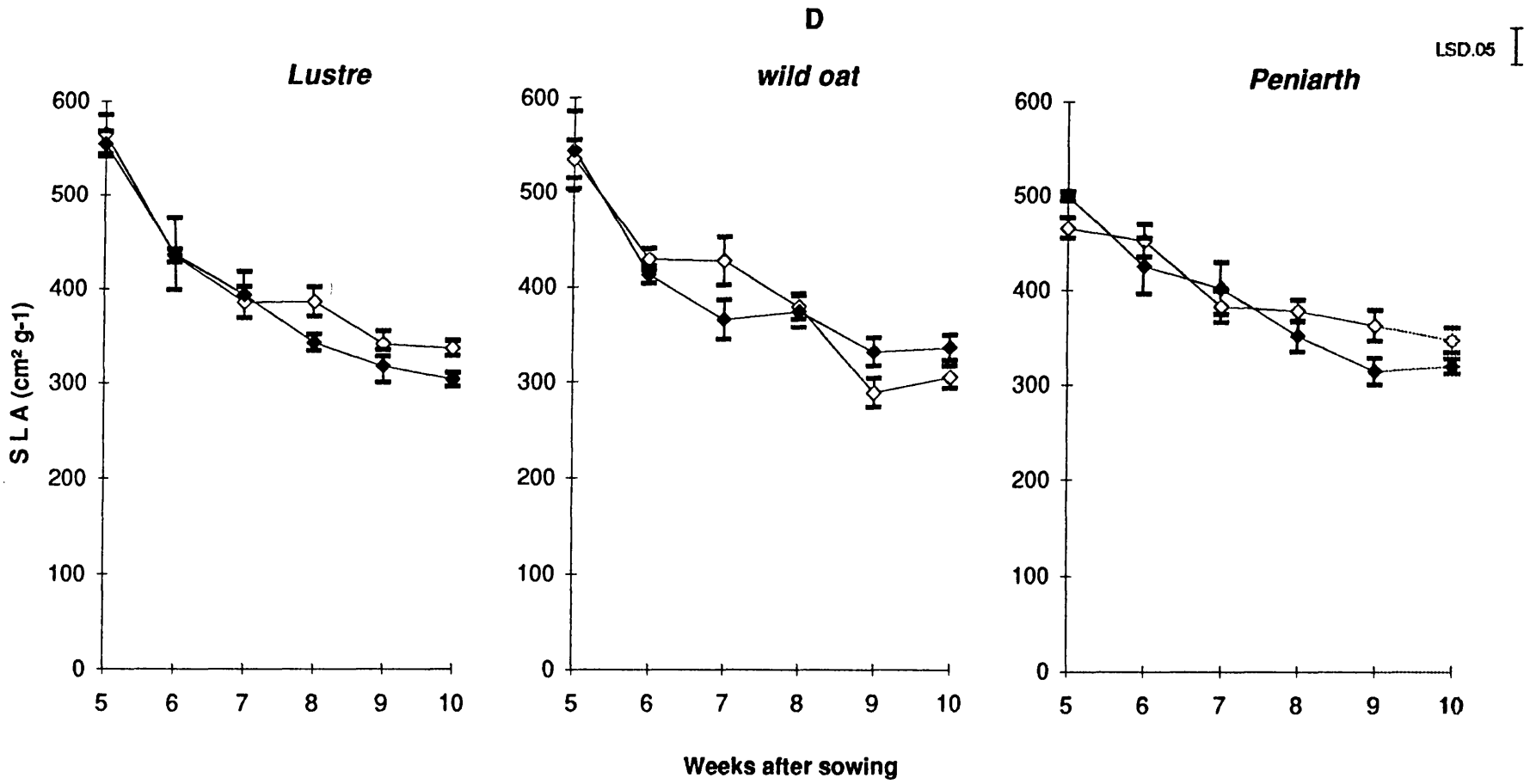
3.3.3.2. Effects on leaf weight ratio

Changes in leaf weight ratios of infected and uninfected plants of each line are plotted in Fig. 20B .

The ratio of leaf dry weight to total plant dry weight of infected and uninfected plants showed a negative correlation with time for all three lines, its value being high during the early stages of growth and low as the plants matured (Fig. 20B). The downward trend of leaf weight ratio indicates that as the plants grow older and as total plant biomass increased, the proportion of dry matter that accumulated in the leaves diminished. The analysis of variance shows that leaf weight ratios of infected plants were significantly higher ($P < 0.05$) than those of uninfected plants of cvs Lustre and Peniarth, but not of wild oat (Fig. 20B). The differences between infected and uninfected plants became apparent around eight weeks after sowing in cv. Lustre when


B
LSD.05
Lustre
wild oat
Peniarth
L W R (gg-1)
0.6
0.5
0.4
0.3
0.2
0.1
0
5
6
7
8
9
10
Weeks after sowing


C
LSD.05
Lustre
wild oat
Peniarth
L A R (cm² g-1)
300
250
200
150
100
50
0
5
6
7
8
9
10
Weeks after sowing


D
LSD.05
Lustre
wild oat
Peniarth
S L A (cm² g-1)
600
500
400
300
200
100
0
5
6
7
8
9
10
Weeks after sowing

total mildew cover was about 25 % and a week later in cv. Peniarth when total mildew cover was about 15 %. It thus appears that although infection reduced the size of the leaves, the relative proportion of total dry matter in leaf tissue was greater in the infected plants than in the uninfected. However, in wild oat, although infection reduced leaf area, the reduction was proportional to the size of the plant and infection did not significantly alter the relative proportion of dry matter in the leaves .

3.3.3.3. Effects on leaf area ratio and specific leaf area

Changes in leaf area ratio and specific leaf area of infected and uninfected plants of each line are plotted in Figs. 20C and 20D .

Leaf area ratios (Fig. 20C) and specific leaf areas (Fig. 20D) of infected and uninfected plants of all three lines showed a negative correlation with time similar to the leaf weight ratio. A decline in specific leaf area suggests that a relatively higher proportion of the assimilates were retained within the leaves. Whether they are retained within the cells as starch or other storage materials or utilised to form new cells was not determined. Since both leaf area ratio and specific leaf area were also determined from leaf area data, they both show a similar negative correlation with time. The declining leaf area ratio suggests a diminishing amount of materials allocated for the development of the assimilatory surface in relation to the total amount available in the plant. The analysis of variance shows that leaf area ratios (Fig. 20C) and specific leaf areas (Fig. 20D) of infected plants were not significantly different from those of uninfected plants in the two cultivars and in wild oat .

3.3.4. Effects of infection on the efficiency of growth

3.3.4.1. Effects on relative growth rate

Changes in the relative growth rates of infected and uninfected plants of each line are plotted in Fig. 21 .

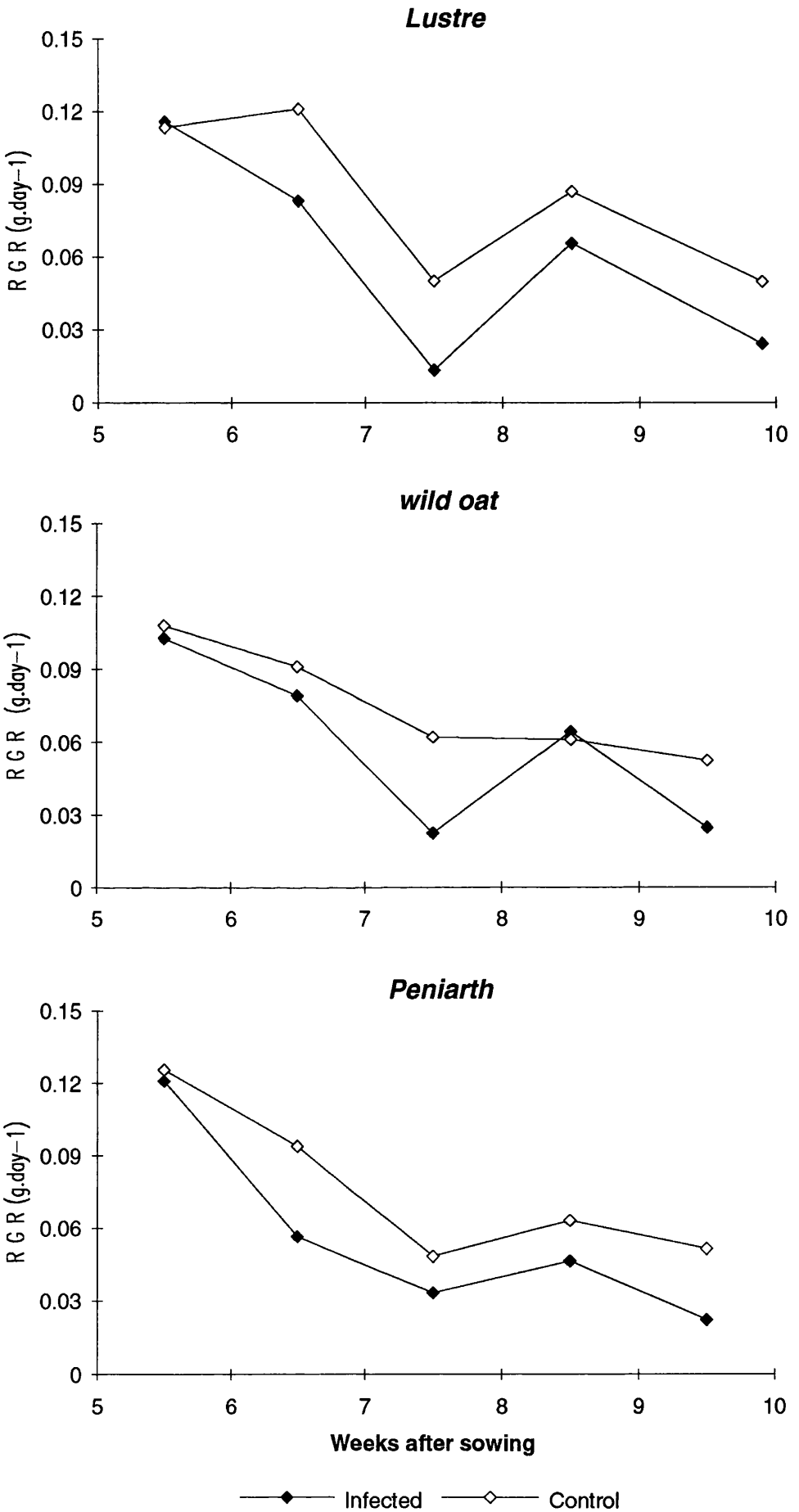


Fig. 21 : Effects of infection on relative growth rates of infected and uninfected plants of wild oat and cvs Lustre, Peniarth.

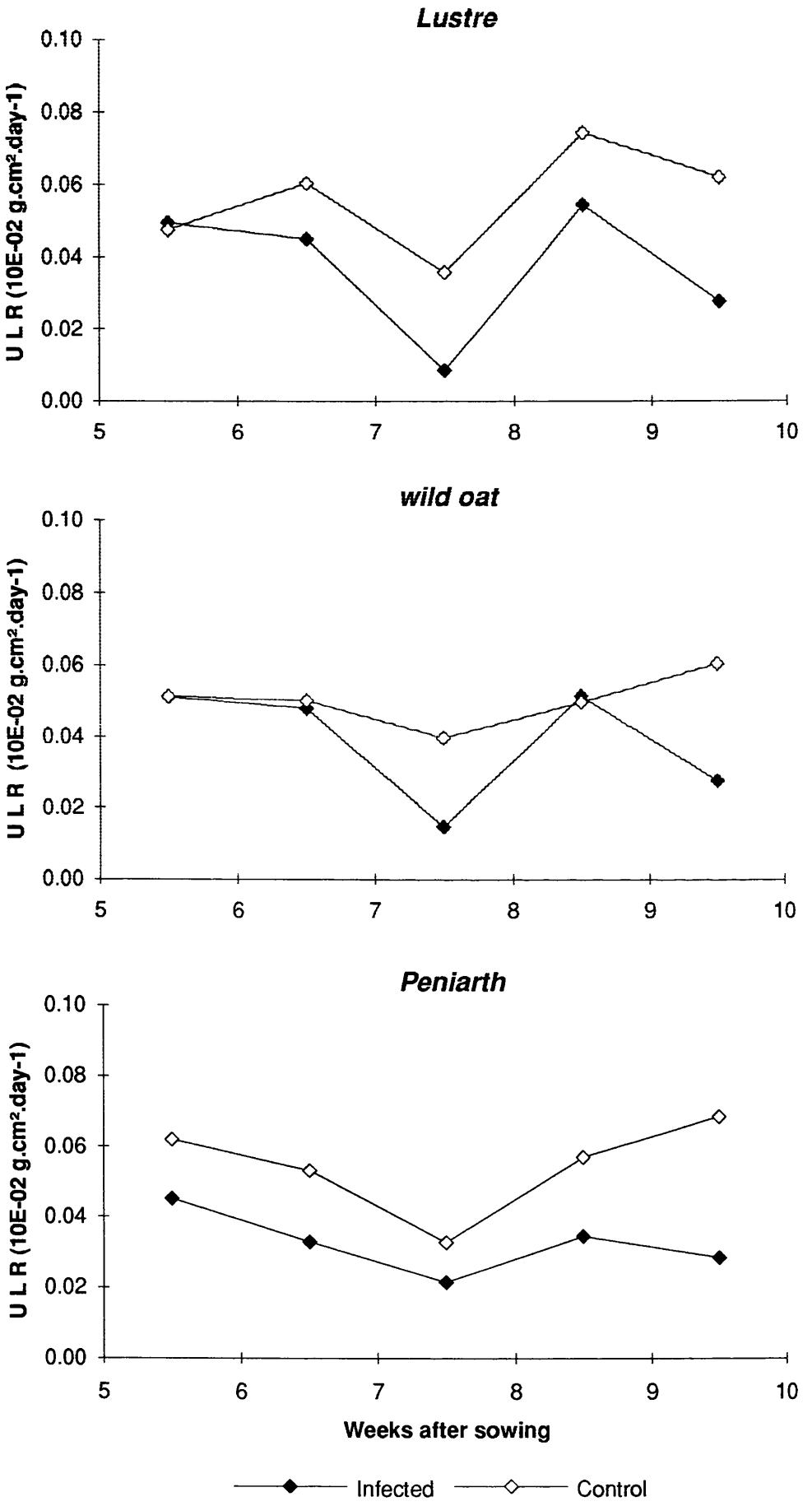


Fig. 22 : Effects of infection on unit leaf rates of infected and uninfected plants of wild oat and cvs Lustre, Peniarth.

The results show a distinct ontogenetic drift in the relative growth rates of both infected and uninfected plants of each line. The relative growth rates are high during the early stages of growth, but decrease as the plants age up to the eighth week after sowing. Between the eighth and ninth week, the relative growth rates increased coincident with the period of main axis elongation and ear emergence, followed by a further decrease up to the tenth week after sowing when the last harvest was made (Fig. 21). The differences between infected and uninfected plants of cvs Lustre and Peniarth and of wild oat became apparent between the sixth and seventh week after sowing, with the differences being greater in the two cultivars than in wild oat (Fig. 21). The total effect of mildew infection was to decrease the relative growth rate by 28 % in cv. Lustre, 26 % in cv. Peniarth but only 21 % in wild oat, 5 to 10 weeks after sowing .

3.3.4.2. Effects on unit leaf rate

The changes in unit leaf rates during the growth of infected and uninfected plants of each line are plotted in Fig. 22 .

The curves for both infected and uninfected plants of each oat line show a negative correlation with time, except during the interval between the eighth and ninth week after sowing where the unit leaf rates of both infected and uninfected plants of each line increased, followed by a decline in the interval between the ninth and tenth week after sowing (Fig. 22). Up to the eighth week after sowing, the amount of mildew increased (Fig. 9) without decreasing leaf blade area on main axis (Fig. 17), but between the eighth and ninth week, a high number of the lower infected leaves senesced leaving only the lightly infected, young and effective leaves (Table 3). Thus, in the interval from eight to nine weeks after sowing, when differences in leaf blade area and total plant dry weight were more conspicuous, leaves on infected plants became almost as efficient as those on healthy plants. The differences in unit leaf rates between infected and control plants were less apparent in wild oat than in cvs. Lustre and Peniarth (Fig. 22) .

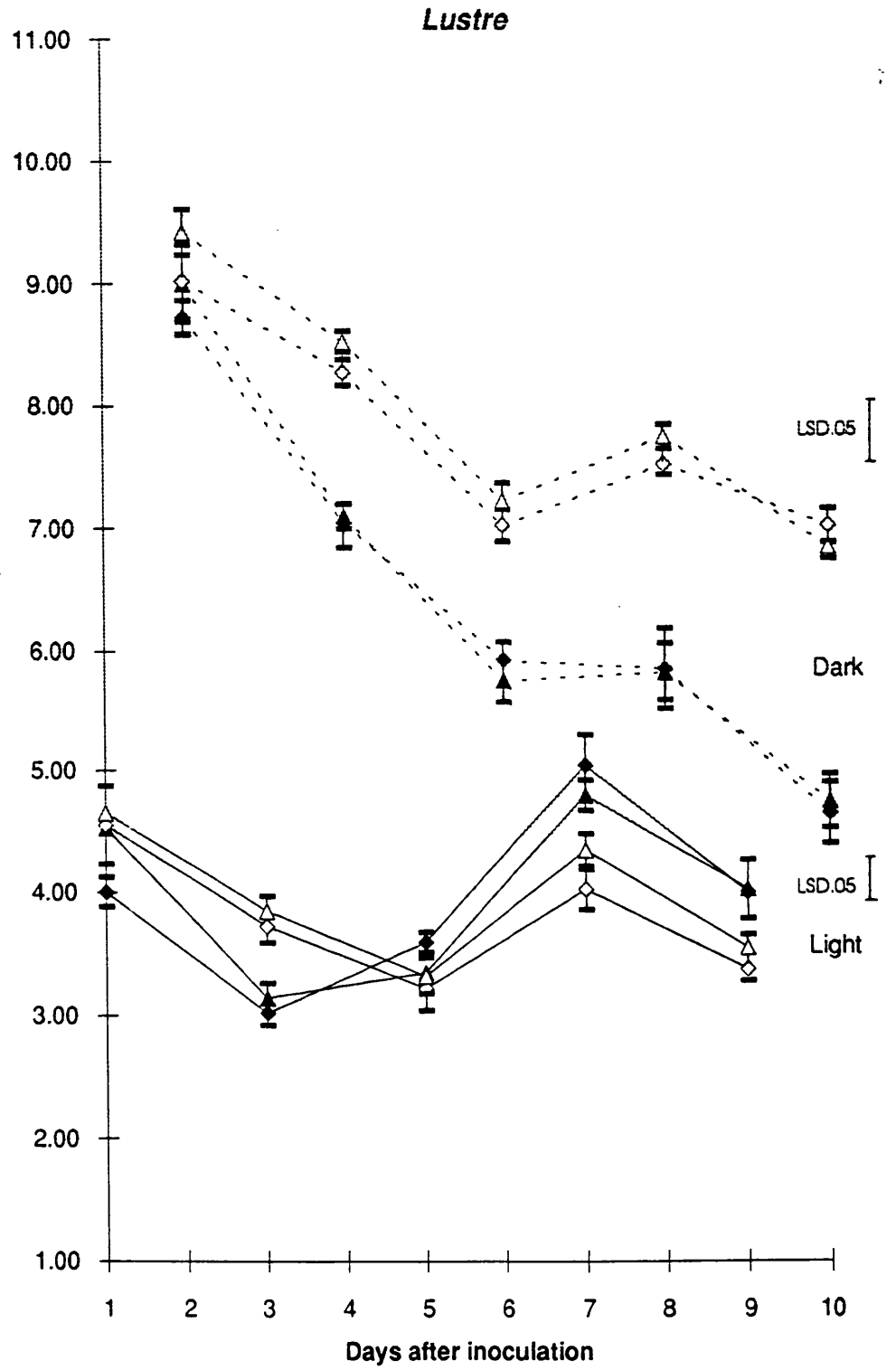
CHAPTER 4

THE EFFECTS OF MILDEW INFECTION ON STOMATAL FUNCTION IN THE LEAVES OF WILD OAT AND TWO LINES OF CULTIVATED OAT.

4.1. Introduction

Stomatal function directly regulates processes of primary importance in the growth and development of plants, such as photosynthesis and transpiration (Meidner and Mansfield, 1968). Through their influence on these major activities, stomata will exert effects on all other processes that are directly or indirectly dependent on them. One element in the diffusion pathway of CO_2 into the leaf to the reaction centres within the chloroplasts is stomatal aperture and so the changes in dry matter production reported in the last chapter could thus result in part from changes in stomatal behaviour.

The object of this investigation was to compare stomatal behaviour in infected and uninfected leaves during both the light and the dark periods in cvs Lustre, Peniarth and in wild oat, and to examine whether the reductions in dry matter production in infected tissues could result from changes in stomatal behaviour.



23 : Changes in diffusive resistances, during the light and dark period, of infected (middle, ◆, and tip, ▲,) and uninfected (middle, ◇, and tip, △,) leaves of cv. Lustre. Measurements taken between 1 and 10 days after inoculation.

4.2. The effects of stomatal diffusive resistance in control leaves

Sixty seedlings of each line were raised in the growth room. The plants were grown singly in 15 cm plastic pots containing S.A.I. potting compost. When 3 weeks old, the fully expanded third leaves on thirty plants of each line were inoculated by applying single conidial isolates (from leaf segments in Petri dishes) to the middle region of the upper surface of the leaf blade using a paint brush. The tip and base of the leaf were not inoculated. The other thirty plants were kept free from mildew by spraying with 0.05 % Benlate solution at weekly intervals. The treated and untreated plants of each line were then placed in the growth room. Six plants per treatment per line were sampled at each harvest. The first measurements in the light were made 24 h after inoculation, whereas those in the dark were made 48 h. after inoculation. Subsequent measurements were made at 2 day intervals until 5 harvests had been made. Porometer measurements were taken in the middle and tip of the upper surface of infected and uninfected third leaves on each plant line .

This experiment was carried out three times with an interval of two weeks between experiments. Similar results were obtained on each occasion and the detailed results of the third experiment are reported .

4.2.1. Ontogenetic changes in stomatal diffusive resistance

Diffusive resistances in both, middle and tip, regions of uninfected control leaves were relatively constant during the light period in all three lines (Figs. 23-25), although it is generally reported that the efficiency of stomatal functioning declines as the leaf ages (Solarova, 1970 cited in Majernik, 1971). However after 24 h. in the dark, stomatal diffusive resistances were initially high in both regions of control leaves in all three lines, particularly in cv. Lustre, but then they declined as the leaf aged in both cultivars but not in wild oat (Figs. 23-25). The reduction in diffusive resistance which occurs as the leaf aged, particularly in cv. Lustre, could be due to the increasing failure of the stomata to close in the dark or to changes in the cuticle leading to increased

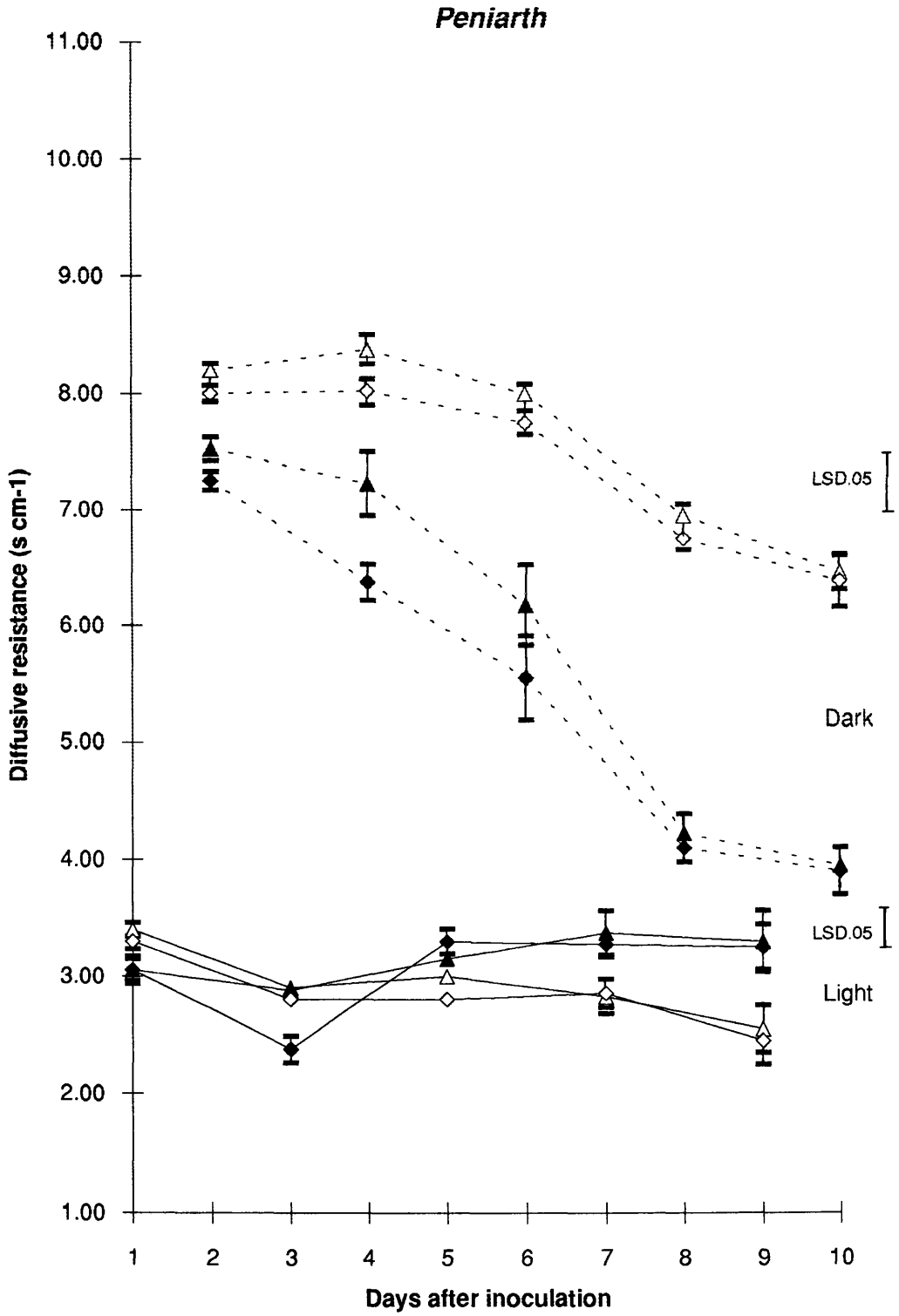


Fig. 24 : Changes in diffusive resistances, during the light and dark period, of infected (middle, ◆, and tip, ▲,) and uninfected (middle, ◇, and tip, △,) leaves of cv. Peniarth. Measurements taken between 1 and 10 days after inoculation.

cuticular transpiration. Whether such changes were due to normal ontogenetic effects or to other external factors is not known .

4.2.2. Effects of infection on diffusive resistance during the light phase of the photoperiod

Measurements were made in the middle of the light phase at 12 pm. The diffusive resistances in both the middle and tip regions of infected and uninfected leaves measured at this time are given in Appendix table 14 and are plotted in Figs. 23-25 .

The diffusive resistances in the light in both, middle and tip, regions of the infected and uninfected leaf showed that during the first 3 days after inoculation, when the percentage of mildew cover on infected leaves was negligible (1 to 2 %), the diffusive resistance in the infected middle region of the leaf fell relative to that in the corresponding region of the control leaf in wild oat (Fig. 25). The fall was even greater ($P < 0.05$) in cvs Lustre and Peniarth (Figs. 23 and 24). The diffusive resistance in the uninfected tip region of the infected leaf was also significantly lower ($P < 0.05$) than that in the corresponding region of the control leaf in cv. Lustre but no significant differences were found between infected and control leaves in either cv. Peniarth or wild oat. However, from 5 to 9 days after inoculation, a period which correlated with maximum fungal growth, assessed by fungal sporulation and chitin analysis (Figs. 27A-B, see chapter 5), the diffusive resistances became greater in both the middle and tip regions of infected leaves than in the uninfected controls in cvs Lustre (Fig. 23), Peniarth (Fig. 24) and in wild oat (Fig. 25). The results of the analysis of variance shows that the differences between diffusive resistances in the middle region of the infected leaf and the corresponding region of the control leaf became significant ($P < 0.05$) from 5 days after inoculation in each line, whereas for the uninfected tip region of the infected leaf and the corresponding region of the control leaf, the differences did not become significant until two days later in all three lines. However, no significant differences were found between the middle and tip regions of either infected or uninfected leaf in each line .

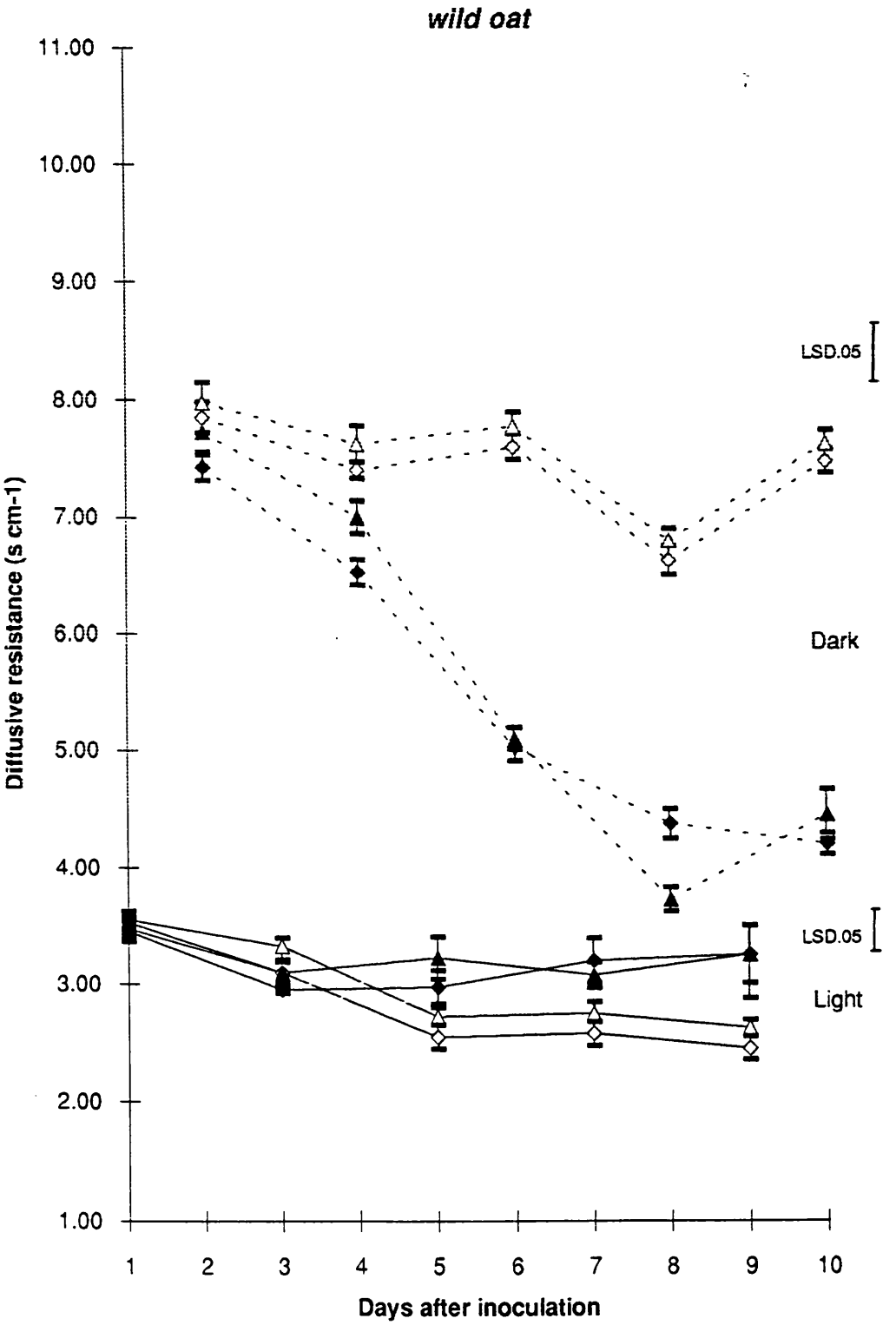


Fig. 25 : Changes in diffusive resistances, during the light and dark period, of infected (middle, ◆, and tip, ▲,) and uninfected (middle, ◇, and tip, △,) leaves of wild oat. Measurements taken between 1 and 10 days after inoculation.

4.2.3. Effects of infection on diffusive resistance during the dark phase of the photoperiod

After measuring diffusive resistances in the light phase, the plants were moved to the dark room and measurements were made 24 h. later. The results of measurements of diffusive resistances in both the middle and tip regions of infected and uninfected leaves during the dark period are given in Appendix table 15 and are plotted in Figs. 23-25 .

After 24 h in the dark, the diffusive resistances in both the middle and tip regions of uninfected leaves were higher than those of infected leaves at each harvest in all three lines (Figs. 23-25). The lower values obtained for diffusive resistances of infected leaves indicate that either infection prevented stomata from closing in the dark as fully as those in uninfected control leaves, or that the presence of mycelium on the leaf surface lead to greater water loss from the infected leaf. In both, middle and tip, regions of uninfected leaves, the diffusive resistances remained high after 24 h. in darkness, with small fluctuations particularly in cvs Lustre and Peniarth, during the course of the experiment indicating that the stomata were closed. However, in both regions of infected leaves, the diffusive resistance in cv. Peniarth began to fall within 48 h. after inoculation (Fig. 24), although percentage mildew cover at this stage was negligible (< 1%), with significant differences ($P < 0.05$) between infected and uninfected leaves thereafter. These differences were not apparent in either cv. Lustre (Fig. 23) or wild oat (Fig. 25) until 4 days after inoculation when percentage mildew cover was between 1 to 2 %. From then on, as the mycelium developed, the diffusive resistances in both the middle and tip regions of infected leaves became significantly lower than those in the corresponding regions in healthy leaves in all three lines, although, the reductions were not as large in wild oat as in the two cultivars. The analysis of variance shows no significant differences between the middle and tip regions of either the uninfected or infected leaf in any line, except during the early stages of infection when the differences between the two regions of the infected leaf were significant ($P < 0.05$) in cv. Peniarth and wild oat but not in cv. Lustre .

4.3. Conclusion

The diffusive resistances in the light and dark period respectively were changed by infection in a similar way in cvs Lustre, Peniarth and wild oat. During the light period, diffusive resistances of control leaves were low indicating that the stomata were open and their aperture appeared to remain constant during the course of experiment. In infected leaves, the decrease in diffusive resistances in the light that occurred during the first 3 days after inoculation, before fungal mycelium had developed to any significant extent, almost certainly reflects an increase in stomatal aperture since water loss from the mycelium would have been insignificant at this stage. This increase is in line with that caused by Erysiphe pisi in pea (Ayres, 1976). The factors responsible for this are not known. The increased diffusive resistances in the light that occurred from about 5 days after inoculation, when mycelium development became significant and so would be expected to contribute to water loss, clearly indicates that the degree of stomatal opening in the light decreased with increasing levels of infection in all three lines .

During the dark period, the diffusive resistances in control leaves showed a distinct ontogenetic drift indicating that as leaves aged either their stomata failed to close completely or cuticular transpiration increased, particularly in cvs Lustre and Peniarth. In infected leaves, 48h. after inoculation when mycelium development was limited, the diffusive resistances were low in cv. Lustre and wild oat but much lower in cv. Peniarth indicating that the stomata failed to close in the dark. An increase in stomatal opening in the dark could not be clearly recorded in cv. Lustre and wild oat because by the time significant differences in diffusive resistances were recorded, there was too much mycelium development to separate water loss from stomata from that from mycelium .

CHAPTER 5

THE EFFECTS OF MILDEW INFECTION ON PHOTOSYNTHESIS IN THE LEAVES OF WILD AND TWO LINES OF CULTIVATED OAT.

5.1. Introduction

In chapter 3, it was shown that infection decreased dry matter production significantly in all three lines. Some of this decrease was due to the reductions in green leaf blade area as a result of enhanced senescence of the older leaves, reduced expansion of individual leaves and reduced rate of leaf production particularly in cv. Peniarth. However, infections of plant tissues by microbial parasites are also reported to decrease the rates of photosynthesis (Allen, 1942; Ayres, 1976 and Walters, 1985), and so some of the loss of dry matter production could result from effects on the efficiency of the photosynthetic systems .

In the last chapter, it was shown that infection was likely to reduce CO_2 diffusion into the leaf through the stomata so that photosynthesis in the infected leaf could be reduced due to a reduced level of CO_2 reaching the reaction centres in the chloroplast .

This chapter describes a series of experiments which determined the effects of mildew development on a leaf on the photosynthetic and respiratory rates and chlorophyll content of that leaf in the same three lines, as were used for the growth analysis experiments, or in cv. Lustre and wild oat only. Photosynthetic and respiratory rates were measured using a Hansatech leaf electrode. Similar trends in photosynthetic parameters were determined when the data were analysed by the two models which describe the photosynthetic-light response curve (PLR), the model of Rabinowitch and that of Marshall and Biscoe (see Materials and Methods, section 2.8.2.3.) .

In the first experiment, the effects of infection were measured on the third leaf in the three lines where the whole of that leaf had been inoculated. For this

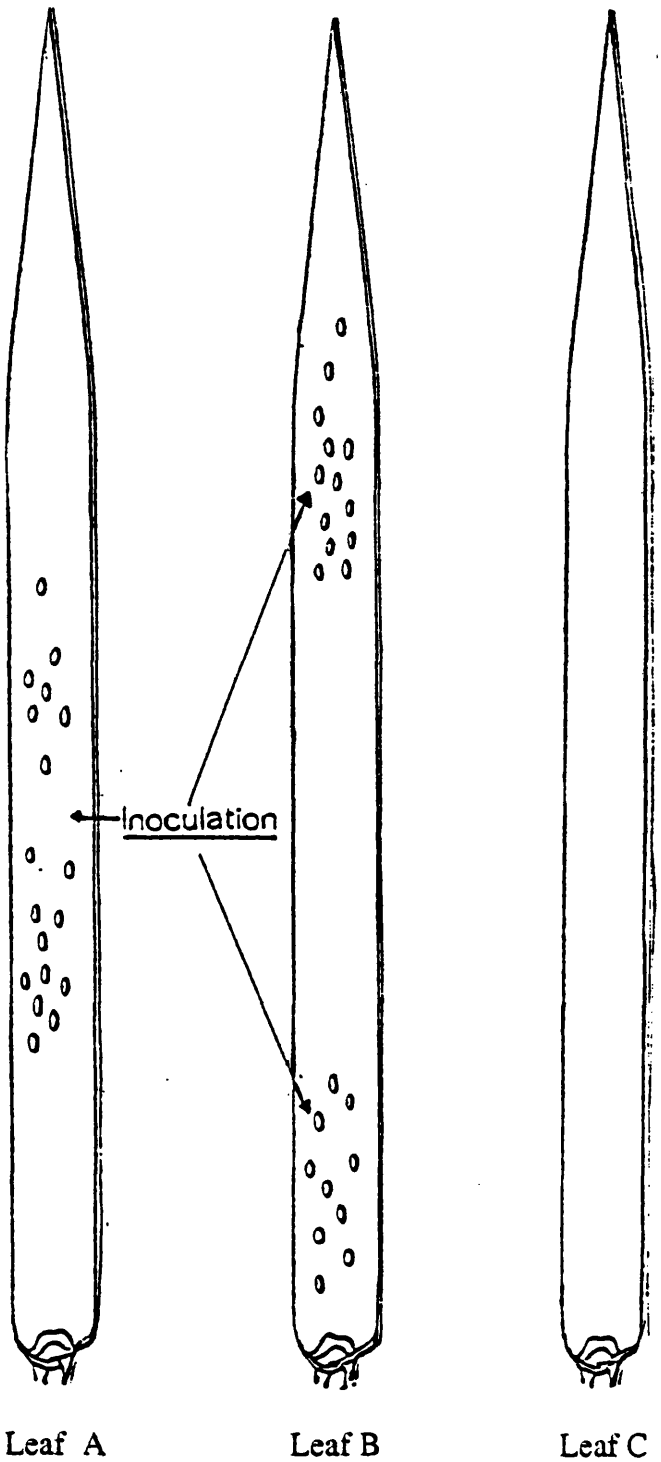


Fig 26 : Diagrammatic representation of the fourth leaf inoculated with fungal spores in the middle region (A), at the tip and base (B) and non-inoculated control (C).

experiment, mildew development on the leaf at each sampling time was assessed. In the second experiment, the middle part or the tip and base of the fourth leaf was inoculated (Fig. 26) and the effects of infection in the inoculated middle part (Fig 26A), in the uninoculated part (Fig. 26B) and in the uninoculated control leaf (Fig. 26C) were studied. This experiment was designed to determine if photosynthesis in uninfected regions of an infected leaf can compensate for any reductions in the infected regions. Chlorophyll fluorescence emission was also assessed in the second experiment.

The data were subjected to an analysis of variance using the GENSTAT statistical programme and significance was assessed using the LSD test.

5.2. Mildew development on the third leaf

Fungal development on the third leaf of the two cultivars and on wild oat was assessed in two ways; by measuring spore production and by measuring the accumulation of chitin.

5.2.1. Fungal development assessed by counting spore production

Twenty eight seedlings of each line were raised singly in 12 cm plastic pots. When three weeks old, the third leaves on all plants of each line were inoculated with a single conidial isolate using inoculum from plants grown in the Isolation Plant Propagator. Inoculation was carried out using a settling tower as described in Materials and Methods. The plants were then replaced in the growth room until required for spore production measurements. Four plants per line were sampled for each measurement. The first measurement was made 4 days after inoculation with subsequent measurements at 2 day intervals until six counts had been made and then a final count was made 4 days later. Counts of spore production per unit leaf area at each sampling time are presented graphically in Fig. 27A.

The mycelium developed rapidly on the inoculated leaf of each line to produce a uniform covering over the leaf within 4 to 6 days (Plate 6). Few spores were produced

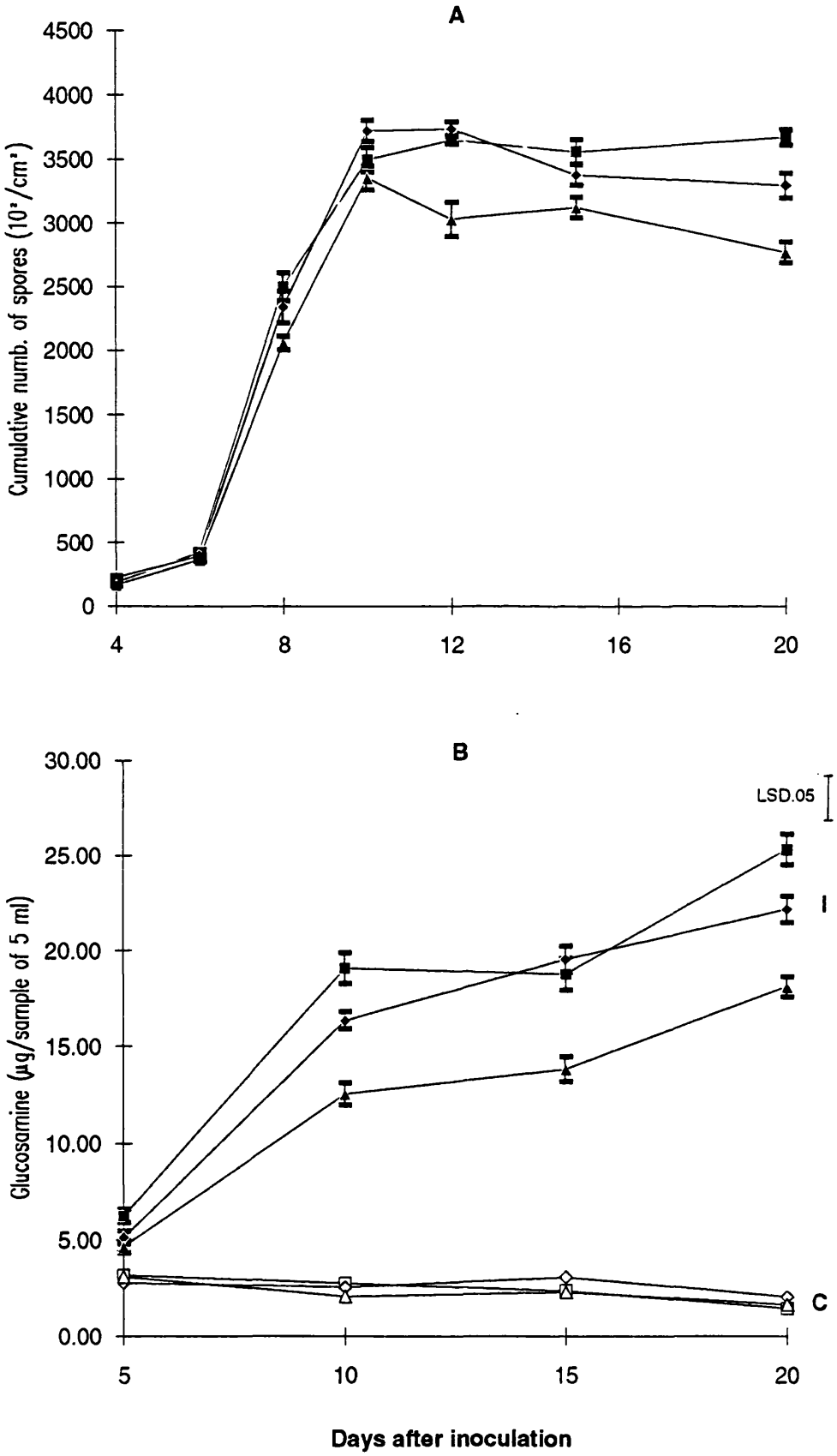


Fig. 27 : Growth of E. graminis on the third leaf of wild oat (■) and cvs Lustre (◆), Peniarth (▲) as determined by spore production per unit area (A) and amounts of glucosamine (B). I = Infected , C = Control

over this period, but spore production became abundant from the sixth day onwards after inoculation. On all three lines the maximum number of spores was reached by about 10 days after inoculation after which there appeared to be no further production (fig. 27A). At each sampling time, the cumulated number of spores present per unit leaf area of wild oat and cv. Lustre was higher than that of cv. Peniarth. However, there was little differences between wild oat and cv. Lustre .

5.2.2. Fungal development assessed by chitin analysis

Forty eight seedlings of each line were raised in 12 cm plastic pots, two seedlings in each pot. Twenty four plants of each line, e.g. 12 pots, were inoculated as above while the plants in the other 12 pots were kept free from mildew by spraying with 0.05 % Benlate solution at weekly intervals. Three pots, e.g. six plants per line per treatment, were sampled at each time; three samples of leaves were taken, each sample consisting of two entire blades of the third leaf. The first sample was taken 5 days after inoculation with subsequent samples 10, 15 and 20 days after inoculation. The results of the chitin analyses are plotted in Fig. 27B .

The amounts of glucosamine detected in the uninfected tissues of plants of each line were consistently well below those of infected tissues (Fig. 27B) indicating that host tissues did not contain sufficient hexosamines to interfere with the assay. Early fungal growth on infected leaves was detectable from around 5 days after inoculation (Fig. 27B). Then glucosamine levels increased dramatically over a further 5 days after inoculation, a period which correlated with sporulation (Fig 27A). By 10 days after inoculation, high levels of glucosamine had accumulated in the leaf tissue of all three lines. At this stage, the amount of glucosamine measured in infected tissues of wild oat was significantly greater ($P < 0.05$) than that in cvs Lustre and Peniarth. These differences were maintained up to 20 days after inoculation when the last measurements were made .

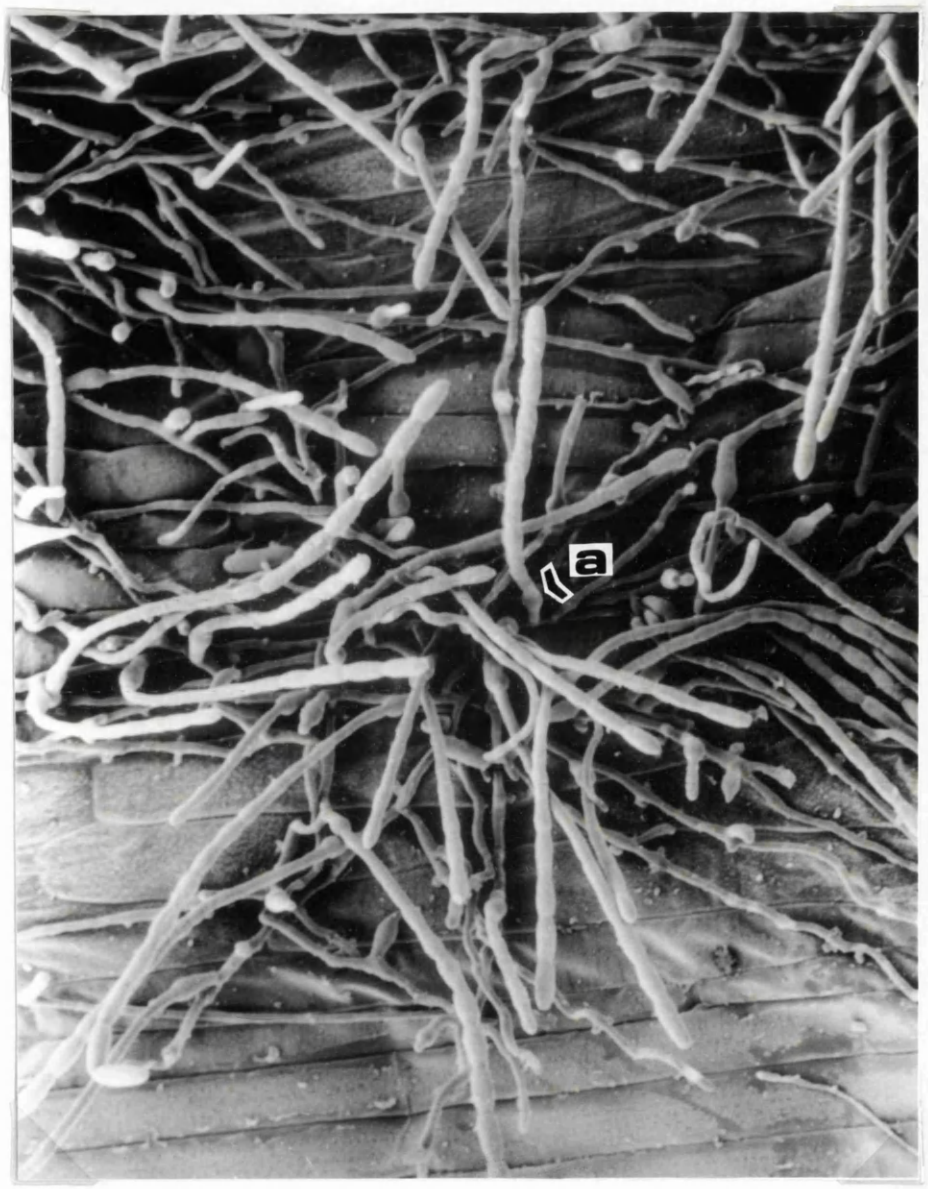


PLATE 6 : Scanning Electron Micrograph of oat leaf infected with mildew. Colonies growing from more than one conidium 5 days after inoculation. (a) chain of conidia developing from a swollen flask-shaped cell from the mycelium.

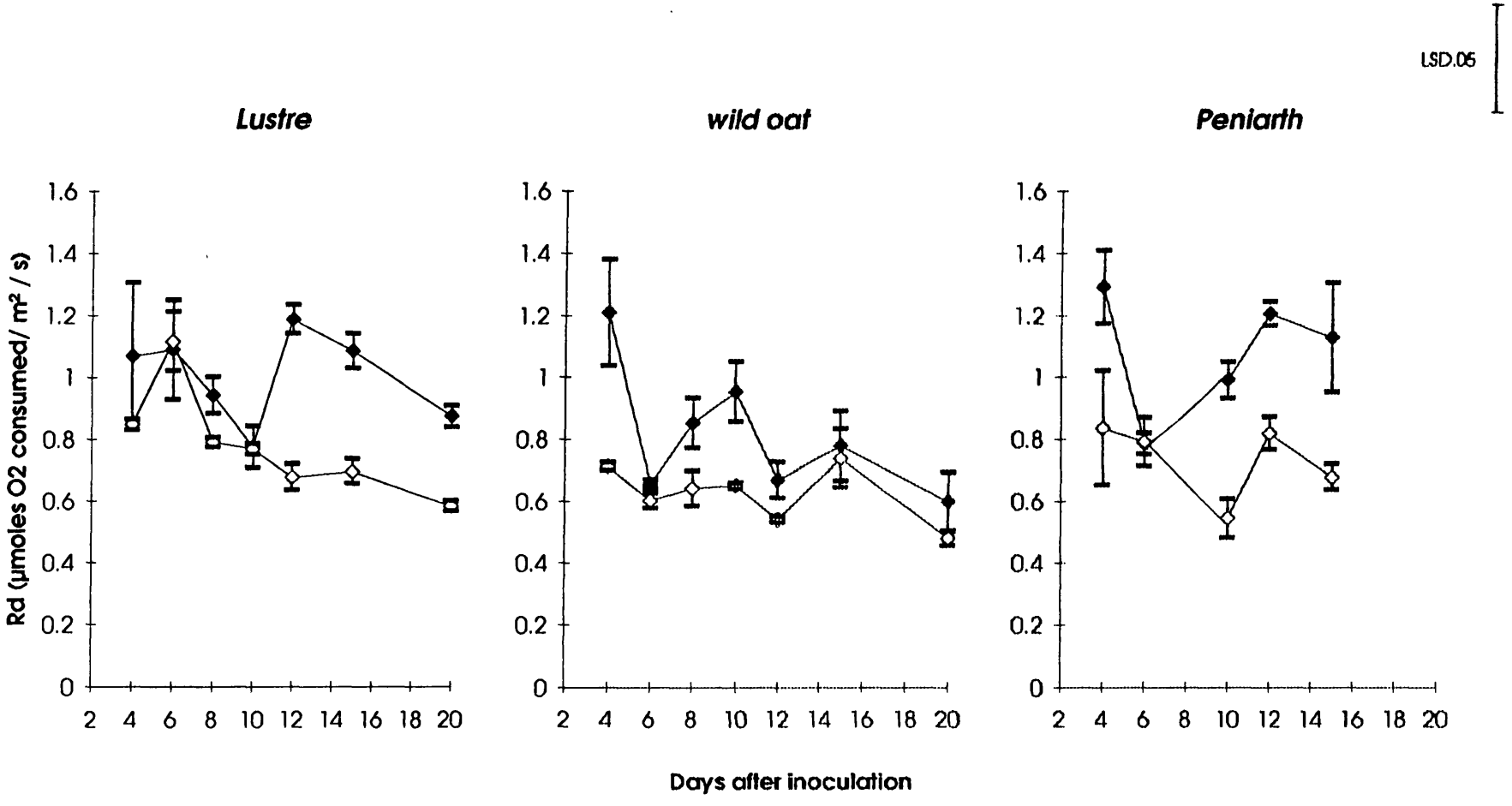


Fig. 28 : Changes in respiratory rates in infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Penlarth. Vertical bars represent standard errors.

5.3. The effects of infection on respiration, photosynthesis and chlorophyll content of the third leaf

Experiment 1

5.3.1. Methods

Fifty six plants of each of the three lines were grown singly in 15 cm plastic pots. When the third leaf blades had fully expanded, twenty eight plants of each line were inoculated and the other twenty eight plants were kept free from mildew as described earlier. The treated and untreated plants of each line were grown in the growth room under the same conditions as the plants used for fungal growth measurements (see section 5.2.1.). Four plants per line per treatment were sampled at each harvest. The experiment was carried out twice and since both gave essentially the same results, the detailed results of one experiment are reported .

In this experiment, the model for photosynthesis vs. PFD proposed by Marshall and Biscoe (see chapter 2, section 2.8.2.3.) was used to calculate: dark respiration (Rd), maximum rate of net photosynthesis (Pnmax), maximum rate of gross photosynthesis (Pgmax), photochemical efficiency of photosynthesis at low light intensity (α) and the ratio of physical to total resistance to CO_2 diffusion (θ) .

5.3.2. Effects of infection on dark respiration

The dark respiratory rates (Rd) of infected and uninfected leaf tissue of each line, at each sampling time, are plotted graphically in Fig. 28 .

The rates of respiration of both infected and uninfected leaves of each line showed a fluctuating decline during the course of the experiment (Fig. 28). The respiration of infected leaves in all three plant lines, however, was generally higher than that of uninfected leaves at all times after inoculation. No measurements of respiration were possible in leaves of cv. Peniarth from 15 days after inoculation because the infected leaf had by this stage senesced .

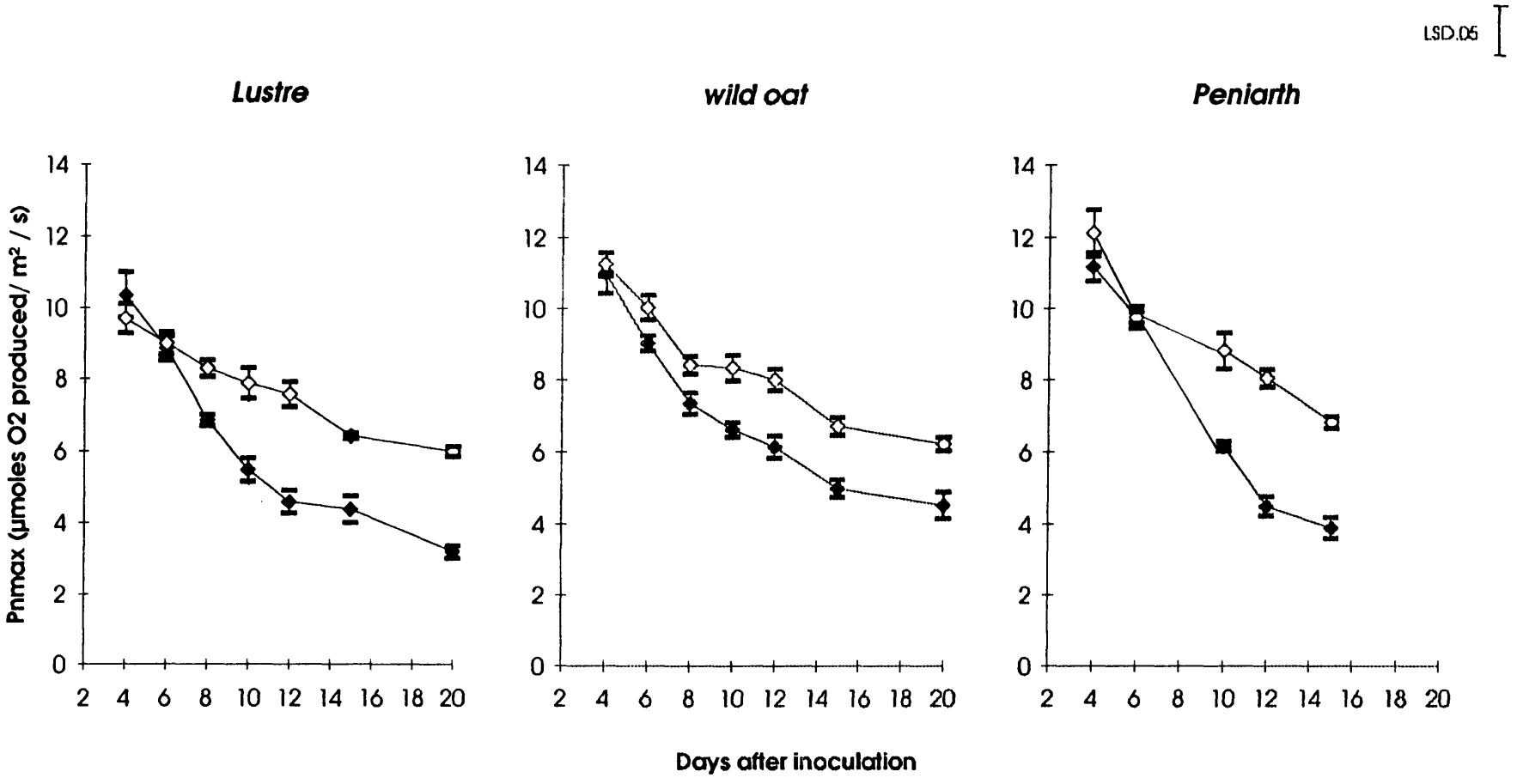


Fig. 29 : Maximum net photosynthesis per unit area of infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

The LSD value shows significant differences ($P < 0.05$) between infected and uninfected leaves in cvs Lustre and Peniarth, but not in wild oat, during the course of infection. (Fig. 28). However, there were no significant differences between the three lines in the respiratory activities of the uninfected leaves over the 15 days of the experiment.

5.3.3. Effects of infection on photosynthetic activity

5.3.3.1. Maximum rate of net photosynthesis

Maximum rates of net photosynthesis (Pnmax) of infected and uninfected leaf tissue, of each line, at each sampling time are plotted in Figs. 29 and 30.

The maximum rates of net photosynthesis per unit area of uninfected leaf tissue of each line decreased progressively during the course of the experiment. However, that of infected leaf tissue showed a much greater reduction, especially in cvs Lustre and Peniarth, which probably includes the reduction due to infection in addition to the reduction which occurs normally during leaf maturation (Fig. 29). Significant differences ($P < 0.05$) were found between infected and control leaves of all three lines, with the differences being more marked in cvs Lustre and Peniarth than in wild oat as infection progressed. The total effect of infection was to decrease the maximum rates of net photosynthesis of infected leaves by about 43 % in cv. Peniarth, with about 3131 spores produced per mm^2 leaf area, about 32 % in cv. Lustre with about 3388 spores produced per mm^2 leaf area but only by 26 % in wild oat with about 3568 spores produced per mm^2 leaf area, between 4 and 15 days after inoculation. No further measurements were possible for cv. Peniarth after 15 days but those for cv. Lustre and wild oat were continued up to 20 days after inoculation. At this stage, Pnmax in the infected leaves was reduced by about 47 % in cv. Lustre, although spore production had by then ceased, but only about 27 % in wild oat although the number of spores produced had increased to about 3684 per mm^2 leaf area.

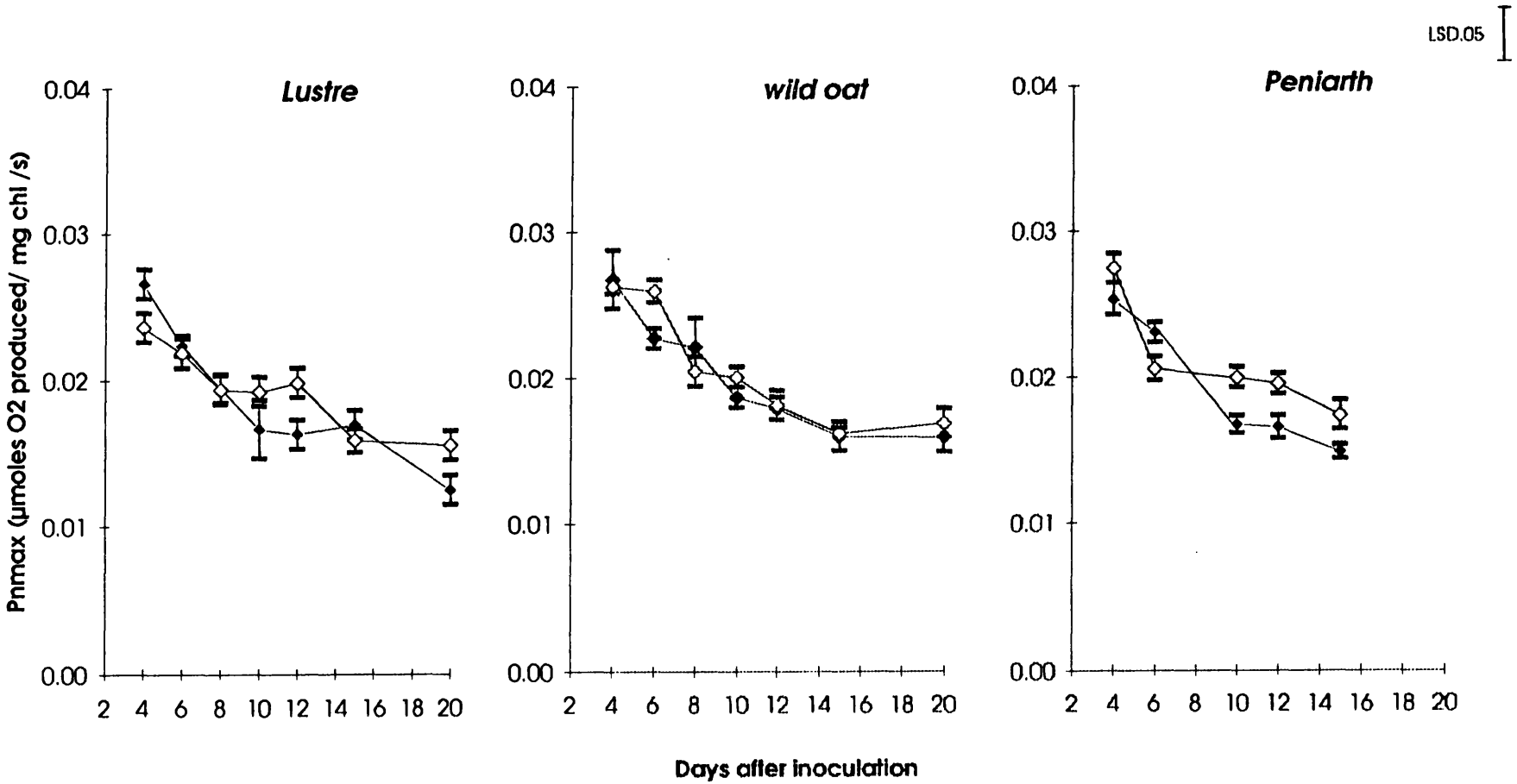


Fig. 30 : Maximum net photosynthesis per mg chlorophyll of infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

However when maximum rates of net photosynthesis were calculated per milligram of chlorophyll, they were also found to decrease as infection progressed, in all three lines, but more slowly than when expressed per unit leaf area basis. However none of the differences between infected and uninfected leaves were significant in any of the three lines at any stage of infection (Fig. 30) .

5.3.3.2. Maximum rate of gross photosynthesis

Maximum rates of gross photosynthesis (Pgmax) of infected and uninfected leaf tissue, of each line, at each sampling time are plotted in Figs. 31 and 32.

The maximum rates of gross photosynthesis per unit area of uninfected leaf tissue of each line decreased as the leaf aged through the course of the experiment. However, that of infected leaf tissue showed a more rapid decline, especially in cvs Lustre and Peniarth, as infection progressed (Fig. 31). The LSD value shows significant differences ($P < 0.05$) between infected and uninfected leaves in all three lines. The total effect of infection was to decrease the maximum rates of gross photosynthesis of infected leaves by about 36 % in cv. Peniarth, about 27 % in cv. Lustre and about 26 % in wild oat, between 4 and 15 days after inoculation. Measurements for cv. Lustre and wild oat were continued up to 20 days after inoculation. At this stage, Pgmax in the infected leaves was reduced by about 32 % in cv. Lustre whereas for wild oat there was no further reduction .

The maximum rates of gross photosynthesis, expressed per milligram of chlorophyll, of infected leaves were found to be not significantly different from those of uninfected leaves in the three lines at all stages of infection (Fig. 32) .

The difference between Pgmax (or Pnmax) when expressed per unit area and per unit chlorophyll suggests that the decrease in Pgmax (or Pnmax) could be due to a low density of the photosynthetic unit and/or a decrease in the amount of light-harvesting chlorophyll, or to an alteration of the efficiency of photosynthetic electron transport .

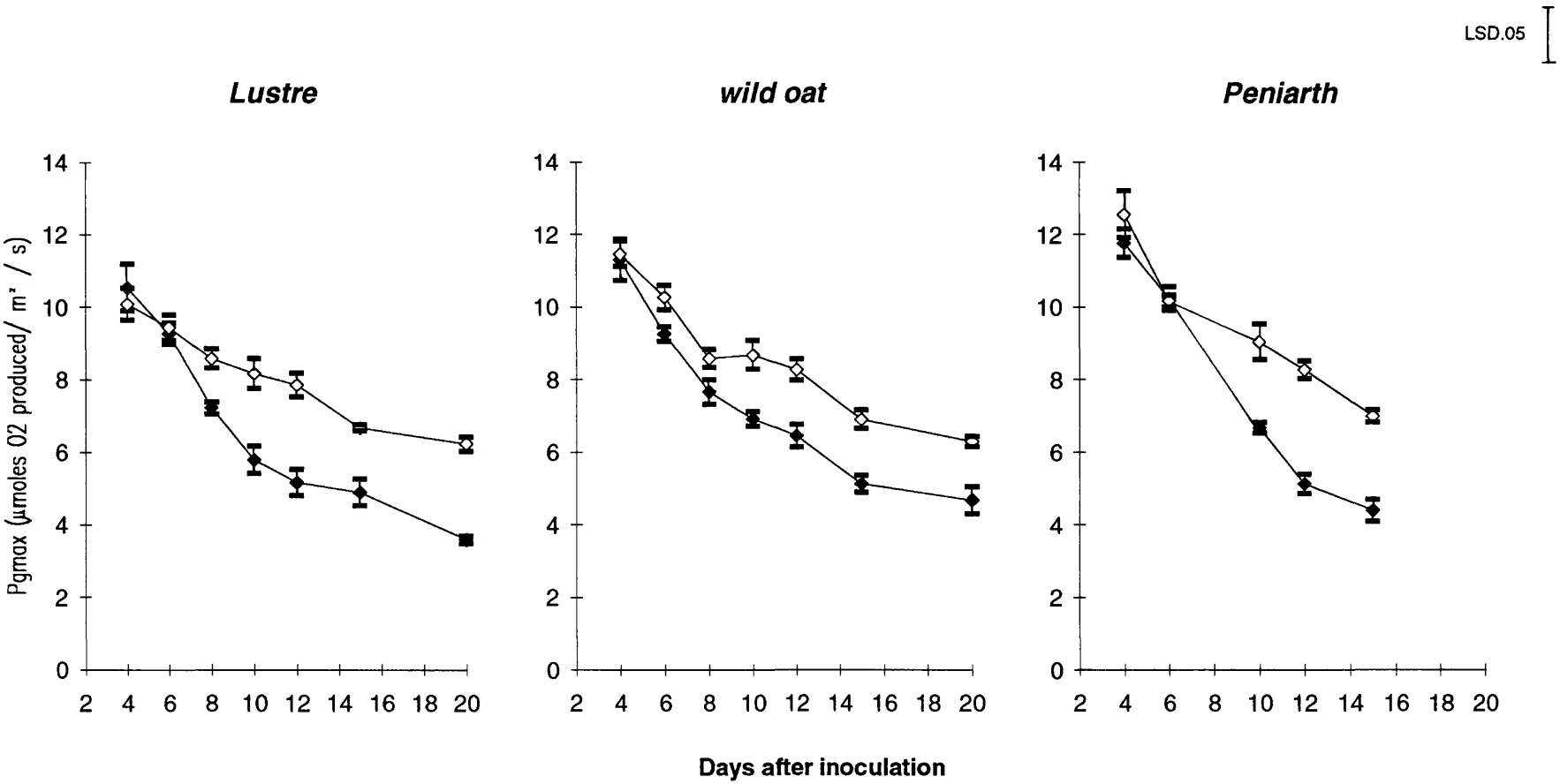


Fig. 31 : Maximum gross photosynthesis per unit area of infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

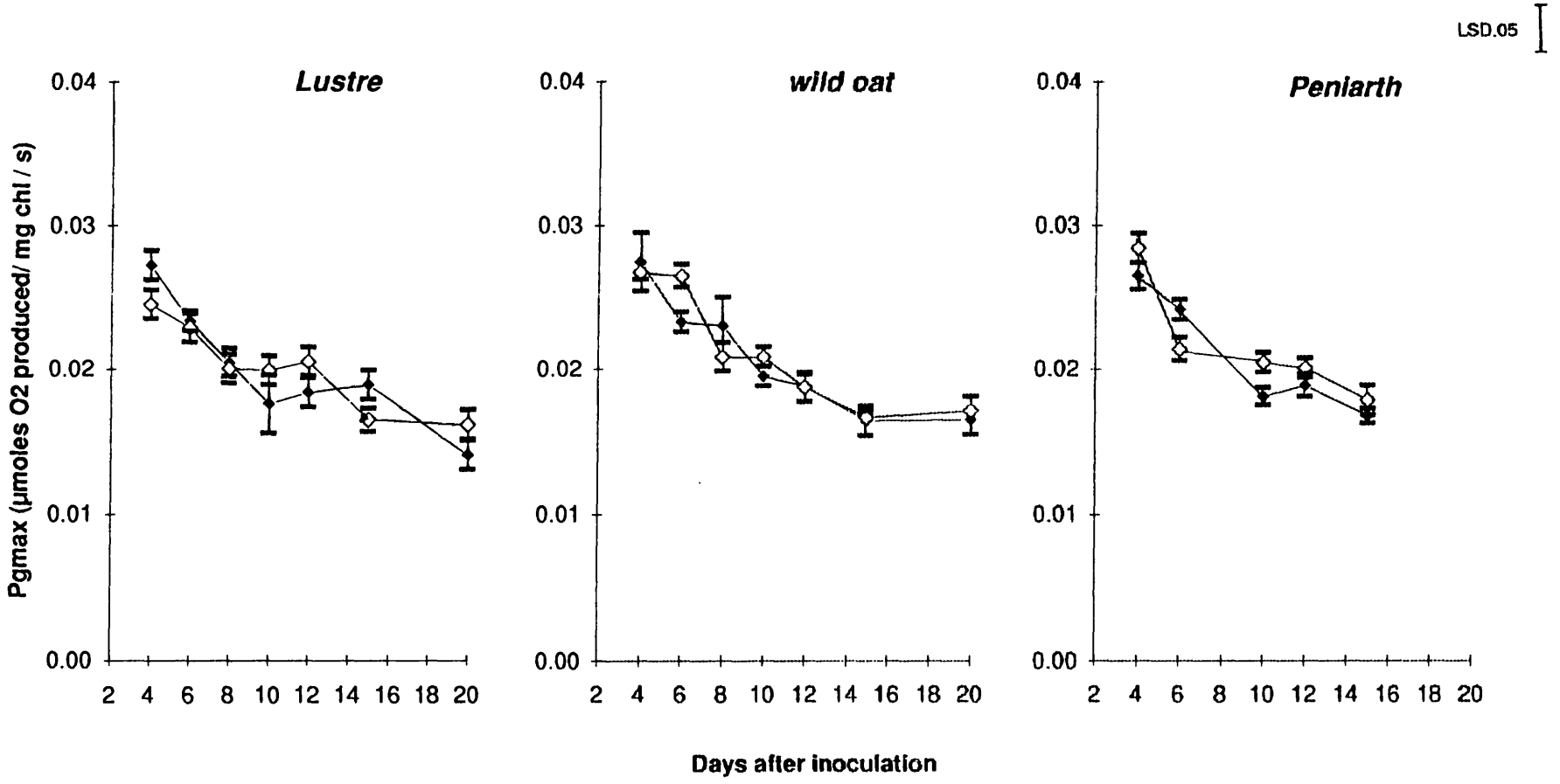


Fig. 32 : Maximum gross photosynthesis per mg chlorophyll of infected (◆) and uninfected (◇) third leaf of wild oat and Lustre, Peniarth. Vertical bars represent standard errors.

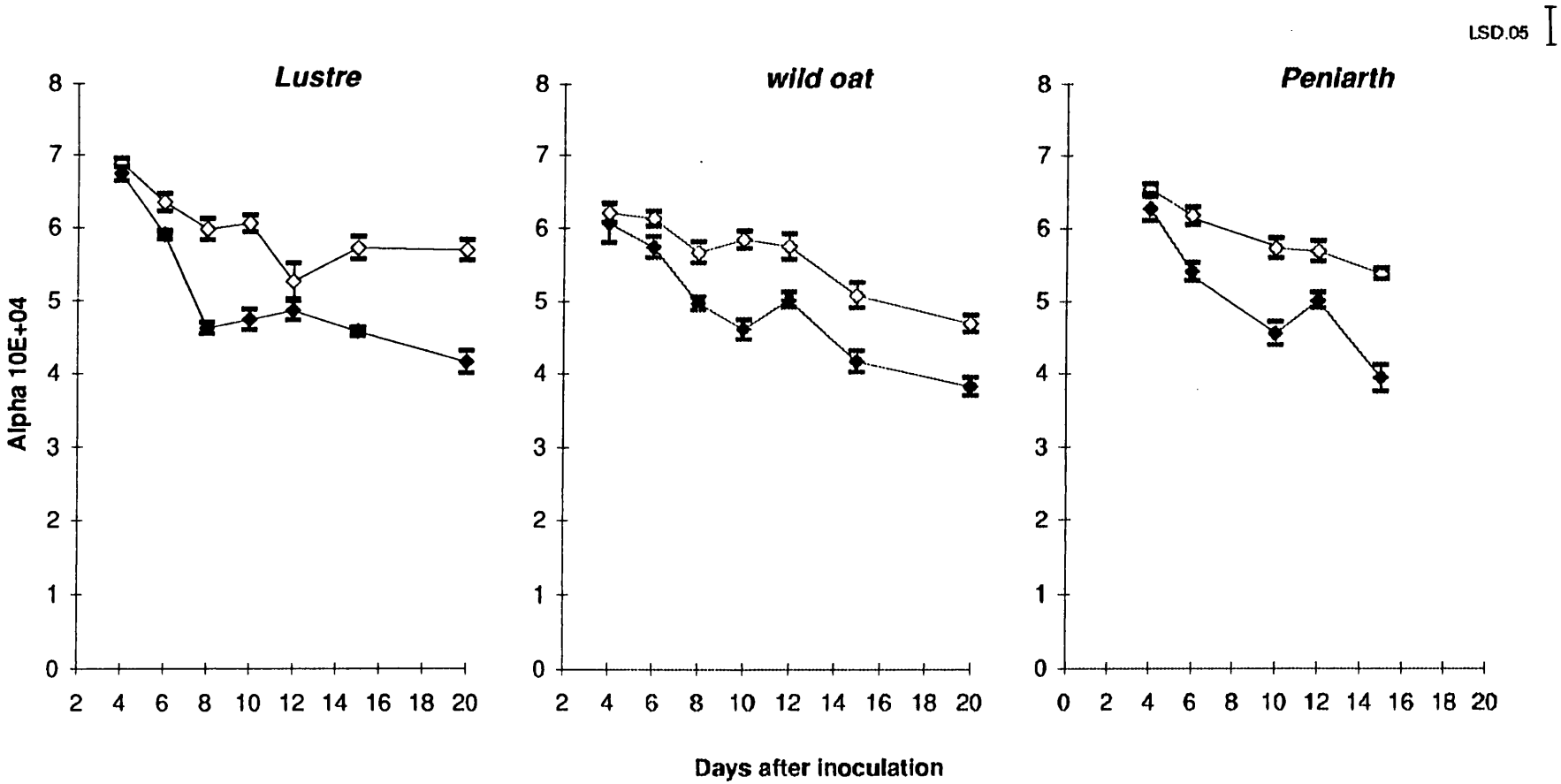


Fig. 33 : Photochemical efficiency of photosynthesis at low light intensity in infected (♦) and uninfected (◇) third leaf of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.

5.3.3.3. Photochemical efficiency of photosynthesis

Photochemical efficiency of photosynthesis at low light intensity (α) at different times after inoculation are plotted in Fig. 33 .

The curves show that as infection progressed, the photochemical efficiency of photosynthesis at low light intensity in infected leaves fell below that in uninfected leaves in all three lines (Fig. 33). The analysis of variance shows significant differences ($P < 0.05$) between infected and control leaves in each line, with the differences becoming greater as infection progressed. The overall effect of infection was to decrease the photochemical efficiency of photosynthesis by about 27 % in cv. Peniarth but about 18 % in cv. Lustre and wild oat, in the period between 4 and 15 days after inoculation. By 20 days after inoculation, the photochemical efficiency of photosynthesis in infected leaves was reduced by about 26 % in cv. Lustre but only about 19 % in wild oat .

The decrease in α also suggests a low density of the photosynthetic unit and therefore a decrease in the light-harvesting capacity (loss of chlorophyll) .

5.3.3.4. Ratio of physical to total resistance to CO_2 diffusion

The values of the ratio of physical to total resistance to CO_2 diffusion (θ) at different times after inoculation are plotted in Fig. 34 .

The results showed that the values of θ for infected leaf tissue were lower than those for uninfected tissue in cvs Lustre and Peniarth whereas in wild oat, although they fluctuated widely between harvests they did not show a consistent fall in infected tissue (Fig. 34). The analysis of variance shows no significant differences between θ values in the infected and uninfected leaf in cv. Lustre and wild oat at any stage of infection. However, in cv. Peniarth, the values of θ in the infected leaf became significantly different ($P < 0.05$) from those in the uninfected leaf from around 12 days after inoculation onwards (Fig. 34) .

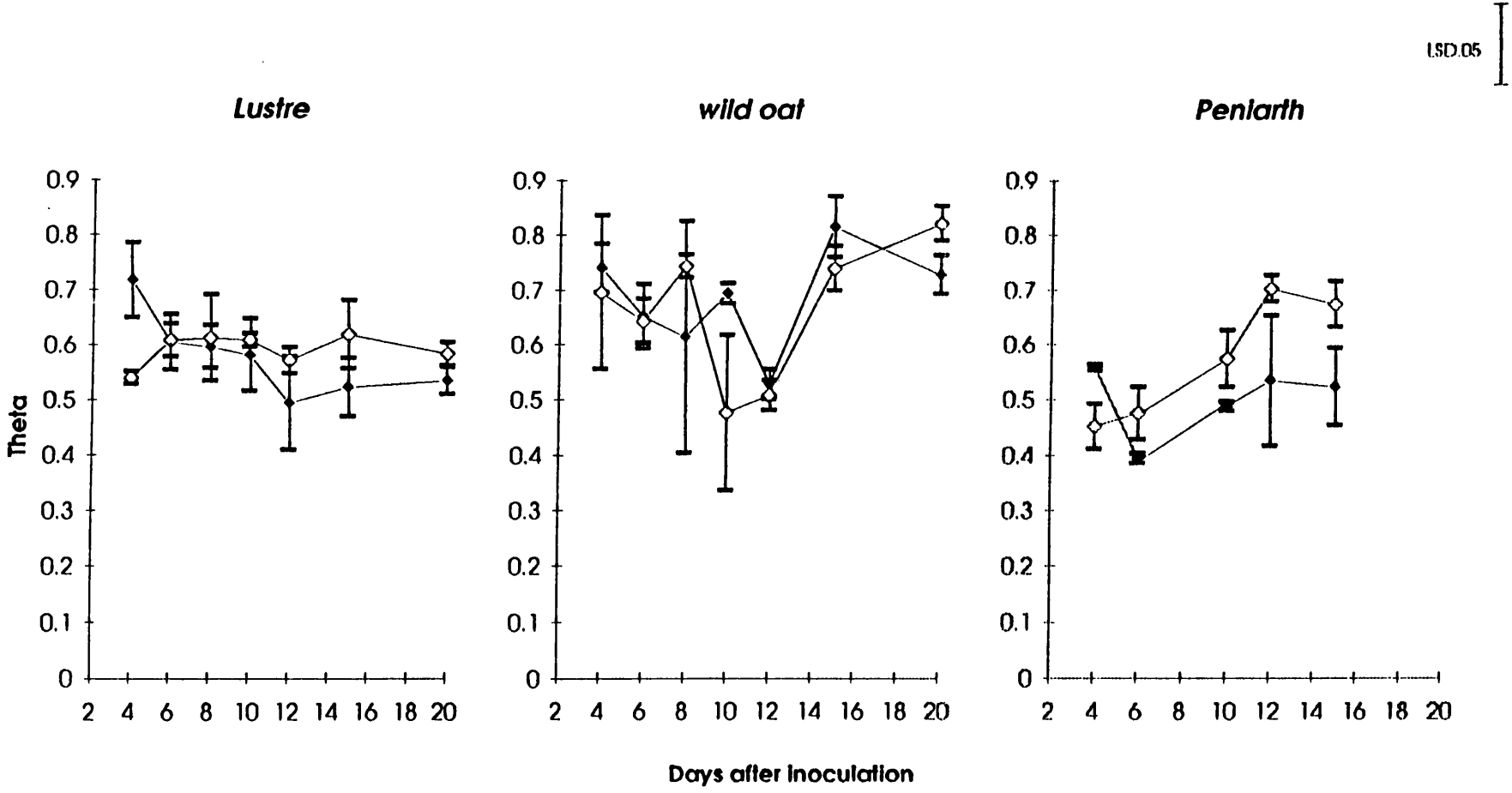
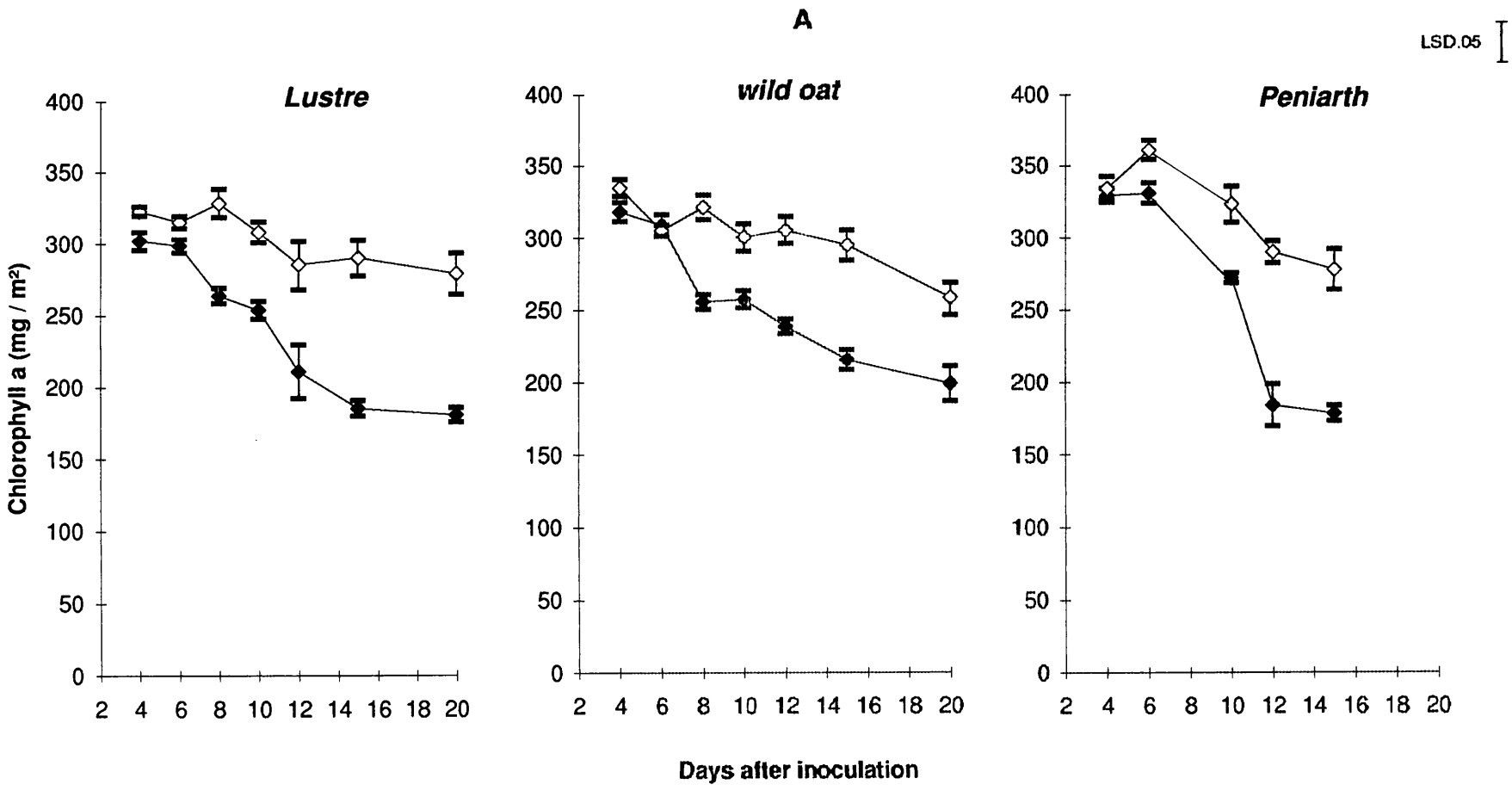


Fig. 34 : The ratio of physical to total resistance in infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Penlarth. Vertical bars represent standard errors.

Figures 35A-C : Chlorophyll a (A), chlorophyll b (B) and total chlorophyll (C) content of Erysiphe graminis infected (◆) and uninfected (◇) third leaf of wild oat and cvs Lustre, Peniarth. Vertical bars represent standard errors.


A
LSD.05
Lustre
wild oat
Peniarth
Chlorophyll a (mg / m²)
400
350
300
250
200
150
100
50
0
2 4 6 8 10 12 14 16 18 20
Days after inoculation

5.3.4. Effects of infection on chlorophyll content

5.3.4.1. Chlorophylls a and b and total chlorophyll

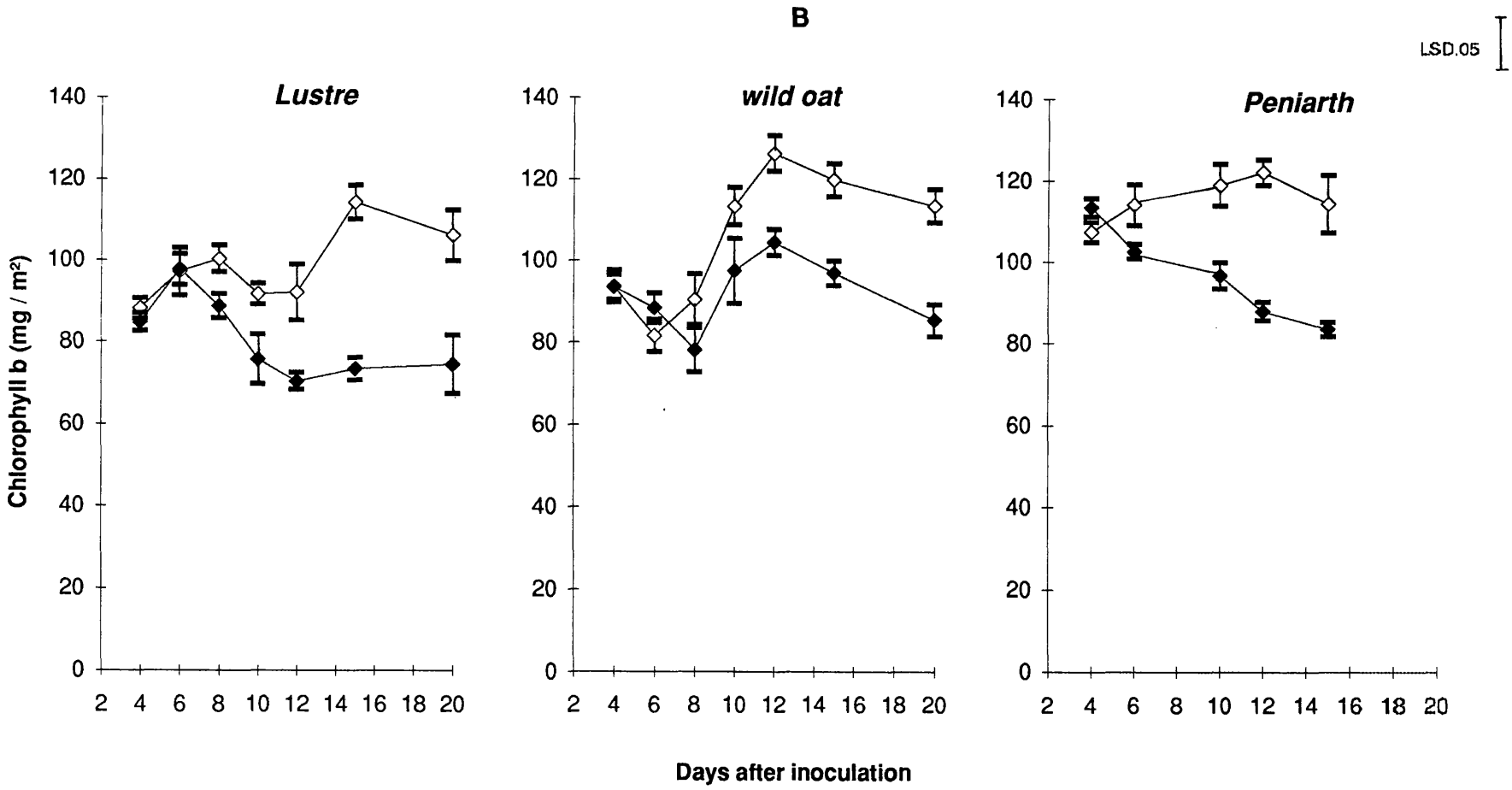
Changes in chlorophylls a and b in infected and uninfected leaf tissue of each line are plotted in Figs. 35A -B .

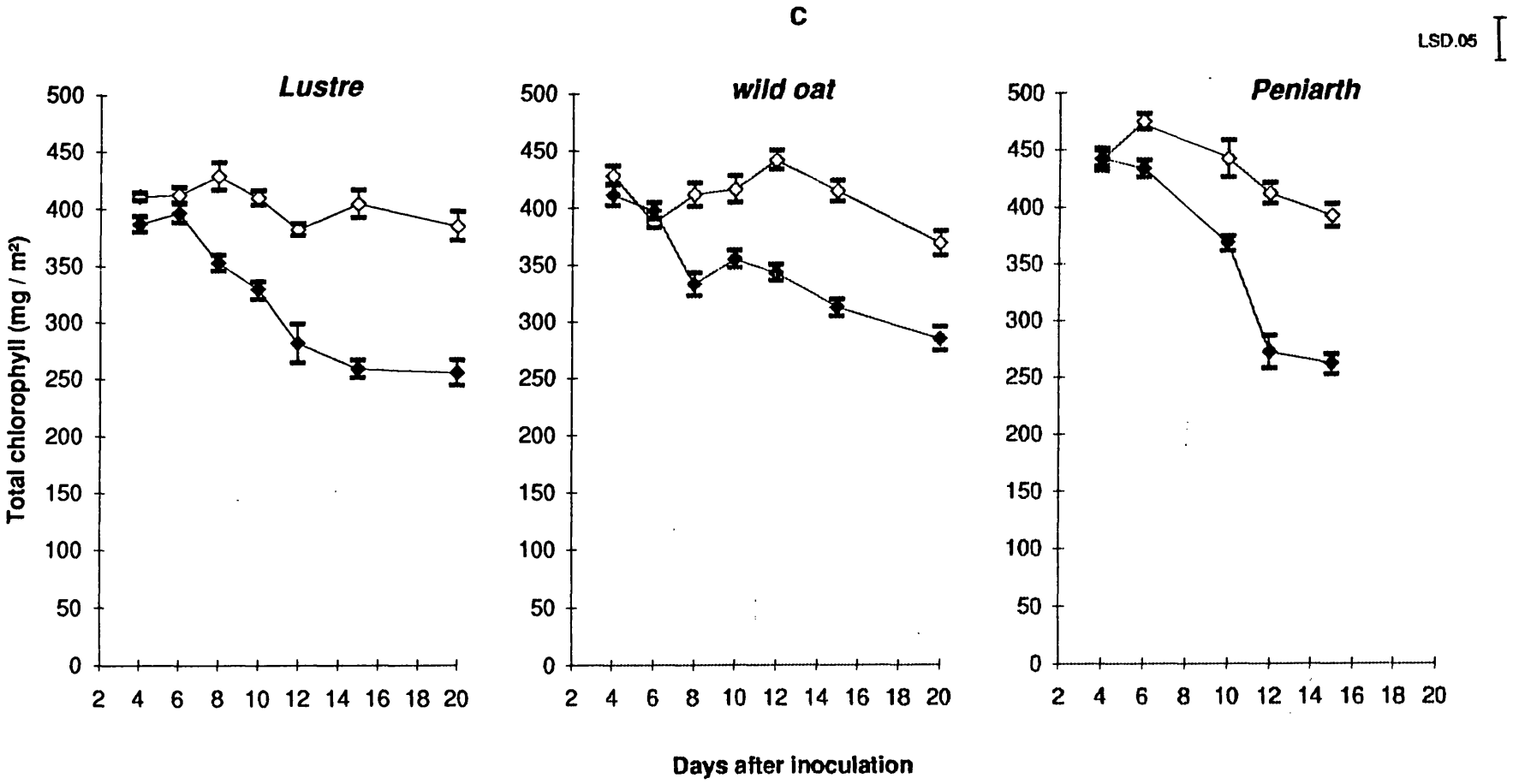
Chlorophylls a and b in uninfected leaf tissues of all three lines declined as the leaf matured but those in infected tissues showed a much greater reduction presumably due to the effects of infection in addition to the normal decline due to leaf maturation (Figs. 35A-B). The analysis of variance revealed significant differences in chlorophylls a and b between infected and uninfected leaves of each line during the course of infection.. The differences between infected and uninfected leaves, for chlorophyll a, became significant ($P < 0.05$) 6 days after inoculation in cv. Peniarth but not until 8 days after inoculation in cv. Lustre and wild oat (Fig. 35A). Differences, with respect to chlorophyll b, became significant ($P < 0.05$) around about 8 days after inoculation in cv. Peniarth, but not until 10 days after inoculation in cv. Lustre and wild oat (Fig. 35B). However the differences, for both chorophylls a and b, were always less marked in wild oat than in cvs Lustre and Peniarth at all stages of infection .

Total chlorophyll content of infected and uninfected leaves of each line are given in Table 11 and the results are plotted in Fig 35C. The analysis of variance shows significant differences ($P < 0.05$) between infected and uninfected leaves of each line, with the differences being more apparent in cvs Lustre and Peniarth than in wild oat as infection progressed. The overall effect of infection, at 15 days after inoculation, was to reduce total chlorophyll content of the infected leaves about 36 % in cv. Lustre, about 33 % in cv. Peniarth but only by about 25 % in wild oat. At 20 days after inoculation, there had been no further reductions in total chlorophyll of infected leaves in either cv. Lustre or wild oat .

5.3.4.2. Ratio of chlorophyll a : b

The ratios of chlorophyll a : b at each harvest are given in Tables 11 and 11A .


B
LSD.05
Lustre
wild oat
Peniarth
Chlorophyll b (mg / m²)
0
20
40
60
80
100
120
140
2 4 6 8 10 12 14 16 18 20
Days after inoculation


C
LSD.05
Lustre
wild oat
Peniarth
Total chlorophyll (mg / m²)
500
450
400
350
300
250
200
150
100
50
0
2 4 6 8 10 12 14 16 18 20
Days after inoculation

Although chlorophyll a appeared to decline more rapidly than chlorophyll b in the infected leaves, as shown in Figs. 35A and 35B, the ratios of chlorophyll a : b were not significantly different from those in the uninfected leaves of the two cultivars and wild oat during the course of the experiment (Tables 11 and 11A). This suggests that infection reduced chlorophylls a and b almost equally in the three lines .

5.3.5. Relation between Pgmax and parasite biomass production

The reduction in maximum gross photosynthesis during infection in each oat line was found to be linearly correlated ($P < 0.001$) with increasing parasite biomass, measured as fungal spore production in the infected leaves (Fig. 36) (Table 12). The results suggest that although infected leaves of wild oat supported the production of more fungal spores than those of cvs Lustre and Peniarth, over the course of the experiment, the maximum gross photosynthesis of the infected leaf of wild oat declined more slowly than that of the two cultivars (Fig. 36) .

5.4. The effects of infection on the fourth leaf on respiration, photosynthesis and chlorophyll content of the uninfected parts of that leaf

5.4.1. Introduction

The results of experiment 1 using the third leaf clearly suggest that infection resulted in increases in respiration and reductions in photosynthetic carbon fixation, the photochemical efficiency of photosynthesis at low light intensity, and chlorophyll contents in cvs Lustre, Peniarth and wild oat . Such reductions were more marked in the two cultivated oats than in wild oat. Thus, the fact that growth and development of wild oat were less reduced than those of the two cultivars by given levels of mildew, the tolerance could be due to the lower effect of that infection on photosynthesis. However a further factor affecting the more limited effects on growth and development in wild oat

TABLE 11

Ratio of *chlorophyll a* : *b* and *total chlorophyll* content of infected (I) and uninfected (C) third leaf of plants of the three oat lines. Each reading is a mean of four replicates ± SE.

Days after inoculation		Ratio *chl a :b*			*Total chlorophyll* (mg / m²)		
		Lustre	wild oat	Peniarth	Lustre	wild oat	Peniarth
4	I	3.56 ± 0.05	3.41 ± 0.07	2.91 ± 0.03	386.67 ± 6.91	411.35 ± 9.16	442.42 ± 6.78
	C	3.67 ± 0.06	3.59 ± 0.13	3.12 ± 0.06	410.78 ± 3.33	428.07 ± 8.49	441.40 ± 9.80
6	I	3.07 ± 0.08	3.51 ± 0.10	3.22 ± 0.07	396.15 ± 8.10	397.30 ± 7.20	433.20 ± 7.39
	C	3.29 ± 0.18	3.78 ± 0.20	3.19 ± 0.17	412.21 ± 6.75	386.75 ± 4.31	474.66 ± 6.78
8	I	2.98 ± 0.07	3.32 ± 0.19	-	352.48 ± 6.94	333.64 ± 10.16	-
	C	3.28 ± 0.11	3.61 ± 0.25	-	428.45 ± 11.81	411.39 ± 10.34	-
10	I	3.34 ± 0.37	2.65 ± 0.20	2.83 ± 0.11	329.52 ± 5.28	354.82 ± 7.54	368.71 ± 4.24
	C	3.48 ± 0.06	2.68 ± 0.05	2.72 ± 0.09	409.87 ± 6.15	415.87 ± 11.54	441.77 ± 16.26
12	I	3.04 ± 0.33	2.29 ± 0.07	2.10 ± 0.17	281.40 ± 17.16	342.92 ± 7.05	271.71 ± 14.54
	C	3.25 ± 0.35	2.50 ± 0.07	2.38 ± 0.07	382.05 ± 5.26	441.45 ± 8.34	411.74 ± 9.08
15	I	2.43 ± 0.06	2.23 ± 0.07	2.14 ± 0.08	258.80 ± 7.71	312.41 ± 3.55	261.77 ± 5.70
	C	2.56 ± 0.16	2.48 ± 0.15	2.50 ± 0.28	404.26 ± 11.92	414.48 ± 9.07	392.16 ± 10.19
20	I	2.42 ± 0.25	2.36 ± 0.23	-	255.39 ± 11.06	284.09 ± 10.50	-
	C	2.67 ± 0.22	2.26 ± 0.13	-	385.22 ± 12.63	368.37 ± 10.73	-

A : Analyses of variance

(1) Ratio *Chl a :b*

Source of variation	df	SS	MS	VR	P
Plant line	2	4.0068	2.0034	13.59	0.001
Treatment	1	0.7782	0.7782	5.28	0.05
Harvest	6	30.1545	5.0257	34.09	0.001
Plant line x Treatment	2	0.0681	0.0340	0.23	NS
Plant line x Harvest	10	6.5853	0.6585	4.47	0.001
Treatment x Harvest	6	0.5087	0.0848	0.58	NS
Plant line x Treatment x Harvest	10	0.6897	0.0690	0.47	NS
Residual	114	16.8074	0.1474		
Total	151	54.1299			

* $LSD_{.05}$ = 0.54

(2) *Total chlorophyll*

Source of variation	df	SS	MS	VR	P
Plant line	2	14662.5	7331.2	16.25	0.001
Treatment	1	241664.3	241664.3	535.64	0.001
Time	6	206351.4	34391.9	76.23	0.001
Plant line x Treatment	2	7469.7	3734.8	8.28	0.001
Plant line x Harvest	10	40893.6	4089.4	9.06	0.001
Treatment x Harvest	6	79493.1	13248.8	29.37	0.001
Plant line x Treatment x Harvest	10	9004.1	900.4	2.00	0.05
Residual	114	51432.9	451.2		
Total	151	574210.8			

* $LSD_{.05}$ = 33.70

* LSD values for plant line x treatment x harvest interactions.

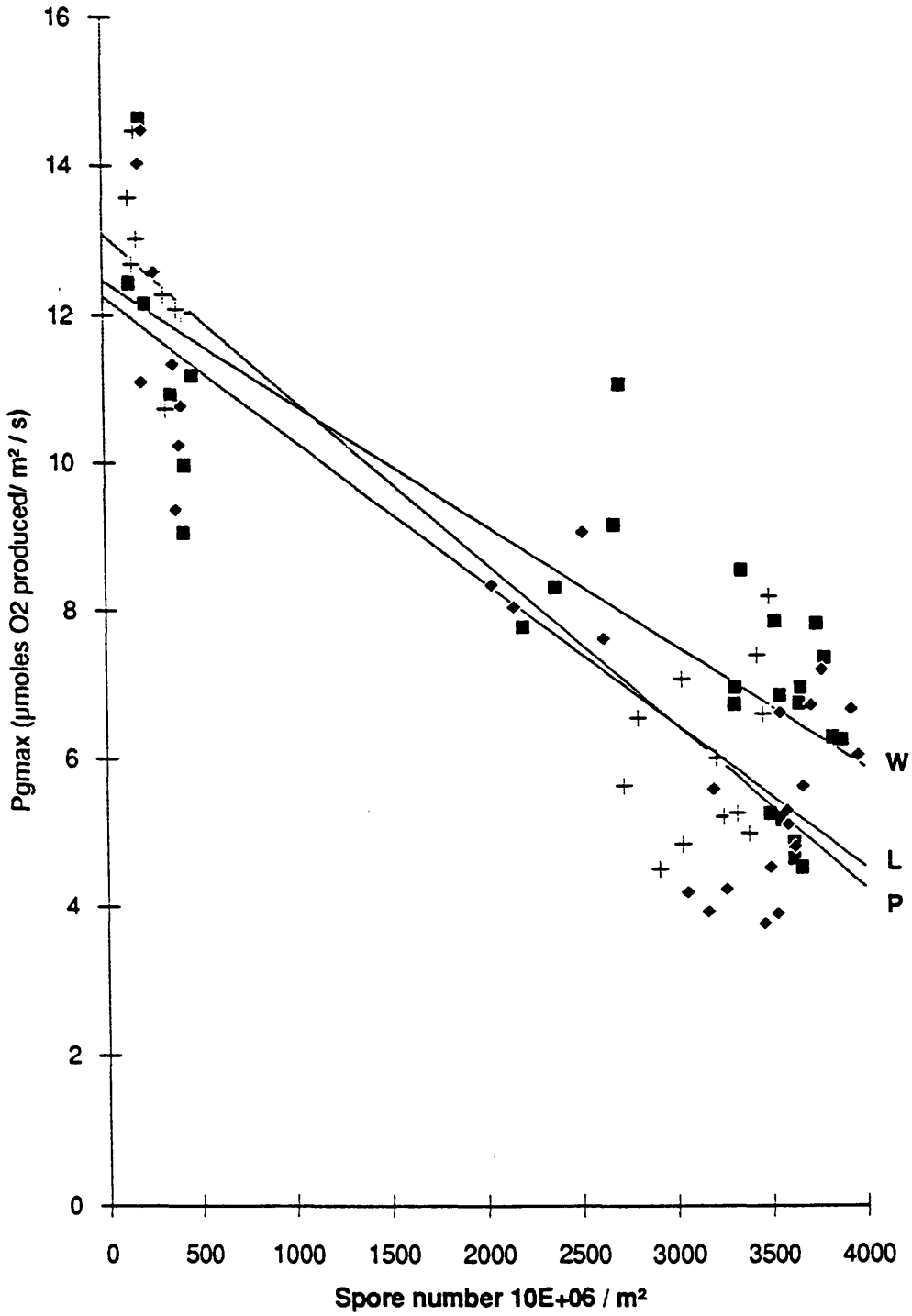


Fig. 36 : Relationship between gross photosynthesis and spore production on the infected third leaf of wild oat (■) and cvs Lustre (◆), Peniarth (+).

TABLE 12

Regression analyses of maximum gross photosynthesis (Pgmax) and fungal spore production in cvs Lustre, Peniarth and wild oat .

Regression parameters	Lustre	wild oat	Peniarth
Intercept (a)	12.257	12.465	13.092
Slope (b)	$1.925\ 10^{-09}$	$1.640\ 10^{-09}$	$2.203\ 10^{-09}$
Standard error for (a)	0.558	0.559	0.497
Standard error for (b)	$0.197\ 10^{-09}$	$0.193\ 10^{-09}$	$2.012\ 10^{-09}$
Coefficient of corr. (r^2)	0.786	0.734	0.870
Degree of freedom	26	26	18
F ratio	95.383	71.942	119.90

The regression analysis using the general equation for straight line $y = a + bx$.

could be the levels at which uninoculated tissues adjacent to infected tissues are able to compensate for reductions due infection. This aspect was investigated in the following experiment.

Experiment 2

5.4.2. Methods

Forty eight plants of cv. Lustre and of wild oat were grown until the fourth leaf blade had fully expanded. The fourth leaves from plants of each line were inoculated by applying single conidial isolates (from leaf segments in Petri dishes) to different parts of the leaf (Fig. 26) using a paint brush: sixteen plants of each line were inoculated in the middle part of the leaf (Leaf A), another sixteen plants were inoculated at the base and tip of the leaf leaving the middle region uninoculated (Leaf B) and the other sixteen plants were kept free from mildew (Leaf C) using fungicides, as described before. The infected and uninfected plants were then placed in the growth room. Four plants per line per treatment were sampled at 5 day intervals until 20 days after inoculation.

The results of experiment 1 applying the model of Marshall and Biscoe showed that infection did not appear to affect the physical (r_p) or carboxylation resistance (r_x) to CO_2 diffusion in both cv. Lustre and wild oat as the numerical value of θ did not change significantly. In this experiment the model of Rabinowitch was used instead to calculate Pgmax, α and Rd (Rd was simply deduced from the equation Pg = Pn + Rd).

5.4.3. Effects of infection on dark respiration

The dark respiratory rates of the infected middle region of leaf A, the uninoculated middle region of Leaf B and the middle region of leaf C, in the two lines, at different times after inoculation are plotted in Fig. 37.

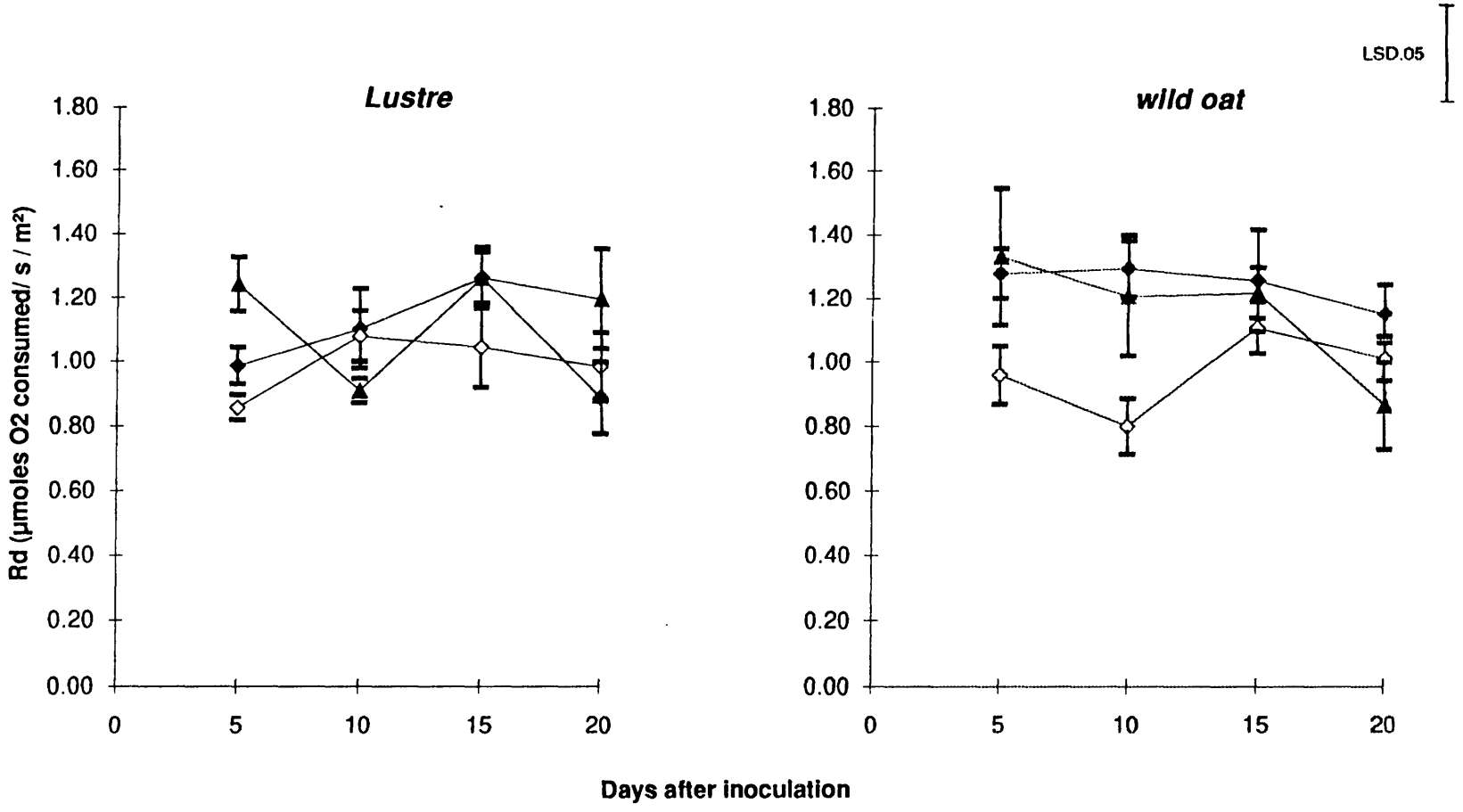


Fig. 37 : Changes in respiratory rates in infected (◆), adjacent uninoculated (▲) and uninoculated control (◇) leaf tissue of cv. Lustre and wild oat. Vertical bars represent standard errors.

The rates of respiration in the infected middle region of leaf A and in the uninoculated middle region of leaf B were generally higher than those of the uninoculated control leaf C in both cv. Lustre and wild oat throughout the experiment (Fig. 37). The LSD value shows that, in wild oat, the rate of respiration in the infected middle region of leaf A and the uninoculated middle region of leaf B were significantly higher ($P < 0.05$) than that in the control leaf C, from 5 to 10 days after inoculation, but thereafter the rate of respiration in both infected regions of leaves A and B (when the uninoculated region of leaf B had became infected) declined to the level of that in the control leaf C (Fig. 37). However, in cv. Lustre, the rate of respiration in the uninoculated region of leaf B was significantly higher ($P < 0.05$) than that in the control leaf C, during the initial 5 days after inoculation, but thereafter the rate of respiration in both infected regions of leaves A and B stayed higher but not significantly higher than that in the control leaf C (Fig. 37) .

5.4.4. Effects of infection on photosynthetic activity

5.4.4.1. Maximum gross photosynthesis

Maximum rates of gross photosynthesis (Pgmax) at different times after inoculation are plotted in Fig. 38 .

Five days after inoculation, the maximum rates of gross photosynthesis, expressed on a unit leaf area basis, in the uninoculated middle region of leaf B was slightly higher but not significantly higher than in the control leaf C, but thereafter it fell below that of the control in both lines (Fig. 38). Differences between the infected middle region of leaf A, the uninoculated middle region of leaf B and the uninoculated control leaf C became significant ($P < 0.05$) 10 days after inoculation, in both cv. Lustre and wild oat. Thereafter, when the middle region of leaf B became infected, the ability of both infected middle regions of leaves A and B to photosynthesise decreased more rapidly than that of the corresponding uninoculated controls, especially in cv. Lustre (Fig. 38). The total effect of infection was to decrease the maximum rates of

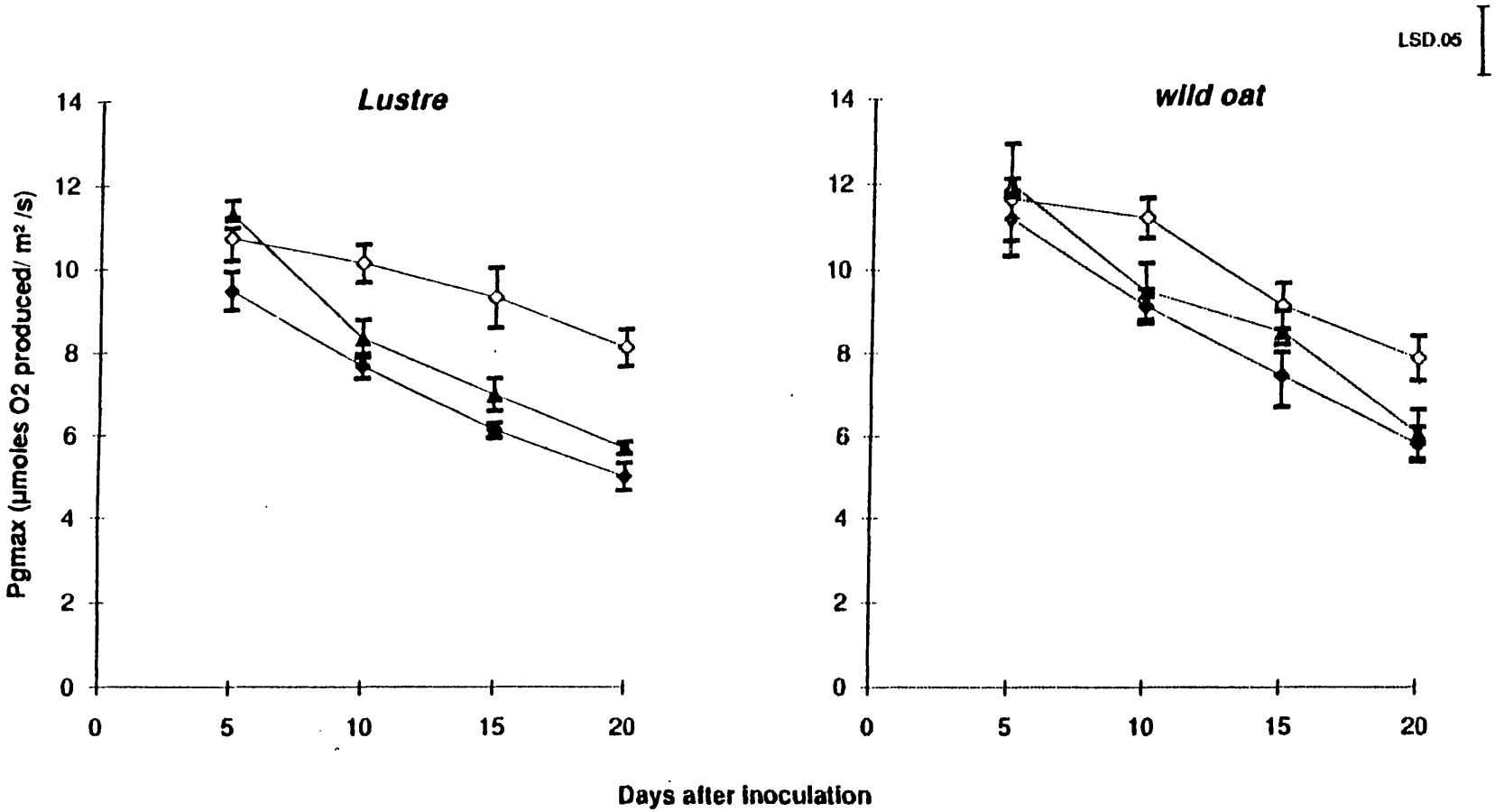


Fig. 38 : Maximum gross photosynthesis per unit area of infected (◆), adjacent uninoculated (▲) and uninoculated control (◇) leaf tissue of cv. Lustre and wild oat. Vertical bars represent standard errors.

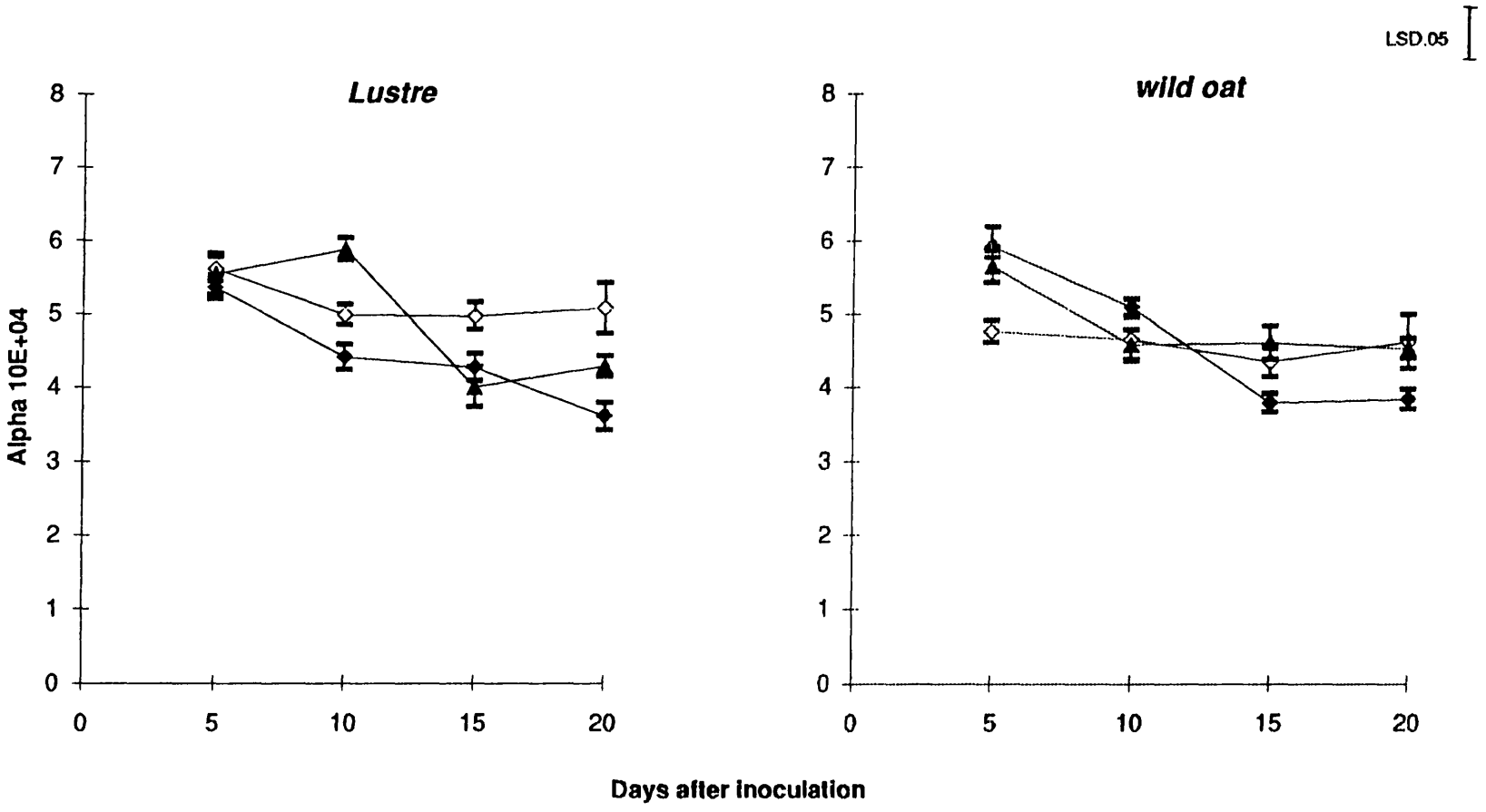


Fig. 39 : Photochemical efficiency of photosynthesis at low light intensity in infected (◆), adjacent uninoculated (▲) and uninoculated control (◇) leaf tissue of cv. Lustre and wild oat. Vertical bars represent standard errors.

gross photosynthesis, from 5 to 20 days after inoculation, in the middle region of leaf A by about 38 % in cv. Lustre, about 26 % in wild oat and that of the middle region of leaf B by about 29 % in cv. Lustre and about 23 % in wild oat.

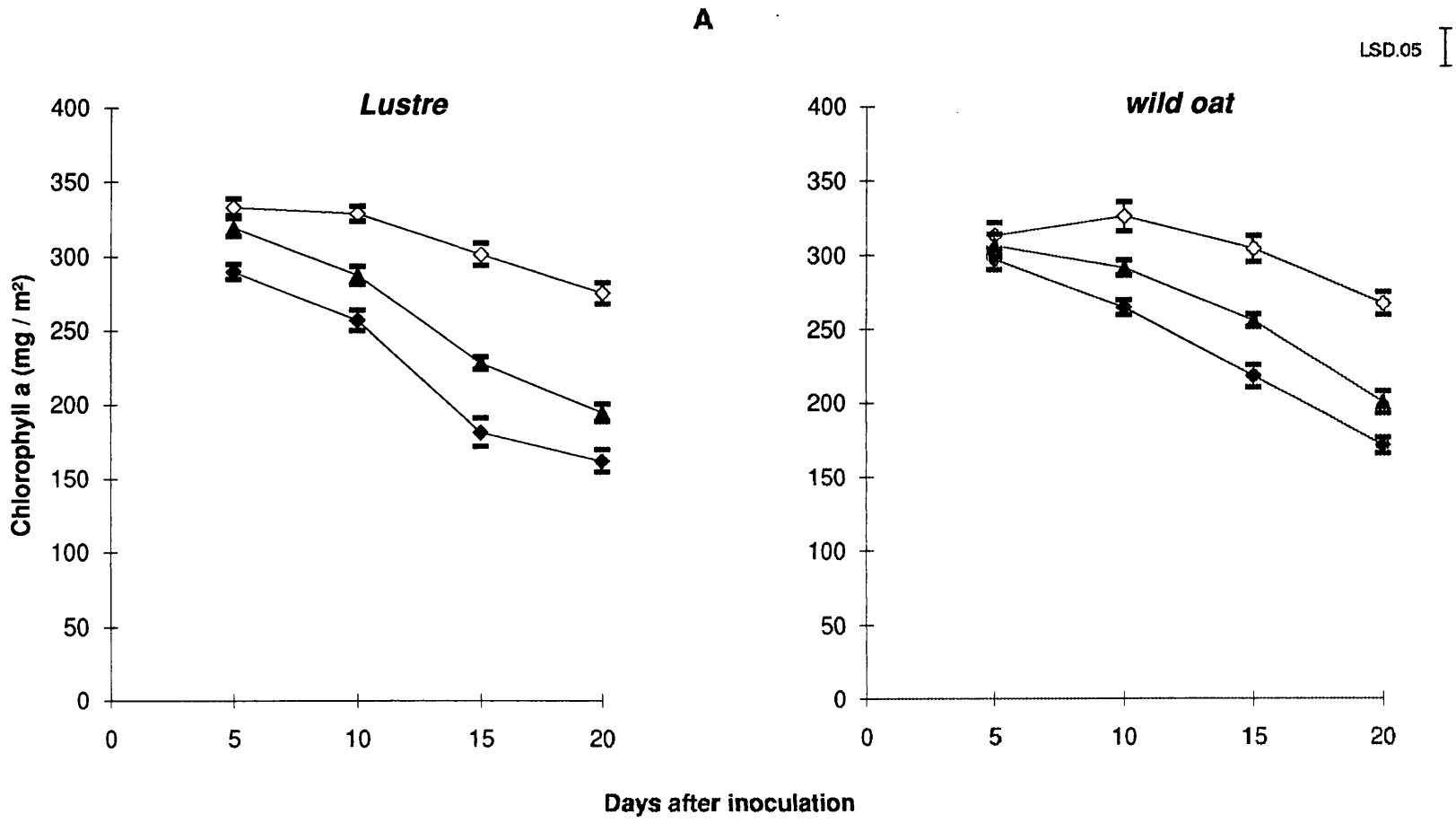
The maximum rates of gross photosynthesis per milligram of chlorophyll slightly decreased in the infected region of leaf A and uninoculated regions of leaf B and C as infection progressed and the differences between the three leaves were not significant in either cv. Lustre or wild oat at any stage of infection (results not given).

5.4.4.2. Photochemical efficiency of photosynthesis

Photochemical efficiencies of photosynthesis at low light intensity (α) at each sampling time are plotted in Fig. 39.

The curves show that the photochemical efficiency of photosynthesis in the middle region of control leaf C in both cv. Lustre and wild oat did not change over the 20 day period of the experiment, whereas that in the infected middle region of leaf A and the uninoculated region of leaf B did, especially in cv. Lustre (Fig. 39). In cv. Lustre, the photochemical efficiency in the infected middle region of leaf A was below that of the uninoculated control leaf C 5 days after inoculation, although the reduction did not become significant ($P < 0.05$) until 15 days after inoculation, whereas the photochemical efficiency in the uninoculated middle region of leaf B was significantly stimulated 10 days after inoculation followed thereafter by a reduction below that of the control leaf C. At 15 days after inoculation, when the uninoculated middle region of leaf B became infected, the photochemical efficiency in the infected middle region of leaf B became significantly different ($P < 0.05$) from that of the control (Fig. 39). In wild oat, the photochemical efficiency in the infected middle region of leaf A and the uninoculated middle region of leaf B were significantly higher than that of the controls 5 days after inoculation. Thereafter, the photochemical efficiency in the infected middle region of leaf A fell below that of the uninoculated control, and the differences did not become significant ($P < 0.05$) until around 20 days after inoculation. In contrast, the photochemical efficiency in the uninoculated middle region of leaf B never fell below that in control leaf C (Fig 39). The total effect of infection was to decrease the

Figures 40A-C : Chlorophyll a (A), chlorophyll b (B) and total chlorophyll (C) content of Erysiphe graminis infected (◆), adjacent uninoculated (▲) and uninoculated control (◇) leaf tissue of cv. Lustre and wild oat. Vertical bars represent standard errors.


A
LSD.05
Lustre
wild oat
Chlorophyll a (mg / m²)
400
350
300
250
200
150
100
50
0
0
5
10
15
20
Days after inoculation

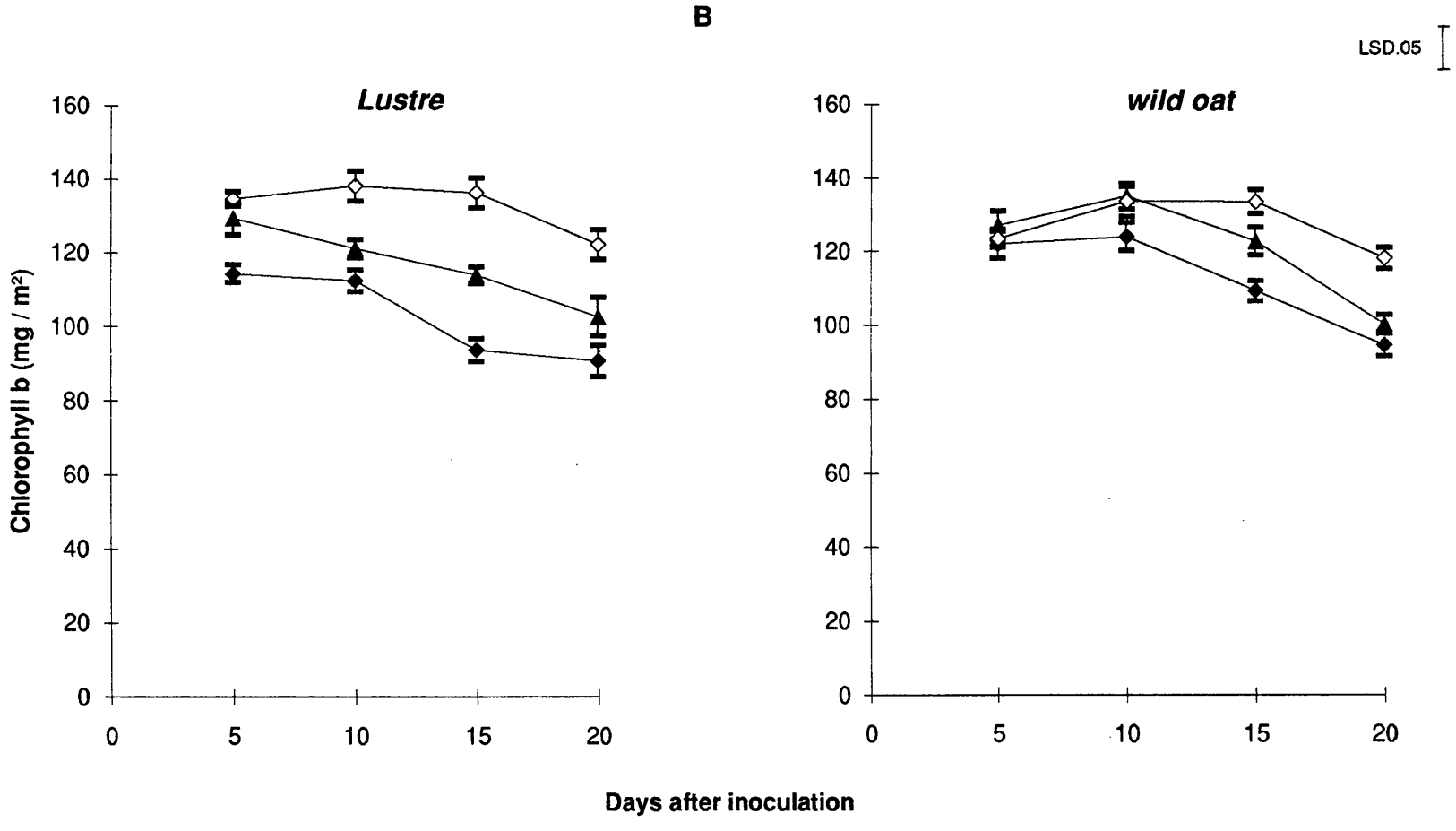
photochemical efficiency of photosynthesis in the middle region of leaf A by about 29 % in cv. Lustre but about 17 % in wild oat and that of the uninoculated middle region of leaf B by about 16 % in cv. Lustre but only about 2 % in wild oat .

5.4.5. Effects of infection on chlorophyll content

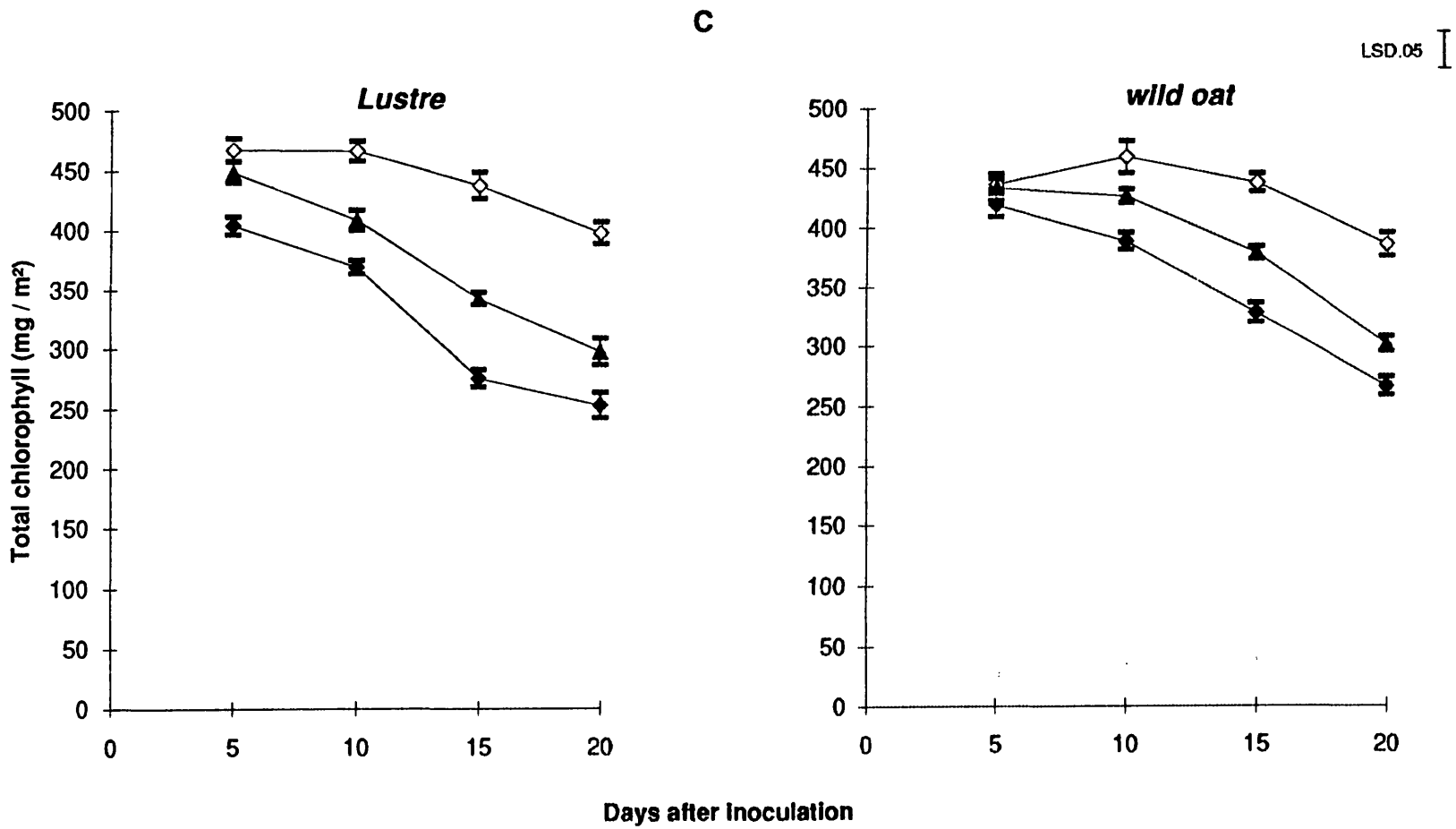
5.4.5.1. Chlorophylls a and b and total chlorophyll

Changes in chlorophylls a and b in the infected middle region of leaf A, the uninoculated middle region of leaf B and the middle region of leaf C, of each line, at each sampling time are plotted in Figs. 40A -B .

Chlorophylls a and b in the control leaf C in the two lines showed a slow decline throughout the course of the experiment but those in the infected middle region of leaf A and the uninoculated middle region of leaf B showed a rapid decline as infection progressed (Figs. 40A-B). The LSD value shows significant differences in the amounts of chlorophylls a and b between the infected middle region of leaf A, the uninoculated middle region of leaf B and the control leaf C in the two lines. In cv. Lustre, the levels of chlorophylls a and b in the infected middle region of leaf A were significantly lower ($P < 0.05$) than in the control leaf C from around 5 days after inoculation whereas those in the uninoculated region of leaf B did not become significantly lower ($P < 0.05$) than in the control leaf C until around 10 days after inoculation, with the differences becoming greater as infection progressed (Figs. 40A-B). In contrast, the levels of chlorophylls a and b in the infected middle region of leaf A of wild oat were not observed to be significantly reduced ($P < 0.05$) until around 10 days after inoculation for chlorophyll a and until 15 days after inoculation for chlorophyll b. Furthermore, the differences between the uninoculated middle region of leaf B and the control leaf C of wild oat were not observed to be significant ($P < 0.05$) until 10 days after inoculation for chlorophyll a and none of the differences were significant until 15 days after inoculation for chlorophyll b (Figs. 40A -B) .


B
LSD.05
Lustre
wild oat
Chlorophyll b (mg / m²)
160
140
120
100
80
60
40
20
0
0
5
10
15
20
Days after inoculation

C


LSD.05
Lustre
wild oat
Total chlorophyll (mg / m²)
500
450
400
350
300
250
200
150
100
50
0
0
5
10
15
20
Days after inoculation

Total chlorophyll in the infected middle region of leaf A and in the uninoculated middle regions of leaves B and C are given in Table 13 and plotted in Fig. 40C. Reductions in total chlorophyll content of the infected middle region of leaf A and the uninoculated middle region of leaf B were due to reductions in both chlorophylls a and b. The total effect of infection, from 5 to 20 days after inoculation, was to reduce total chlorophyll in the infected middle region of leaf A by about 36 % in cv. Lustre, about 31 % in wild oat, and that of the uninoculated middle region of leaf B by about 25% in cv. Lustre and about 22 % in wild oat.

5.4.5.2. Ratio of chlorophyll a : b

The ratios of chlorophyll a : b at different times after inoculation are given in Tables 13 and 13A.

The ratios in the infected middle region of leaf A and in the uninoculated middle region of leaf B were reduced in both cv. Lustre and wild oat (Table 13). However, none of the differences between the infected middle region of leaf A, the uninoculated middle region of leaf B and the control leaf C were observed to be significant ($P < 0.05$) until 15 days after inoculation (Table 13A).

TABLE 13

Ratio of *chlorophyll a* : *b* and *total chlorophyll* content of infected (I), adjacent uninoculated (I*) and uninoculated control (C) leaf tissue of plants of the two oat lines. Each reading is a mean of four replicates ± SE.

Days after inoculation		Ratio *chl a :b*		*Total chlorophyll* (mg / m²)	
		Lustre	wild oat	Lustre	wild oat
	I	2.54 ± 0.02	2.44 ± 0.05	404.07 ± 7.43	419.02 ± 10.29
5	I*	2.48 ± 0.06	2.42 ± 0.02	448.46 ± 8.92	433.30 ± 11.21
	C	2.47 ± 0.02	2.54 ± 0.04	467.41 ± 4.80	436.27 ± 4.56
	I	2.29 ± 0.02	2.25 ± 0.08	369.18 ± 5.91	388.22 ± 7.00
10	I*	2.37 ± 0.01	2.26 ± 0.05	408.39 ± 8.58	426.03 ± 5.77
	C	2.39 ± 0.09	2.43 ± 0.04	466.52 ± 4.30	458.92 ± 13.51
	I	1.94 ± 0.05	2.00 ± 0.06	275.10 ± 7.21	327.34 ± 3.01
15	I*	2.01 ± 0.05	2.09 ± 0.03	342.16 ± 4.40	378.19 ± 5.43
	C	2.29 ± 0.04	2.28 ± 0.03	447.43 ± 10.97	437.04 ± 7.73
	I	1.79 ± 0.03	1.83 ± 0.08	252.68 ± 10.65	265.75 ± 4.55
20	I*	1.90 ± 0.04	2.01 ± 0.11	297.18 ± 10.86	300.61 ± 6.14
	C	2.25 ± 0.07	2.18 ± 0.07	397.43 ± 7.12	385.22 ± 7.91

I : infected region of leaf A
I* : adjacent uninoculated region of leaf B
C : uninoculated control Leaf C

A : Analyses of variance

(1) Ratio *Chl a : b*

Source of variation	df	SS	MS	VR	P
Plant line	1	0.00667	0.00667	0.44	NS
Treatment	2	1.00396	0.50198	33.16	0.001
Harvest	3	3.37250	1.12417	74.26	0.001
Plant line x Treatment	2	0.01521	0.00760	0.50	NS
Plant line x Harvest	3	0.08750	0.02917	1.93	0.1
Treatment x Harvest	6	0.40437	0.06740	4.45	0.01
Plant line x Treatment x Harvest	6	0.11813	0.01969	1.30	NS
Residual	72	1.09000	0.01514		
Total	95	6.09833			

* $LSD_{.05}$ = 0.19

(2) *Total chlorophyll*

Source of variation	df	SS	MS	VR	P
Plant line	1	2392.0	2392.0	7.73	0.01
Treatment	2	174922.2	87461.1	282.56	0.001
Time	3	192488.3	64162.8	207.29	0.001
Plant line x Treatment	2	10068.0	5034.0	16.26	0.001
Plant line x Harvest	3	2274.6	758.2	2.45	0.1
Treatment x Harvest	6	23661.1	3943.5	12.74	0.001
Plant line x Treatment x Harvest	6	4096.0	682.7	2.21	0.1
Residual	72	22286.4	309.5		
Total	95	432188.7			

* $LSD_{.05}$ = 56.73

* LSD values for plant line x treatment x harvest interactions.

5.5. The effects of infection on the fourth leaf on chlorophyll fluorescence changes in the uninoculated parts of that leaf

5.5.1. Introduction

The results obtained in the previous section showed that infection results in reductions in the rates of photosynthesis in the infected and also the adjacent uninoculated leaf tissue particularly in cv. Lustre, and also, although to a lesser extent, in wild oat. The following section describes measurements of chlorophyll fluorescence induction which were carried out to investigate the probable mechanisms by which photosynthesis was reduced in wild and cultivated oat.

5.5.2. Methods

Forty eight seedlings of cv. Lustre and wild oat were raised in 15 cm plastic pots, two seedling per pot. Plants were grown until the fourth leaf had fully expanded. The fourth leaves from plants of each line were inoculated and incubated as described for the photosynthesis measurements in the previous experiment (see section 5.4.2.). Four plants per line per treatment were sampled at 5 day intervals until 20 days after inoculation. At each sampling time, the infected leaves A and B and the uninoculated control leaf C (see Fig. 26) were excised and a leaf segment of 30 mm length was cut from the middle region of each leaf and used for chlorophyll fluorescence measurements. The experiment was carried out twice and since both gave essentially the same results, the detailed results of one only are reported.

5.5.3. Effects of infection on chlorophyll fluorescence

Chlorophyll fluorescence was examined at different times after inoculation. Photochemical (qQ) and non-photochemical (qE) components of fluorescence quenching were determined from the fluorescence induction curves (see Materials and Methods, section 2.9.2.2., Fig. 6b) and were used to examine the

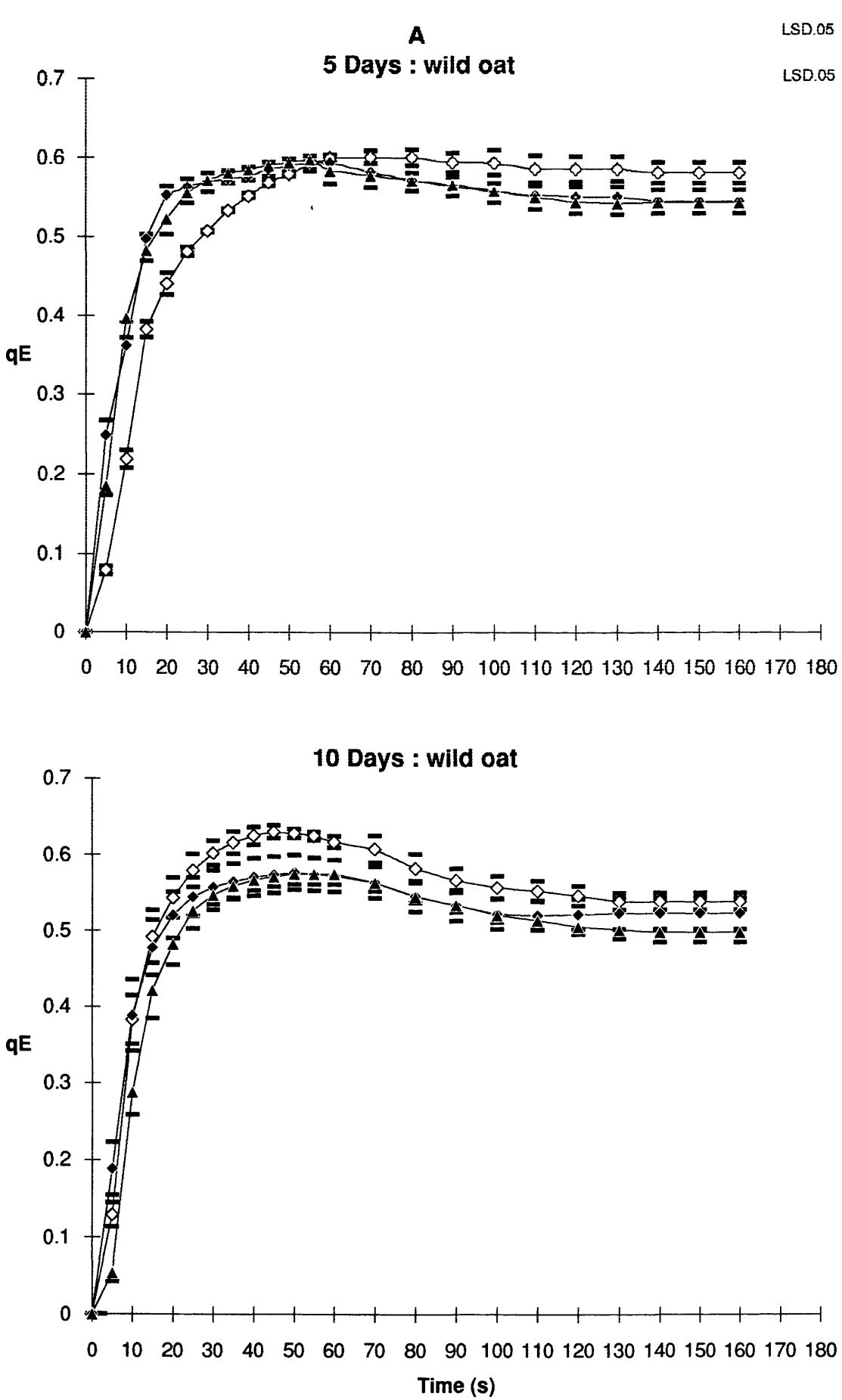
relationship between fluorescence quenching parameters and photosynthetic electron transport. The fluorescence parameter qQ was determined in the same way as Genty *et al.* (1989).

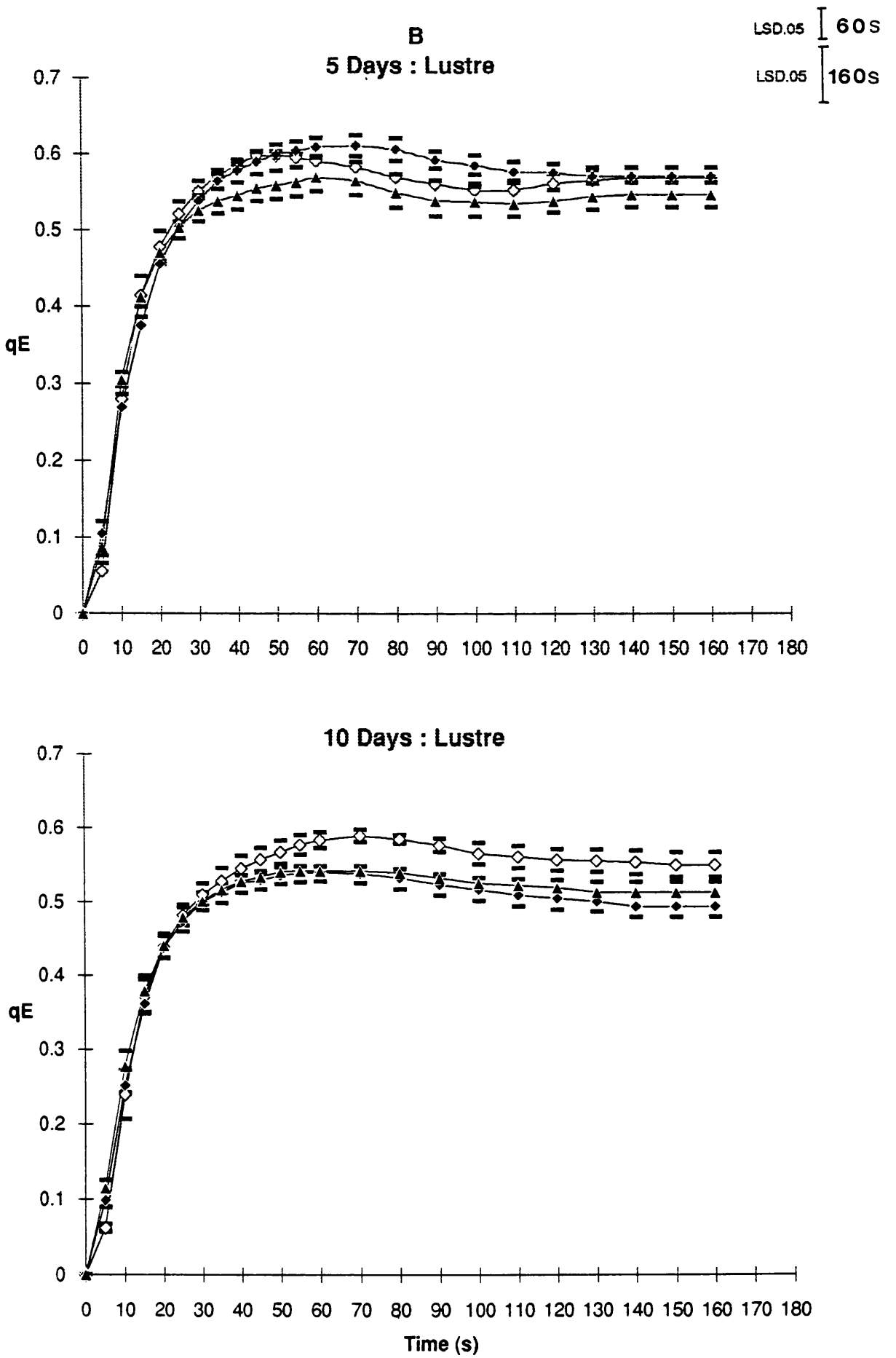
5.5.3.1. Effects of infection on non-photochemical fluorescence quenching , qE

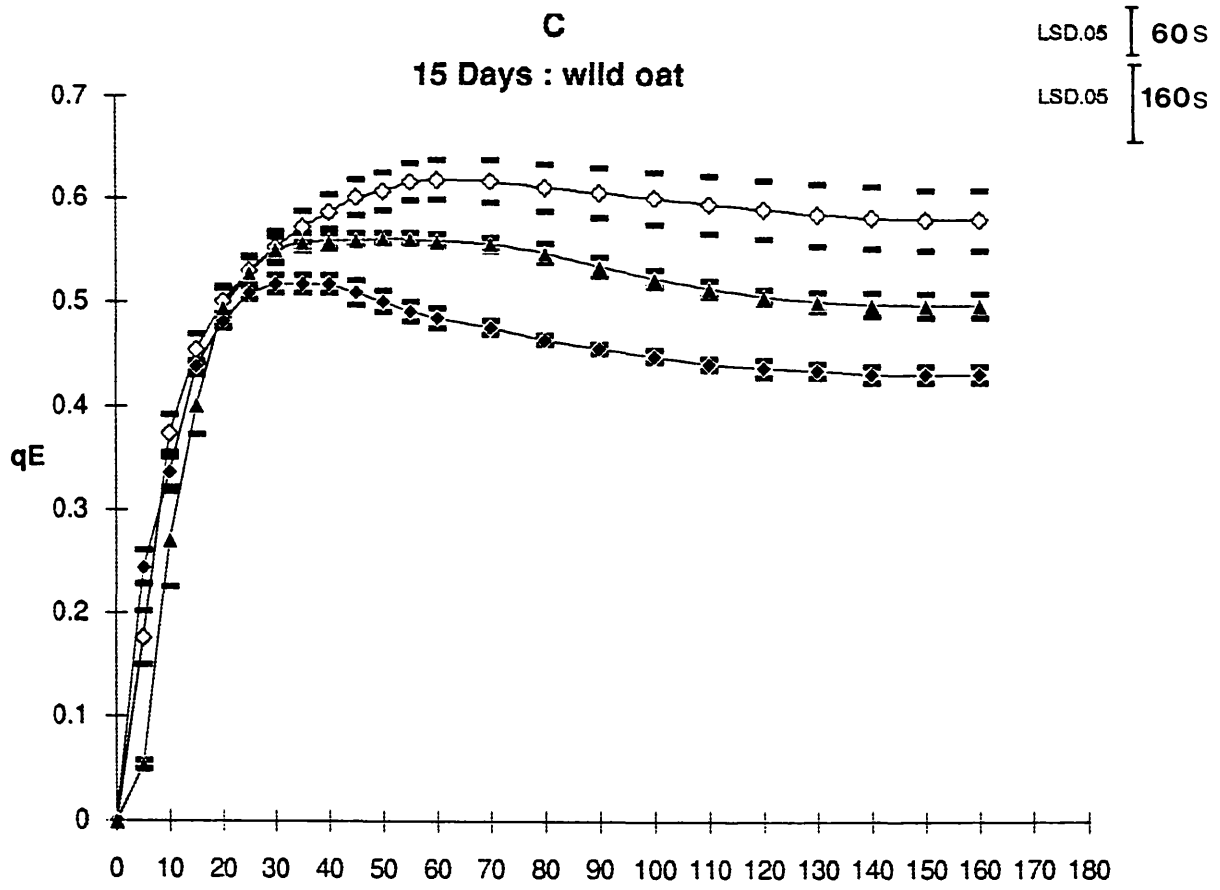
Non-photochemical fluorescence quenching (qE) in the infected middle region of leaf A, the uninoculated middle regions of leaf B and control leaf C are presented in Figs. 41A-D .

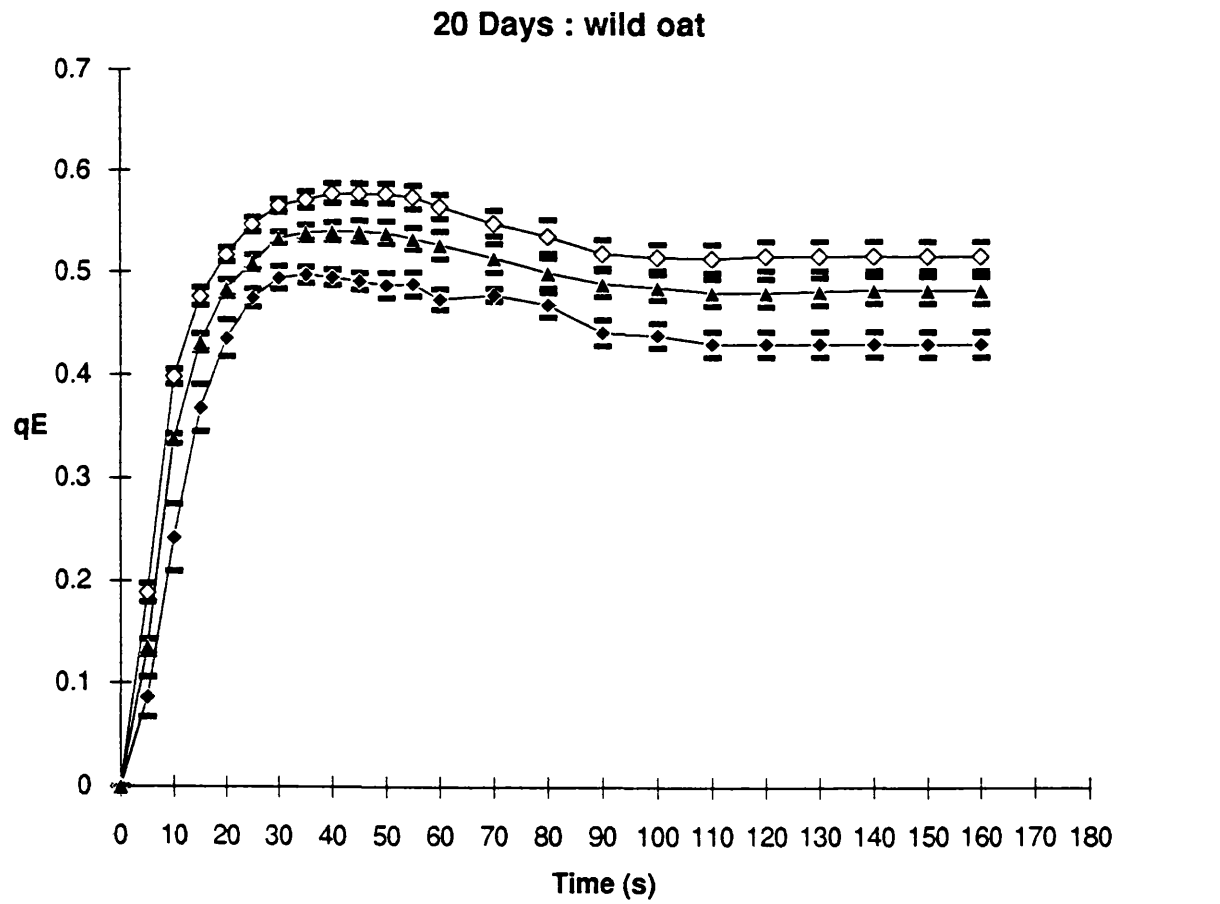
The qE value calculated as the ratio F1/Fmo (see Materials and Methods, section 2.9.2.2.) gives some indication about the proton motive force across the thylakoid membrane (Horton, 1983). The qE values obtained after 60 s (time of maximum fluorescence quenching) and 160 s (steady state fluorescence) of fluorescence induction were chosen for comparison between infected and uninoculated leaf tissue in each line. Five and ten days after inoculation, the fluorescence ratio (F1/Fmo) in the infected middle region of leaf A and the uninoculated regions of leaves B and C in the two lines reached the maximum level after about 60s, which was then followed by a quenching of fluorescence to a steady state at 160s (Figs. 41A-B). At 5 days after inoculation, there were no significant differences between the values of qE at 60 s and 160 s in the infected and uninoculated leaf tissue in each line, but by 10 days after inoculation, the value of qE at 60 s in the uninoculated control leaf C was significantly higher ($P < 0.05$) than that in the infected region of leaf A and uninoculated region of leaf B in wild oat and cv. Lustre (Figs. 41A-B). However, there were no significant differences between the qE value at 160 s in the infected region of leaf A and the uninoculated regions of leaves B and C in either cv. Lustre or wild oat. The differences in qE after 60 s and 160 s became more pronounced as infection progressed. This probably indicates that the proton gradient across the thylakoid membrane was not altered in the infected tissue of leaf A and the uninoculated tissue of leaf B until around 10 days after inoculation, when it began to decrease slowly in both cv. Lustre and wild oat. By 15 days and 20 days after inoculation, the fluorescence ratio (F1/Fmo) decreased slowly from 60 s to 160 s in the control leaf C, whereas in the

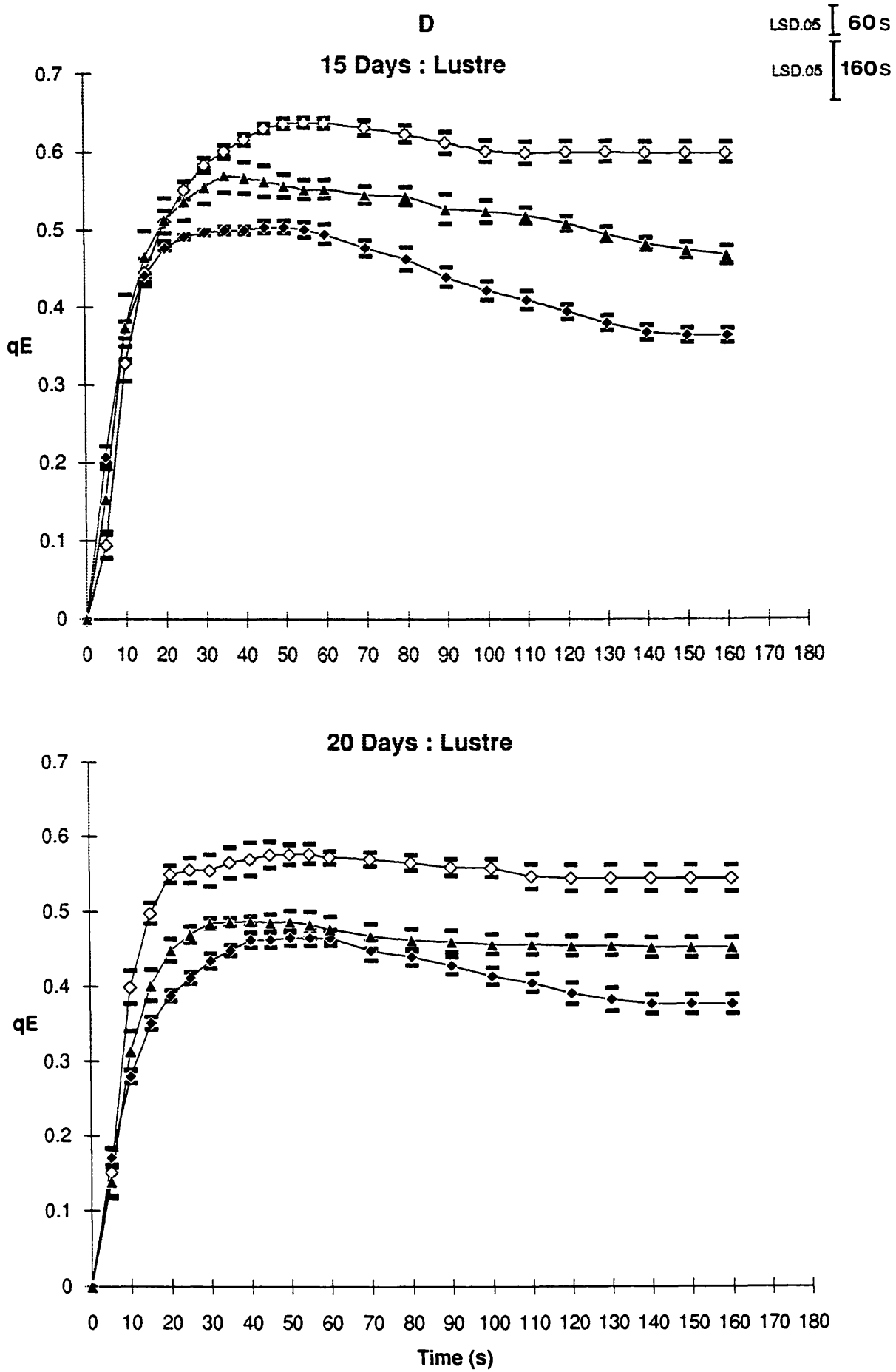
Figures 41A-D : The time course of fluorescence quenching qE determined in Erysiphe graminis infected (◆), adjacent uninoculated (▲) and uninoculated control (◇) leaf tissue of cv. Lustre and wild oat, between 0 and 160 s of fluorescence induction. Vertical bars represent standard errors.


A
5 Days : wild oat
LSD.05 60 S
LSD.05 160 S
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
10 Days : wild oat
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
Time (s)


B
5 Days : Lustre
LSD.05 60s
LSD.05 160s
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
10 Days : Lustre
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
Time (s)


C
15 Days : wild oat
LSD.05 60s
LSD.05 160s
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180


20 Days : wild oat
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
Time (s)


D
LSD.05 60s
LSD.05 160s
15 Days : Lustre
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
20 Days : Lustre
qE
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0 10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
Time (s)

infected regions of leaves A and B (when the uninoculated region of leaf B had became infected), it reached a maximum level earlier (around about 35 to 40 s in both lines) than in the uninoculated controls (around about 60 s in both lines) and then it decreased rapidly. The LSD values show that the qE values at 60 s and 160 s in the infected regions of leaves A and B were significantly lower ($P < 0.05$) than those in the control leaf C; this was most noticeable in cv. Lustre (Figs. 41C-D) .

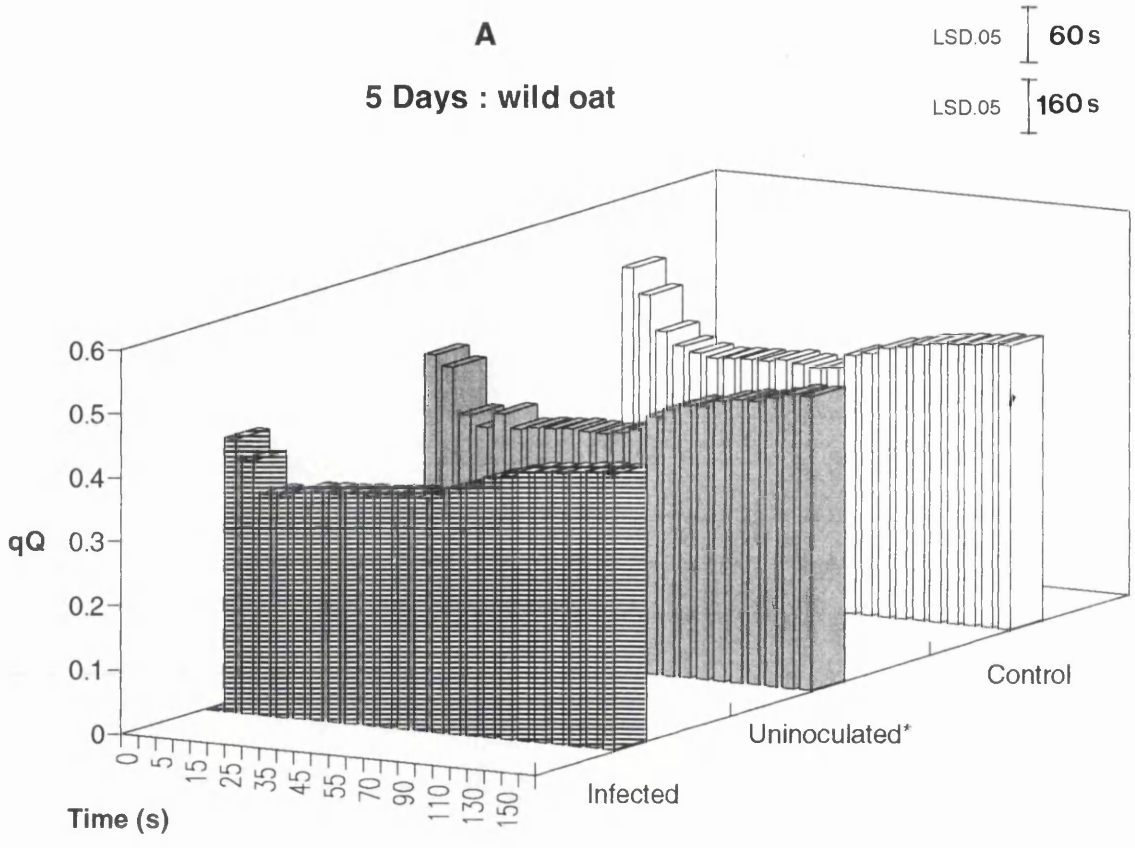
These results suggest that the proton gradient across the thylakoid membrane in the control leaf tissue decreased slowly with leaf age, whereas in the infected and adjacent uninoculated tissue, it decreased rapidly due to the additional effects of infection upon age effects. Furthermore, there were no significant differences in the infection-dependent decrease of qE when leaves were infected as leaf A and leaf B in both lines. The effect of infection on qE appeared to be more pronounced in cv. Lustre than it was in wild oat .

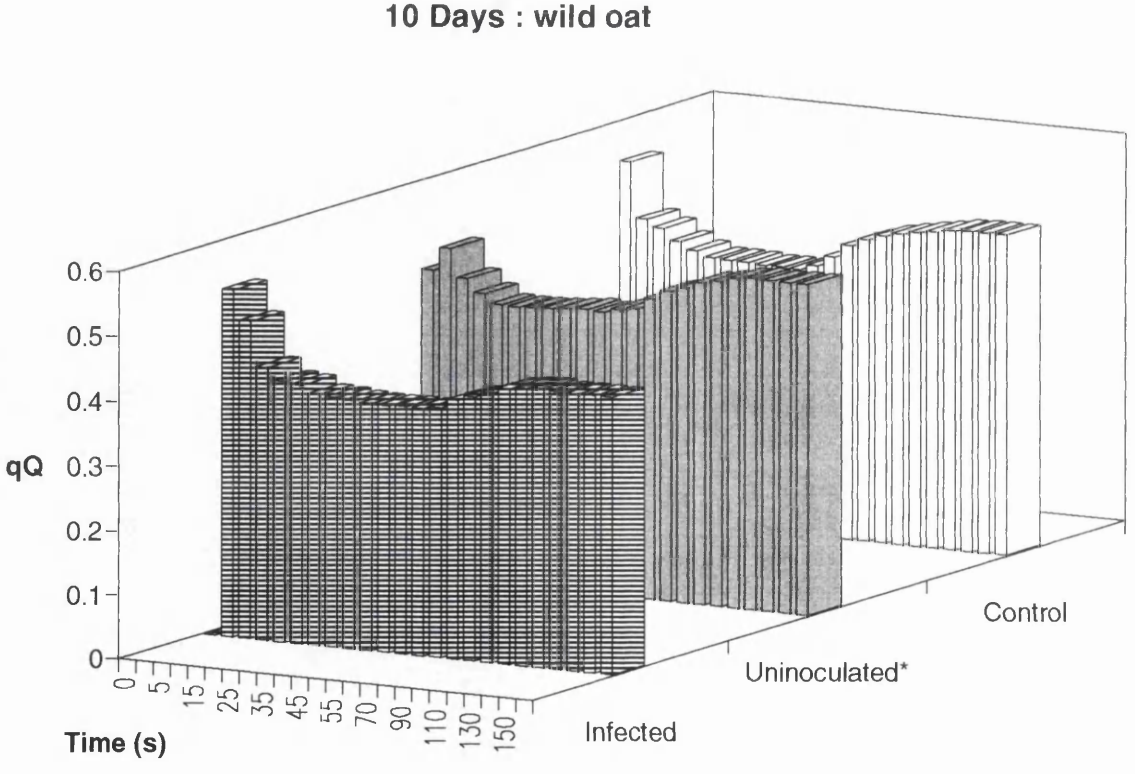
5.5.3.2. Effects of infection on photochemical fluorescence quenching , qQ

Photochemical fluorescence quenching (qQ) in the infected region of leaf A, the uninoculated region of leaf B and control leaf C are presented in Figs. 42A-D .

The qQ value calculated as F2/Fmt (see Materials and Methods, section 2.9.2.2.) estimates the oxidation state of the Q_B site of the electron transport chain (Horton, 1983). The qQ values after 60 s and 160 s of fluorescence induction were also compared in the infected and uninoculated leaves of each line. At 5 days after inoculation, the qQ values at 60 s and 160 s in the infected region of leaf A were significantly less ($P < 0.05$) than those in the uninoculated region of leaf B or in the control leaf C in cv. Lustre (Fig. 42B). However, the corresponding qQ values in the infected region of leaf A in wild oat were not different from those in the uninoculated region of leaf B or in the control leaf C (Fig. 42A). This suggests that in cv. Lustre, 5 days after inoculation, Q_B in the infected leaf tissue was not fully re-oxidised as in the control tissue after 60 s and 160 s of fluorescence induction. However, there were no significant differences between the qQ values at 60 s and 160 s in the uninoculated region of leaf B and the control leaf C in the two lines (Figs. 42A-B). Ten days after

Figures 42A-D : The time course of fluorescence quenching qQ determined in Erysiphe graminis infected, adjacent uninoculated (*) and uninoculated control leaf tissue of cv. Lustre and wild oat, between 2 and 160 s of fluorescence induction.





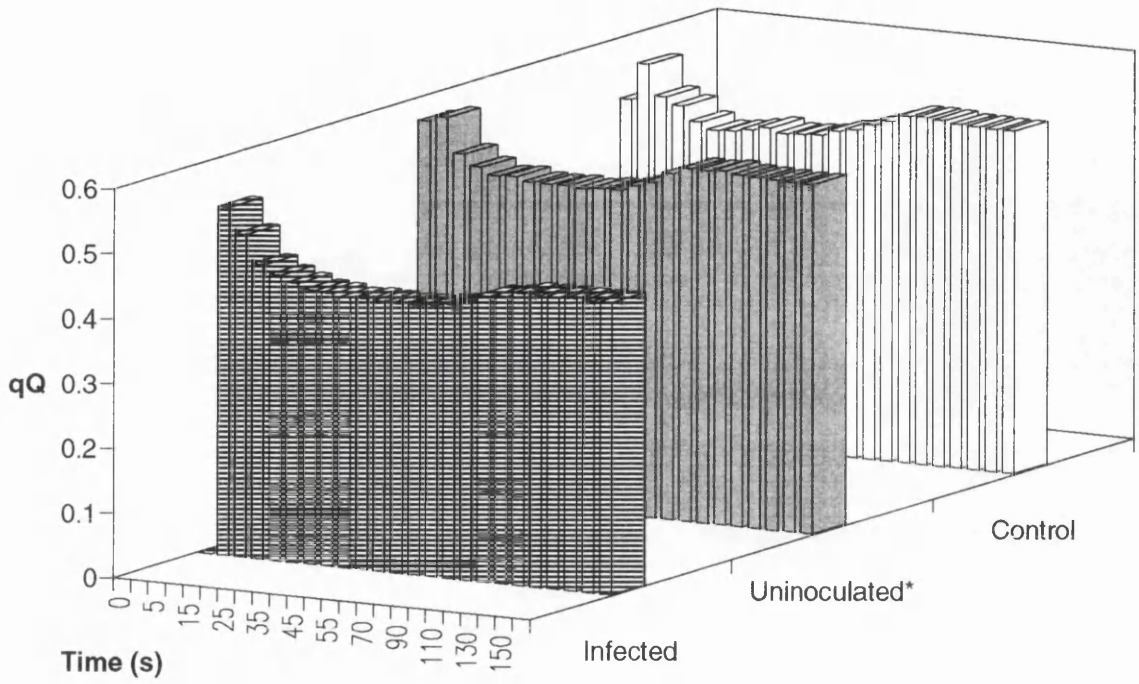
* adajcent uninoculated tissue of infected leaf

B

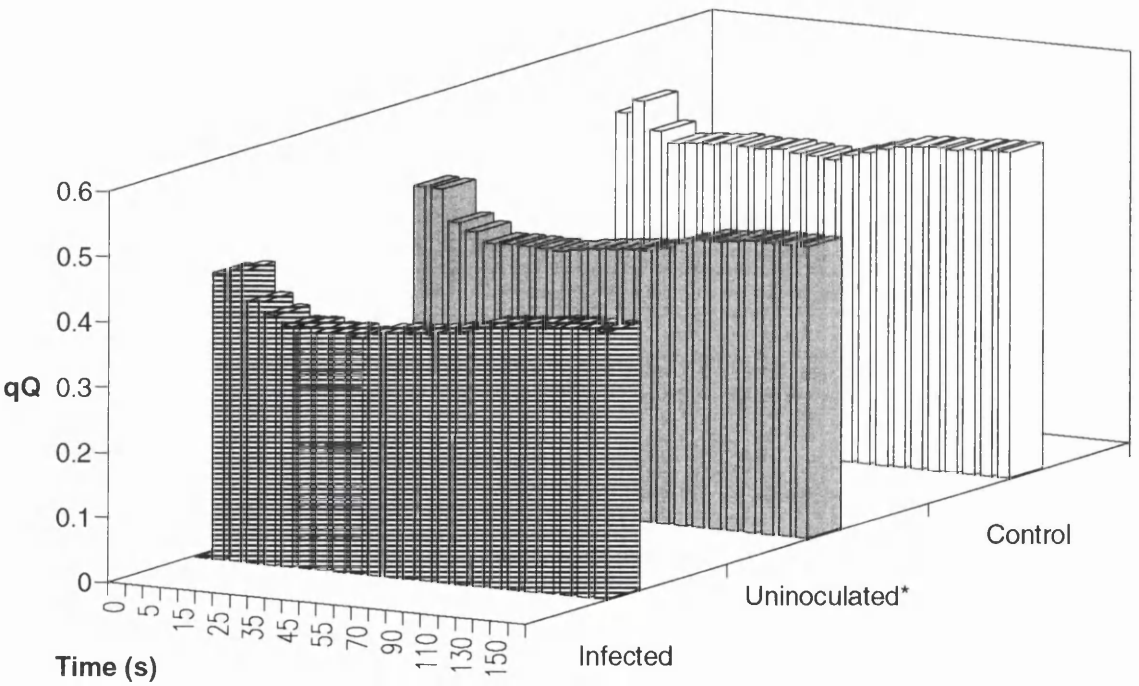
LSD.05 60s

LSD.05 160s

5 Days : Lustre



10 Days : Lustre

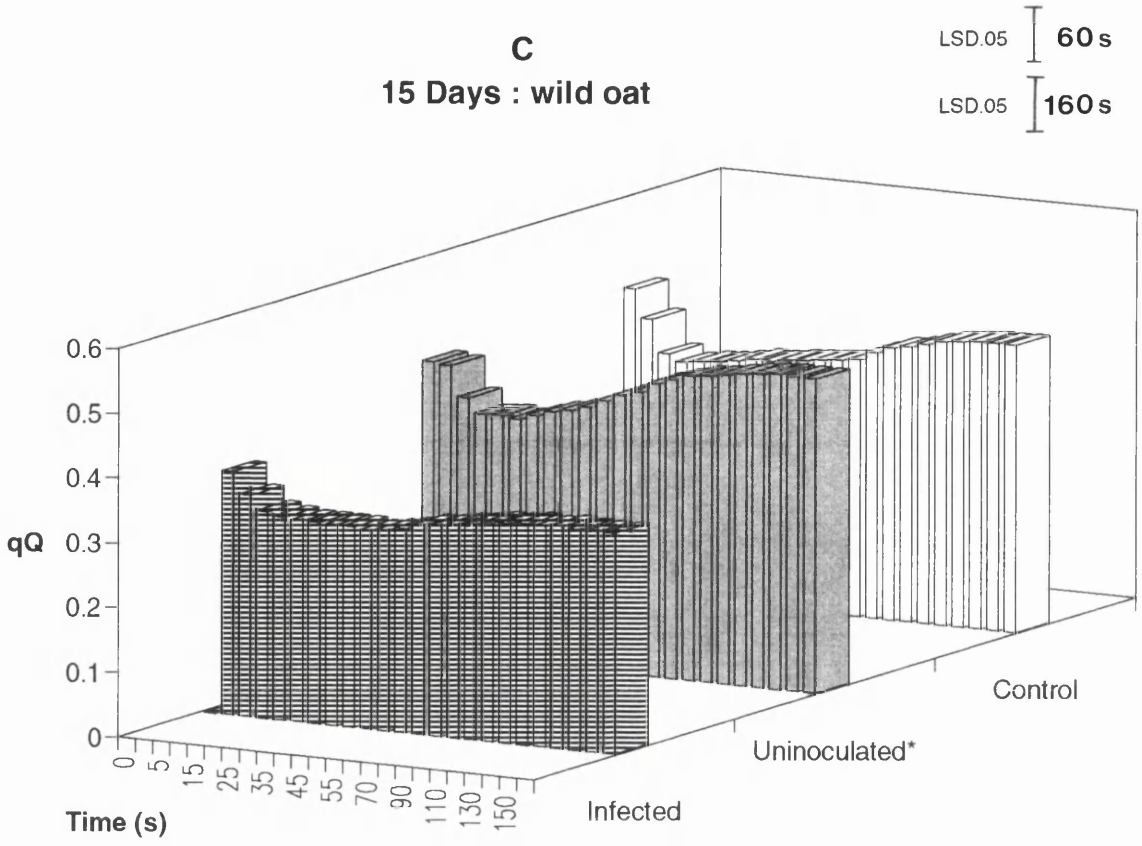


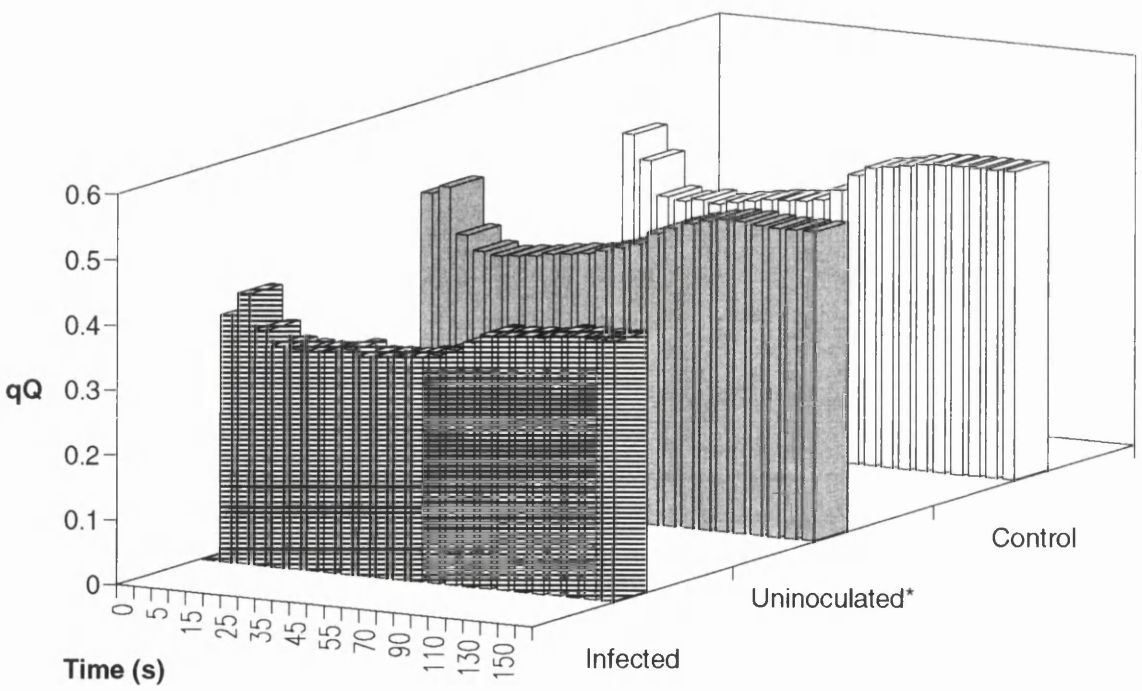
* adjacent uninoculated tissue of infected leaf

inoculation, the qQ values at 60 s and 160 s in the infected region of leaf A of wild oat became significantly lower ($P < 0.05$) than those in the uninoculated region of leaf B, and the control leaf C (Fig. 42A) whereas in cv. Lustre, the qQ values in both, the infected region of leaf A and the uninoculated region of leaf B, were significantly lower ($P < 0.05$) than those in the uninoculated control leaf C (Fig. 42B). These differences were maintained until 20 days after inoculation when the experiment was terminated. At 15 and 20 days after inoculation, a decrease in the qQ value in the infected region of leaf A occurred during the initial 2 s of fluorescence induction in the two lines, which was more apparent in cv. Lustre than in wild oat, with further decreases at 60 s and 160 s especially in cv. Lustre (Figs. 42C-D).

The high values of qQ in the uninoculated control leaf tissue suggest that Q_B is extensively re-oxidised during the fluorescence induction. However, the low corresponding values of qQ in the infected and adjacent uninoculated leaf tissue indicate that Q_B is not re-oxidised to the same extent and that this effect is exacerbated as infection progressed; cv. Lustre was more affected than wild oat.

The greater reduction of the Q_B sites of photosynthetic electron transport chain in the infected and adjacent uninoculated leaf tissue could be interpreted in two ways. Firstly, if the density of functional Q_B sites is lower in the infected and adjacent uninoculated tissue than in the controls, PSII unit connectivity would ensure a more reduced state of this acceptor. Secondly, if the rate of oxidation of the Q_B pool is impaired in infected and adjacent uninoculated tissue, but the rates of reduction are similar to that of the controls, the QB pool would be more reduced (Dominy, P.J.) (personal communication).





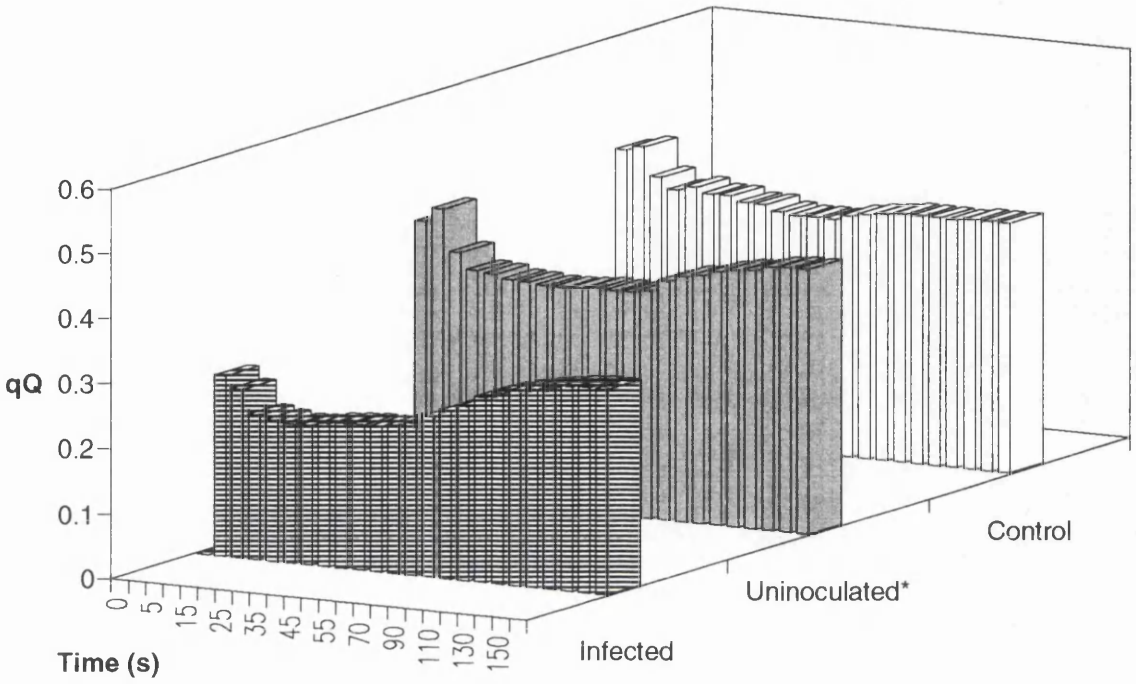
* adjacent uninoculated tissue of infected leaf

D

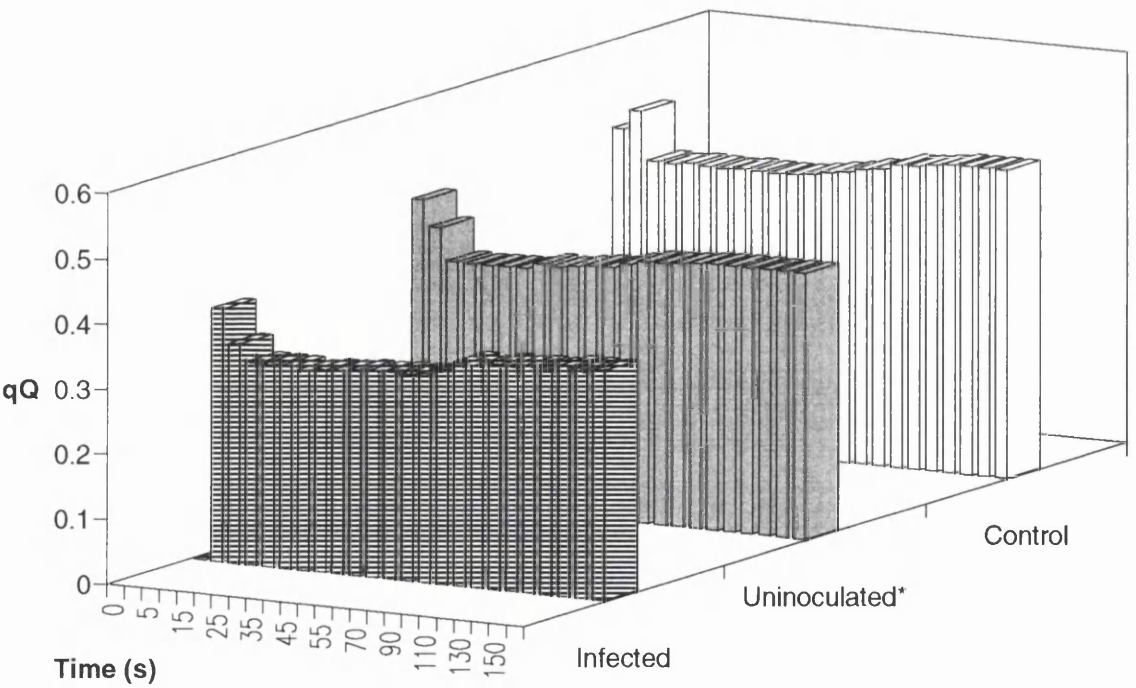
15 Days : Lustre

LSD.05 | 60s

LSD.05 | 160s



20 Days : Lustre



* adjacent uninoculated tissue of infected leaf

CHAPTER 6

DISCUSSION

Parasitic infections, particularly heavy infections, are bound to affect the growth and development of their host to some extent. Some of the effects are likely to be due to parasite activities which are essential if the parasite is to infect and complete its life cycle successfully, and such effects can be termed unavoidable disease (Clarke, 1986). Even the most tolerant host genotypes would be expected to suffer this level of disease. Levels of disease over and above those which are an inevitable consequence of infection have been termed avoidable and considered to reflect intolerance (Clarke, 1986). However, avoidable damage is likely to have been reduced to a minimum, or even eliminated altogether, during the course of evolution in wild plant pathosystems because natural selection should favour host genotypes which suffer the least disease for given levels of infection (Clarke, 1986).

A plant's overall tolerance of a parasitic infection will be determined partly by its tolerance of the parasite and partly by its tolerance of any disease the parasite may cause. Overall tolerance can be measured by relating the accumulation of parasite biomass to any reduction in the overall performance of the plant in terms of growth or yield. The overall tolerance of a plant should also be determined by the individual tolerance of its different organs, tissues and metabolic systems to the parasite or any disease it may cause (Clarke, 1988).

The interaction between wild oat, Avena fatua, and Erysiphe graminis f.sp. avenae is likely to be the product of a long period of co-evolution and therefore avoidable damage should have been eliminated through natural selection. Thus any effects of infection on the growth of A. fatua will reflect the unavoidable damage caused by that level of infection. However, in cultivated oats, Avena sativa, selection for important economic traits through oat breeding programmes may have inadvertently resulted in host genotypes in which the genetic systems controlling tolerance have been disrupted so that some degree of original tolerance may have been lost. In other words, avoidable damage may occur. A comparative study of the effects of mildew infection on wild and cultivated oats could be useful because it could indicate the nature and

extent of the unavoidable damage which are likely to result from given levels of infection and the extent of the avoidable damage which may occur in some cultivated oats. These studies are important, not only because they could indicate the likely importance of tolerance in the survival strategy of wild plants, which enable them to survive despite parasite attack, but also because they could indicate the potential value of tolerance as a component of a protection strategy for a crop in the absence of adequate sources of resistance (Harry and Clarke, 1992).

In the present work, as well as overall tolerance, tolerances of different tissues and metabolic systems of wild and cultivated oats were examined, i.e., leaf growth and development, photosynthesis and dry matter accumulation, the partitioning of photosynthates to different parts of the plant and stomatal function.

Since this work involves a comparative study of the degree of tolerance in wild and two cultivated oats, a preliminary measurement of fungal growth was carried out to determine any differences in the ways mildew infection developed and the amounts of that infection which developed on each line. Under controlled conditions, the line of wild oat supported more fungal growth at all stages than the cultivated oats did. Measurements of fungal development as percentage leaf area covered showed that within 1 week of exposure to natural infection, pustules of Erysiphe graminis appeared on the lower leaves of plants of each line and subsequently the infection spread rapidly. The level of infection increased so that within 4 to 5 weeks, mildew colonies covered a greater percentage of leaf area in wild oat (about 30 %) than in cvs Lustre (about 25 %) and Peniarth (about 20 %). As infection progressed, the levels of infection decreased due to a greater loss of the older leaves through enhanced senescence and to the upper younger leaves which became only lightly infected, or not at all, especially in cvs Lustre and Peniarth. Thus the three lines showed adult plant resistance but this was expressed at a later stage in wild oat.

Measurements of fungal development as number of spores also showed that spore production on individual leaves on the main axis and in total per plant of wild oat was higher than that on cv. Lustre and higher still than that on cv. Peniarth. Spore

production on the upper leaves was found to decrease on each line as infection progressed but more so on the two cultivars than on wild oat.

Mildew infection altered growth and development in essentially the same way in the three lines although the magnitude of the effects were not the same in each line. Infection substantially reduced dry matter production in each line and this was expressed in reductions in shoot and root development. The reduction in shoot development was reflected in a shortening of the main axis, fewer tillers, reductions in leaf area and green leaf area, reductions in yield components and in final grain yield. The reductions in total plant growth were greater in the two cultivated oats than in wild oat although wild oat at all stages of infection supported the greater levels of mildew development.

Infection reduced the relative growth rates of all three lines and thus total dry matter production. The relative growth rates were reduced by about 28 % in cv. Lustre, about 26 % in cv. Peniarth but only about 21 % in wild oat 7 weeks after inoculation. The reductions in dry matter production first became significant at about 20 % mildew cover in cv. Peniarth, about 23 % in cv. Lustre but not until about 30 % mildew cover in wild oat. The percentage reduction in total dry weight was about 30 % in the two cultivars but only 15 % in wild oat 4 weeks after inoculation. The percentage reduction became progressively greater as infection progressed so that 7 weeks after inoculation it was about 61 % in cv. Lustre, about 55 % in cv. Peniarth but only about 45 % in wild oat.

Last (1962) found that infection of barley with E. graminis f.sp. hordei reduced total dry matter production by about 59 % when about 20 % of the foliage was affected. The relative growth rate of barley was also found to be reduced by mildew infection (Walters and Ayres, 1981). Owera *et al.* (1981) showed that infection of barley with Puccinia hordei caused a reduction of 20 % in plant dry weight 16 days after inoculation. More detailed studies on Senecio vulgaris (groundsel) infected with Erysiphe fischeri (Ben-Kalio, and Clarke, 1979; Harry, 1980) and other studies involving Elymus repens (couch grass) infected with E. graminis and Epilobium montanum (broad-leaved willow herb) infected with Sphaerotheca epilobii

(unpublished undergraduate studies) have shown that none of these infections reduced dry matter accumulation until more than 30 % of the aerial surfaces were colonized.

Mildew infection also reduced the partitioning of dry matter between the different parts of the plant in the two cultivars but not in wild oat. The proportion of dry matter going to the roots was reduced relative to that going to the shoot in cv. Peniarth, but not in cv. Lustre or wild oat. Infection also affected the balance between the assimilatory surface and the rest of the plant by increasing the dry matter content of the leaves per unit plant dry weight and this was evident in the two cultivars but not in wild oat. Changes in the relative proportions of dry matter in the different organs and tissues are a result of the effects of infection on translocation patterns within the plant. Such effects were more apparent in the two cultivars than in wild oat. Thus in cvs Lustre and Peniarth, although infection reduced the size of the leaves, the relative proportion of total dry matter in the leaf tissue was greater in the infected than in the uninfected plants. However, in wild oat, although infection reduced leaf area the reduction was proportional to the size of the plant and infection did not appear to alter the relative proportion of dry matter in the leaves. Thus infection appeared to have little or no effect on the pattern of translocation within wild oat whereas it did in the cultivated oats, especially in cv. Peniarth .

In crop plants, such as cereals, root growth has been shown to be reduced substantially more than shoot growth even by low levels of infection (Ayres, 1984). The ratio of root to shoot dry weight was found to be decreased in barley infected with mildew (Last, 1962; Fric, 1975). In addition, Owera *et al.* (1981) showed that the overall decrease in dry weight of barley plants infected with P. hordei was paralleled by an increase in weight of the infected leaves. However, studies on S. vulgaris infected with E. fischeri have shown that the distribution of the dry matter in relation to the development of various organs was not affected even at 90 % total mildew cover (Ben-Kalio and Clarke, 1979; Harry and Clarke, 1992) .

The reductions in total dry matter production in the three lines, following infection, was probably related to the reductions in green leaf blade area, in unit leaf rates and in chlorophyll levels per unit of leaf area. Part of the reductions in green leaf

blade area resulted from premature senescence of the older lower leaves. Normally, senescence commences in the older lower leaves and then progresses to the upper leaves and mildew infection which occurs first on the lower leaves appears to increase the rate at which the leaves senesce. The extent to which infection caused the leaves to senesce on the main axis or on tillers, was significantly greater in the two cultivars than in wild oat .

The production of new leaves on the main axis in both cvs Lustre and Peniarth, but not in wild oat, was slower than in the controls. In the case of cv. Lustre, the final number of leaves expanding on the main axis of infected plants was eventually the same as on the control plants but infection reduced the number of leaves expanded on the main axis in cv. Peniarth by one leaf. It is possible that, in the cases of cv. Lustre and wild oat, those leaves of the full complement which had not unfolded at the time of inoculation were already formed in the apical bud and their development was not totally inhibited by infection. In the case of cv. Peniarth, which developed one less leaf in response to heavy infection, either the last leaf primordium was never formed or it aborted, or it might have been transformed to a reproductive structure. However the growing points were not dissected to investigate these possibilities .

Infection also reduced the size of individual leaves in both wild oat and cv. Peniarth. The cv. Lustre was not included in this experiment. The reductions in individual leaf blade area were found to be due to reductions in both cell division and cell expansion. The effects of infection on cell division and cell expansion were deduced from counts of stomata and epidermal cells per unit area of the lower epidermis because, although infection increased the number of stomata per unit area, it did not change the ratio between the numbers of stomata and epidermal cells. Thus the increase in number of stomata per unit area was due to reduced expansion of the epidermal cells. The reduction in epidermal cell expansion in the infected leaves is probably not a specific response to infection because similar reductions have been reported to occur in response to other stress factors (Wilkinson, 1979). However, reductions in the expansion of the epidermal cells did not account for all the reduction in size of the infected leaf, and so there must have been fewer epidermal cells in the

leaves of the two lines. The reductions in leaf area were also due to reduced cell division and it was calculated that there were about 13 % fewer epidermal cells in wild oat but about 17 % fewer epidermal cells in cv. Peniarth. Clearly the reductions in green leaf blade area of infected plants found in this study were due to increased losses through senescence as well as reductions in the growth of the individual leaves through reduced cell division and cell expansion .

Reductions in leaf growth and development have been reported for many crops plants infected with various pathogens, including viruses, bacteria and fungi. Last (1962) reported that barley plants infected with E. graminis f.sp. hordei , besides producing fewer tillers than uninfected plants, also produced smaller leaves. Furthermore, leaves on infected plants senesced earlier than leaves on uninfected plants. Macfarlane and Last (1959) showed that cabbage plants infected with Plasmodiophora brassicae had fewer, smaller and thinner leaves, and the leaves expanded more slowly than the corresponding leaves on uninfected plants. They suggested that the reduction in leaf size was probably due to the effects of infection on cell division in the leaf primordium caused by the drain of materials to the gall, in addition to effects on cell expansion (Macfarlane and Last, 1959). Recent studies on the effects of E. fischeri on the growth of S. vulgaris (Harry and Clarke, 1992) similarly showed that green leaf tissues were reduced partly through rapid loss of the older leaves due to senescence and partly to the reduction in leaf expansion. These smaller leaves contained fewer and smaller cells than the corresponding leaves on control plants, indicating that, as in this study, mildew infection inhibited both cell division and cell expansion in the developing leaf .

Both cell division and cell expansion are physiological processes which are very sensitive to the changes in leaf water potential (Ayres, 1981). Such changes have been observed to be brought about by infection (Ayres, 1972). Although the water relations of the infected leaves were not investigated in this study, the mildew mycelium alone over the surfaces of oat leaves is likely to increase the rate of water loss not only by increasing the surface area from which water loss can occur, but through effects on stomatal function. In fact, the study of stomatal function did show that it was

impaired by infection, stomatal resistance of infected leaves was reduced in the dark and became more reduced as infection progressed. Thus leaf water potential would be expected to decrease as a result of increased stomatal transpiration from infected tissues. However, all the plants were well watered and so some of the increased loss should have been compensated for by increased uptake unless root function was impaired. Walters and Ayres (1981, 1982) showed that in barley infected with mildew, not only was the total activity (^{14}C) in the roots reduced but that the length and branching of main seminal and nodal root axes of the plant were also reduced. It is also possible that infection altered the water relations in the infected leaves by effects on cell membranes reducing the turgor relations of the epidermal cells (Ayres, 1972) .

In addition to the effects on leaf growth, the mildew colonies probably physically obstructed the plant's photosynthetic surface area and in this way limited the plant's capability of dry matter production. In this study, the operation of the photosynthetic system was altered by infection in all three lines, evidenced by reductions in unit leaf rates and chlorophyll levels per unit leaf area. The unit leaf rates followed similar ontogenetic progressions in infected and uninfected plants although the levels were always lower in the infected plants throughout the course of infection. The differences in unit leaf rates between infected and uninfected plants were more apparent in the two cultivars than in wild oat .

In barley plants infected with E. graminis f.sp. hordei, reductions in dry matter production, which occurred when plants had only about 20 % mildew cover, were partly attributed to a reduction in the assimilatory surface and partly to a reduction in the unit leaf rate (Last, 1962). However, Harry (1980) showed that unit leaf rates in S. vulgaris infected with E. fischeri were not reduced until more than 90 % of the plant was infected. This is indicative of high levels of tolerance in groundsel to mildew infection .

The rates of photosynthesis in the infected third leaf were found to be more reduced by low levels of infection in the cultivated oats than by higher levels of infection achieved in wild oat. Thus, the results indicate clearly that although mildew development per unit area of the leaf, measured as spore production or accumulation of

chitin, was higher in wild oat than in cvs Lustre and Peniarth, the maximum rate of photosynthesis in that area of the leaf declined more slowly than in the two cultivars. For example, 15 days after inoculation, the maximum rate of gross photosynthesis per unit area of the infected leaf was reduced by about 36 % in cv. Peniarth, about 27 % in cv. Lustre but about 26 % in wild oat. However, the mildew development on the leaves showed an inverse relationship to the suppression of photosynthesis since about 3131 spores per mm^2 leaf area were present on cv. Peniarth, about 3388 spores per mm^2 leaf area were present on cv. Lustre but about 3568 spores per mm^2 leaf area were present on wild oat, 15 days after inoculation. No measurements were possible on leaves of cv. Peniarth from 15 days because the infected leaf by this stage had senesced so far, but measurements on cv. Lustre and wild oat were still possible and by 20 days after inoculation, maximum gross photosynthesis in the infected leaf was reduced by about 32 % with no further spore production on cv. Lustre whereas in wild oat there was no further reduction in photosynthesis although the number of spores produced had continued to increase to about 3684 per mm^2 leaf area .

The rates of photosynthesis per unit area of the infected tissue and the adjacent uninoculated tissue, when the tip and base regions of the fourth leaf were inoculated, were also found to be more reduced in cv. Lustre than in wild oat. cv. Peniarth was not included in this experiment. The maximum rate of photosynthesis in the adjacent uninoculated tissue slightly increased during the first 5 days after inoculation, but thereafter it fell below that of the control in both cv. Lustre and wild oat. The mechanism by which the increase was brought about is obscure. Infection decreased the maximum rate of gross photosynthesis per unit area of the infected tissue, 20 days after inoculation, by about 38 % in cv. Lustre but only 26 % in wild oat whereas that of the adjacent uninoculated tissue was reduced by about 29 % in cv. Lustre and about 23 % in wild oat .

Reports exist of stimulation of photosynthesis in the healthy leaves of infected plants. Wang (1960) reported higher CO_2 uptake by healthy regions of inoculated leaves than by infected areas in wheat leaves infected with Puccinia graminis f.sp. tritici .

Stimulation of photosynthesis in uninfected leaves might allow the plant to compensate for the loss of assimilates due to the fungus (Walters, 1985). Ayres (1979) suggested that stimulation of photosynthesis in uninfected tissues during the initial stage of infection is probably linked with changes in photorespiration .

The photochemical efficiency of photosynthesis at low light intensity was also found to be more reduced in the infected tissues of the two cultivars than in wild oat, the percentage reduction being about 27 % in cv. Peniarth but only about 18 % in cv. Lustre and wild oat at 15 days after inoculation, with further reductions to 26 % in cv. Lustre and to 19 % in wild oat by 20 days after inoculation. Similarly, the photochemical efficiency of photosynthesis in low light was more reduced 20 days after inoculation in the infected tissue (about 29 %) and adjacent uninoculated tissue (about 16 %) in cv. Lustre than in wild oat where the respective values were only about 17 % and 2 % .

Infection reduced total chlorophyll content per unit area of the infected tissues in cvs Lustre and Peniarth, and also, but to a lesser extent, in wild oat. The percentage reductions in total chlorophyll contents, at 15 days after inoculation, were about 36 % in cv. Lustre, about 33 % in cv. Peniarth but about 25 % in wild oat, with no further reductions in cv. Lustre and wild oat at day 20. Total chlorophyll contents of the infected tissue and the adjacent uninoculated tissue were also more reduced in cv. Lustre than in wild oat. The percentage reductions in the infected tissue were about 36 % in cv. Lustre and about 31 % in wild oat whereas the reductions in the adjacent uninoculated tissue were only about 25 % in cv. Lustre and about 22 % in wild oat by 20 days after inoculation .

Reductions in the rate of photosynthesis following fungal infection have been shown by several authors. Sugar beet leaves infected with E. polygoni showed reductions in photosynthesis by about 35, 70 and 75 % at 9, 16 and 22 days after inoculation respectively. The photosynthetic efficiency of photosynthesis at low light was also reduced by about 17 % and 22 % at 14 and 18 days after inoculation respectively (Gordon and Duniway, 1982a). Scholes and Farrar (1985) showed a reduction in photosynthesis per unit area and per unit chlorophyll within pustule areas

of Uromyces muscari infected bluebell (Hyacinthoides non-scripta) leaves .

Reductions in photosynthesis have been attributed to altered rates of CO_2 diffusion into the leaf through stomata (Gordon and Duniway, 1982b). Photosynthesis is in part a diffusion process where the flux of CO_2 must first diffuse through the leaf boundary layer, into the leaf through the stomata and into the mesophyll layer before being fixed at the carboxylation sites in the chloroplast. Measurements of stomatal function showed that it was altered by infection in all three lines. The diffusive resistances in the light in both the middle and tip regions of infected leaves were first low when compared with those in control leaves, particularly in cvs Lustre and Peniarth, but then they increased in all three lines as infection progressed. In contrast, the diffusive resistances in the dark in both the middle and tip regions of infected leaves were lower than those in control leaves, especially in cv. Peniarth. The increase in diffusive resistances in the light occured from around 5 days after inoculation in all three lines, a period which correlated with maximum fungal growth as assessed by fungus sporulation and chitin analysis. This increased diffusive resistance of the infected leaves in the light is likely to reduce the diffusion of CO_2 to the mesophyll cells and could be then partly responsible for the reduction in the rate of photosynthesis that occurred. Furthermore, the increased diffusive resistances in the light could also be expected to contribute to water loss from infected leaves. Thus the changes in the transpiration rate in the light in infected leaves would be expected to be the result of not only an increase in stomatal resistance to the diffusion of water vapour but also of an increase in boundary layer resistance caused by the presence of the fungus mycelium over the leaf surface. During the dark period, a decrease in diffusive resistances, almost certainly due to greater than normal stomatal opening, was recorded 48 h after inoculation in cv. Peniarth when mycelium development was limited (< 1 %), indicating that the stomata were unable to control water loss from an early stage of infection. These reductions were not apparent in either cv. Lustre or wild oat until 4 days after inoculation when percentage mildew cover was between 1 to 2 % .

The increased stomatal opening in the light that occurred in the first 3 days after inoculation in oat leaves was similar to that in pea leaves infected with E. pisi,

although the increase occurred 48 h after inoculation in pea (Ayres, 1976). However, the reduction in stomatal opening in the light was shown to occur 3 days after inoculation in pea leaves infected with E. pisi (Ayres, 1976), 24 h after inoculation in barley infected with E. graminis f.sp. hordei (Majernik, 1965) but only 6 h after inoculation in wheat leaves infected with E. graminis f.sp. tritici (Martin *et al.*, 1975). Martin *et al.* (1975) have suggested that stomatal closure is induced by volatile or diffusible substances produced by the fungus. If this is correct, it seems that either E. graminis f.sp. avenae does not produce these substances or that it takes longer for a critical concentration to be reached in oat than in pea or barley. The difference in the effects of the mildew fungus on stomatal function between these three species could also be due to differences in the levels of infection in each species and/or to different mechanical interactions (turgor pressure) between guard and epidermal cells as the fungi begin their invasion (Ayres, 1976).

Changes in the transpiration rate of mildewed peas (Ayres, 1976), barley (Majernik, 1965) and wheat (Martin et al, 1975) followed closely changes in stomatal opening in the light and dark period. The difference between pea and the cereals in the timing of these changes probably results from the different times at which changes in stomatal behaviour begin following infection (Ayres, 1976).

In the leaf oxygen electrode experiments, photosynthesis was measured under conditions of saturating CO_2 (Delieu and Walker, 1981) and so any change in rates of photosynthesis was not due to a reduced supply of CO_2 but could probably be attributed to changes within chloroplasts. In this study, two models describing the photosynthetic light-response curve have been used. The model derived by Marshall and Biscoe (1980) in which the resistance to CO_2 diffusion to the carboxylation sites, expressed as the ratio of physical to total resistance ($\theta = r_p/r_p+r_x$), is greater than zero, and the model of Rabinowitch (1951) in which the resistance to CO_2 diffusion to the carboxylation sites, θ, is equal to zero. The results obtained in this study, using the model of Marshall and Biscoe, showed that the reduction in photosynthetic efficiency of the infected tissue was unlikely to be due to changes in the amounts of CO_2 diffusing into the leaf to the carboxylation sites in either cv. Lustre or wild oat because the values

of θ in the infected tissue were not significantly different from those in the controls at any stage of infection. However, in cv. Peniarth, the differences in θ values between the infected and uninfected tissue became significant from around 12 days after inoculation and onwards. The changes in θ values in the infected tissue of cv. Peniarth suggest that the physical or carboxylation, or both, resistances to CO_2 diffusion into the leaf were altered by infection but at a later stage .

Reductions in photosynthesis may also be due to direct effects of the pathogen at the biochemical or chloroplast level. Alterations in the photosynthetic apparatus could be attributed to changes within the chloroplast which affect: (a) the supply of CO_2 to the carboxylation sites in the chloroplast; (b) the light-harvesting capacity; (c) photosynthetic electron transport which provides reducing power NADPH and/or ATP from the light reaction centres, PSI or PSII; (d) the turnover rate of enzymes of the C-3 cycle (carboxylation efficiency) .

This study showed that the reductions in the maximum rate of photosynthesis in the infected and adjacent uninoculated tissue could have been due to an impairment of photosynthetic electron transport which reduced the supply of reducing power (NADPH) and/or ATP. Secondly, the turnover of enzymes of the C-3 cycle could have been impaired and this would result in low carboxylation efficiency. In cv. Peniarth, the reduction in the rate of photosynthesis of the infected leaf could also be due to reduced supply of CO_2 to the carboxylation sites within the chloroplast, evidenced by the changes in θ values at later stages of infection .

The photochemical efficiency of photosynthesis at low light intensity is thought to be a sensitive indicator of damage to the electron transport chain (Sharkey, 1985). At low light intensity, the number of quanta of light reaching the reaction centres determines the rate of electron transport, and this is dependent upon the amount of light-harvesting chlorophyll. In the present investigation, the reduction in the photochemical efficiency of photosynthesis at low light in the infected and adjacent uninoculated tissue could be due to a low density of the photosynthetic unit and therefore a reduction in the amount of light-harvesting chlorophyll (from the light-harvesting complex or a specific loss of chlorophyll a from the reaction centres of PSI

or PSII) and this would result in reduced light-harvesting capacity. Loss of chlorophyll was confirmed in chlorophyll content measurements. However, inhibition of photosynthesis did not appear to be due to reductions in the effectiveness of the light-harvesting chlorophyll since the maximum rate of photosynthesis per milligram of chlorophyll in the infected or adjacent uninoculated leaf tissue was not affected either at low or high levels of infection in the three lines .

Scholes and Farrar (1985) showed that in bluebell leaves infected with Uromyces muscari, the rate of photosynthesis was reduced, whether measured on either a unit area or a unit chlorophyll basis. Contrarily, the rate of photosynthesis per unit chlorophyll was found to increase in barley infected with brown rust Puccinia hordei (Scholes and Farrar, 1986) .

The chlorophyll fluorescence studies also indicated alterations to the photosynthetic apparatus and that these alterations were more pronounced in the infected and adjacent uninoculated tissue in cv. Lustre than in wild oat .

Measurements of chlorophyll fluorescence provide more evidence on the probable mechanisms by which photosynthesis was impaired in infected and adjacent uninoculated tissues. The results indicated that infection results in a decrease in the rate of photosynthetic electron transport after the PSII Q_B binding site (Q_B is more reduced). These findings can be interpreted in two ways. If photosynthetic electron transport was directly impaired by infection, then the proton motive force (pmf) across the thylakoid membrane would be expected to be low. Alternatively, if carboxylation processes downstream of the photosynthetic electron transport were impaired, low ATP demand, and possibly cyclic electron transport, would establish a high proton motive force. The experiments on non-photochemical qE quenching in infected and adjacent uninoculated tissue clearly show that infection causes a decrease in qE quenching and this decrease has been interpreted to result from a decrease in the thylakoid proton motive force (Horton, 1983). This suggests that photosynthetic electron transport is affected directly. Although the photochemical efficiency of photosynthesis at low light intensity and the light harvesting capacity has been decreased due to a loss of chlorophyll, these are not limiting factors as photochemical qQ quenching showed that

Q_B was more reduced in the infected and adjacent uninoculated tissue in cv. Lustre than in wild oat .

It has also been pointed out that qQ quenching is not only a measure of the redox state of the Q pool but is also a measure of the efficiency of photosynthetic electron transport at any point of *in vivo* fluorescence induction curve (Genty *et al.*, 1989). Therefore, in the present study, the implication is that infection inhibited the rate of photosynthetic electron transport as shown by qQ and also qE quenching .

The exact site of infection-induced inhibition of photosynthetic electron transport is uncertain but inhibition may be due to photoinhibition of the Q_B binding-protein (Kyle, 1985) or impairment of any subsequent component of the photosynthetic electron transport system including PSI .

Such alterations of the photosynthetic apparatus were greater in the infected tissue in the cultivated oats than in wild oat and also greater in the adjacent uninoculated tissue in cv. Lustre than in wild oat. Furthermore, the rate of photosynthesis of the uninoculated tissue in infected leaves showed an initial increase, before it dropped below that of the control tissues, suggesting that the photosynthetic efficiency in those tissues was not affected to the same extent as in infected tissues. This was apparent in cv. Lustre but particularly so in wild oat .

Montalbini and Buchanan (1974) showed reductions in the rate of electron transport and in the accompanying ATP formation in non-cyclic phosphorylation of chloroplasts of Vicia faba infected with Uromyces fabae . Buchanan *et al.* (1981) found that non-cyclic electron transport chain was inhibited by up to 45 % in chloroplasts isolated from barley leaves infected with P. hordei .

In contrast to reductions in the photosynthetic systems, the rate of dark respiration was increased in the infected tissue in the three lines and also in the adjacent uninoculated tissue in wild oat and cv. Lustre .

One of the earliest reports of respiratory changes in diseased plants is that of Allen and Goddard (1938) who showed that dark respiration was increased in wheat infected with E. graminis f.sp. tritici. The rate of dark respiration in barley leaves infected with E. graminis f.sp. hordei was found to be at least twice that of the controls

at the time of sporulation (Scholes and Farrar, 1986). These studies, as well as the present study, contrast with recent studies on oat plants infected with E. graminis f.sp. avenae where infection reduced rates of photosynthesis but had little effect on the rate of dark respiration (Haigh *et al.*, 1991) .

Bushnell and Allen (1962) suggested that the accumulation of metabolites, often in a mobile form, around lesions could be the cause of increased respiration. However, the most likely cause of the increased respiration caused by biotrophic fungi could be the enhanced activity of the oxidative pentose phosphate pathway (Daly, 1976).

This account has so far shown that the parasite alters the host metabolic systems, i.e. photosynthesis, respiration, metabolic pathways, transport systems etc, by a variety of mechanisms during the course of infection. Parasites produce, during infection, a wide range of metabolites some of which may have fairly localized effects on host tissues, while others are released into the host and act at a distance. They include recognition factors, growth regulators, inhibitors, toxins and enzymes (Clarke, 1986). For example, changes in photosynthesis occur in tissues not infected by the fungus and therefore they must be the result of a signal, e.g., a translocatable metabolite, produced by the parasite growing in the epidermal cells but acting on the mesophyll cells. However, there is no evidence to indicate the nature of the signal .

The consequence of the reductions in dry matter production were evident in reductions in yield components and final grain yield. The components of grain yield were affected in all three lines. Comparisons between the two cultivars and wild oat showed reductions in the numbers of tillers but the differences between infected and uninfected plants were not significant in any of the three lines. However, infection significantly reduced the number of fertile tillers so that by the end of the experiment, infected plants had about 2.8 fertile tillers compared with 3.6 in uninfected plants in cv. Peniarth, about 2.5 compared with 3.1 in cv. Lustre and about 3.0 compared with 3.7 in wild oat. The numbers of grains per main axis panicle and per plant were also reduced in all three lines. Infection reduced the number of grains per main axis panicle by about 24 % in cv. Lustre but only about 19 % in cv. Peniarth and wild oat whereas the

number of grain per plant was reduced by about 47 % in cv. Peniarth and about 43 % in cv. Lustre and wild oat. Hundred grain weight per plant was also found to be reduced by about 19 % in the two cultivars but only about 11 % in wild oat. Similarly, reductions in total grain yield were greater in the two cultivars than in wild oat with percentage reductions being about 57 % in cv. Peniarth, about 55 % in cv. Lustre but about 48 % in wild oat. However, although infection reduced the assimilatory surface and total plant dry weight, the proportion of total biomass converted to grain, i.e., the harvest index, was not significantly reduced in any of the three lines .

Fric (1975) showed that mildew infection of barley reduced total grain yield per plant by about 32 %. Inoculation of the plants at an early stage was found to result in reductions in the number of fertile tillers, the number of grains per ear and thousand grain weight (Griffiths and Scott, 1980). Roderick and Jones (1988) found that in eight cultivars of oat infected with E. graminis f.sp. avenae, losses in grain yield were accounted for by reductions in numbers of fertile tillers and thousand grain weights. Infection also significantly reduced the harvest index. Carver and Griffiths (1981) showed that in mildew infected barley, the number of fertile tillers and grain yield were reduced. The reduction in grain yield was a consequence of the reduction in green leaf area pre-anthesis .

In conclusion, this study has shown that the facets of growth investigated, for example leaf growth and development, dry matter production, stomatal function, photosynthesis, the partitioning of photosynthates to different parts of the plant and the final grain yield were less affected in wild oat by given levels of infection than in the cultivated oats. The changes brought about in almost all growth parameters measured in wild oat probably reflect unavoidable damage and the greater effects of similar levels of infection on the cultivated oats probably reflect the extent to which they suffer avoidable damage in addition to unavoidable damage .

The wild oat line used in this investigation appears to be more tolerant of mildew infection than the two cultivated oats. The tolerance of infection of wild oat might be due to the way the mildew fungus grows and reproduces on the tissues of this line. For example, if the changes to the photosynthetic systems are brought about by

translocatable metabolite produced by the fungus at the site of infection, then on wild oat the fungus may produce less than are produced on the cultivated oats. It is also possible that the metabolic systems and physiological processes of wild oat have the greater tolerance of any metabolite produced by the fungus than the cultivated oats .

The magnitude of the reductions in growth and development of the wild oat line used in this study were quite large. However, the levels of infection achieved under the controlled conditions used appear to be much higher over a longer period than those generally found in the field. Studies under controlled conditions may be testing the tolerance of wild oat to the limits. Clearly, the wild oat line was significantly more tolerant of infection than the cultivated oats. However, the levels were not great enough to suggest that it is likely to be useful in breeding programmes. Since tolerance appears to be reflected in the responses of a range of growth processes such as cell division and cell expansion, photosynthesis, transport systems etc, it is unlikely that its genetical control is simple and therefore it would be difficult to breed for .

Speculation about the role of tolerance in wild oat in relation to its survival strategy is not clear. Dinoor (personal communication) suggested that mildew attacks on wild grasses in arid regions may be useful to plant survival. Once the plant has developed yielding structures, the lower leaves become surplus to requirements because most of the photosynthates required during the reproductive stage of the plant come from the flag leaf and glumes. The lower leaves are mainly a site for water loss and thus their presence could lead to significant water stress so that their loss through enhanced senescence caused by mildew infection would benefit the plant. However, the flag leaf and glumes have adult plant resistance so that the parts of the plant involved directly in grain fill are protected. Clearly tolerance is not the only component of the survival strategy of wild oat. Field studies are required to assess its precise role .

APPENDICES

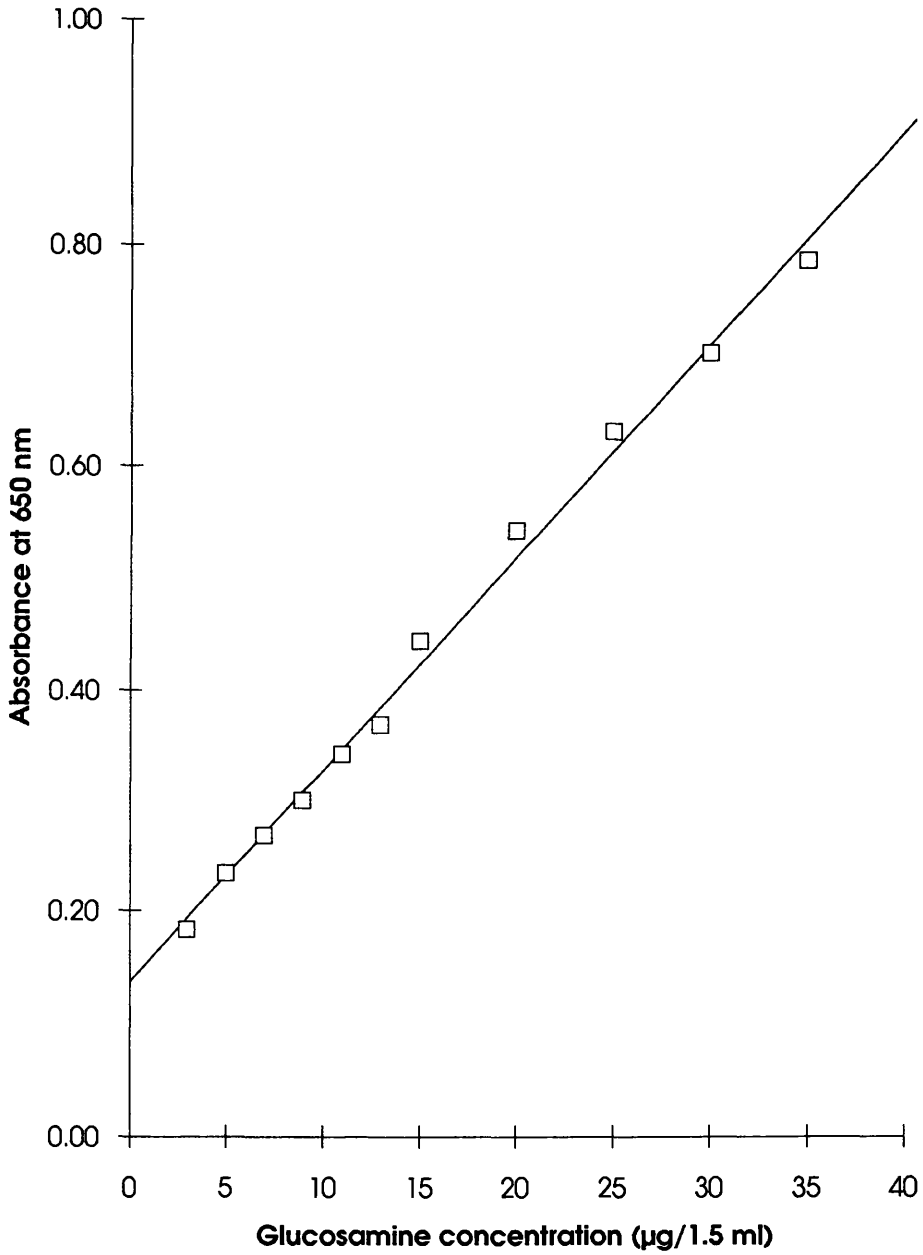


Fig. 3 : Linear relationship between glucosamine hydrochloride concentration (µg/1.5 ml) and absorbance at 650 nm.

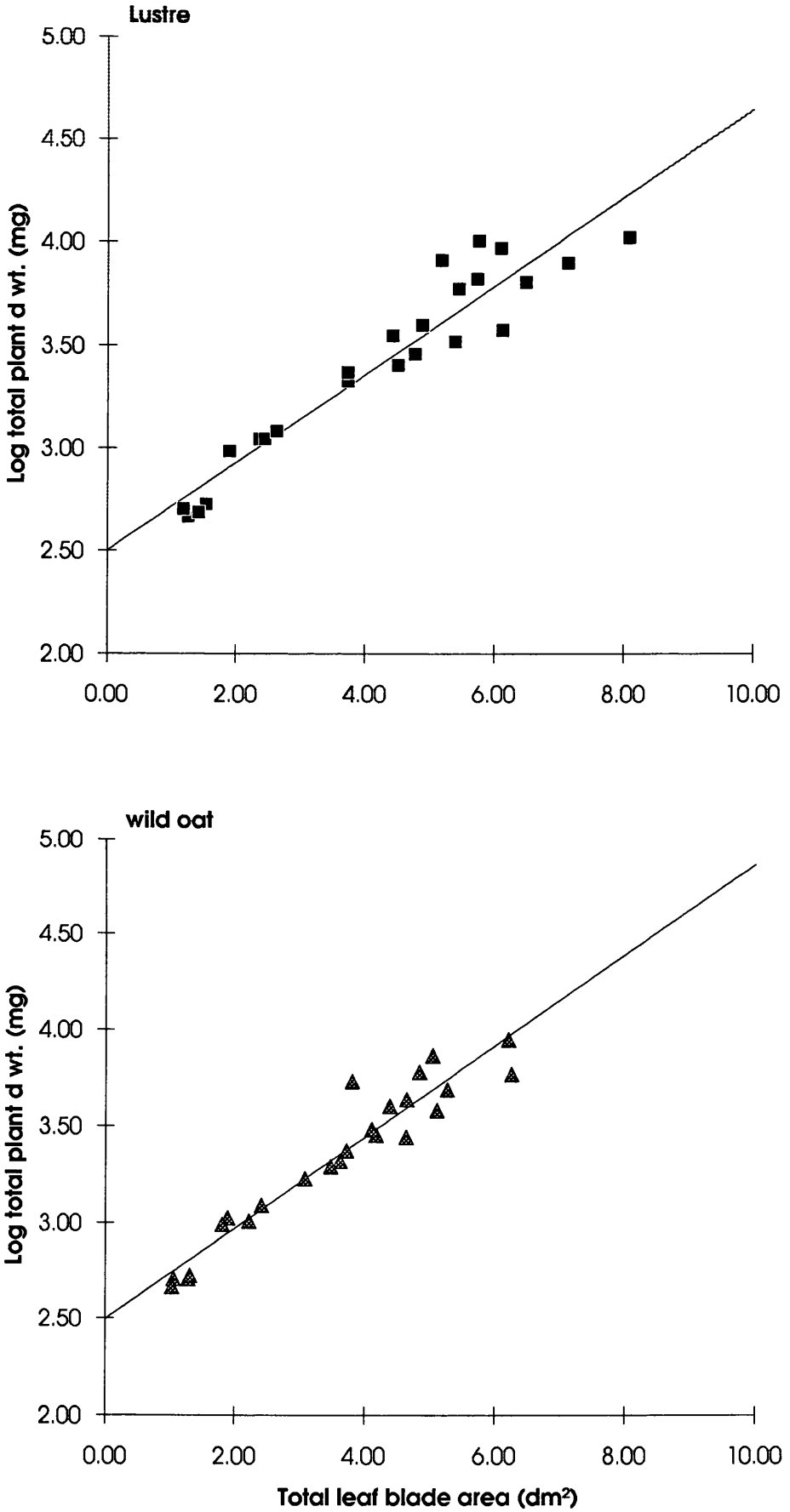
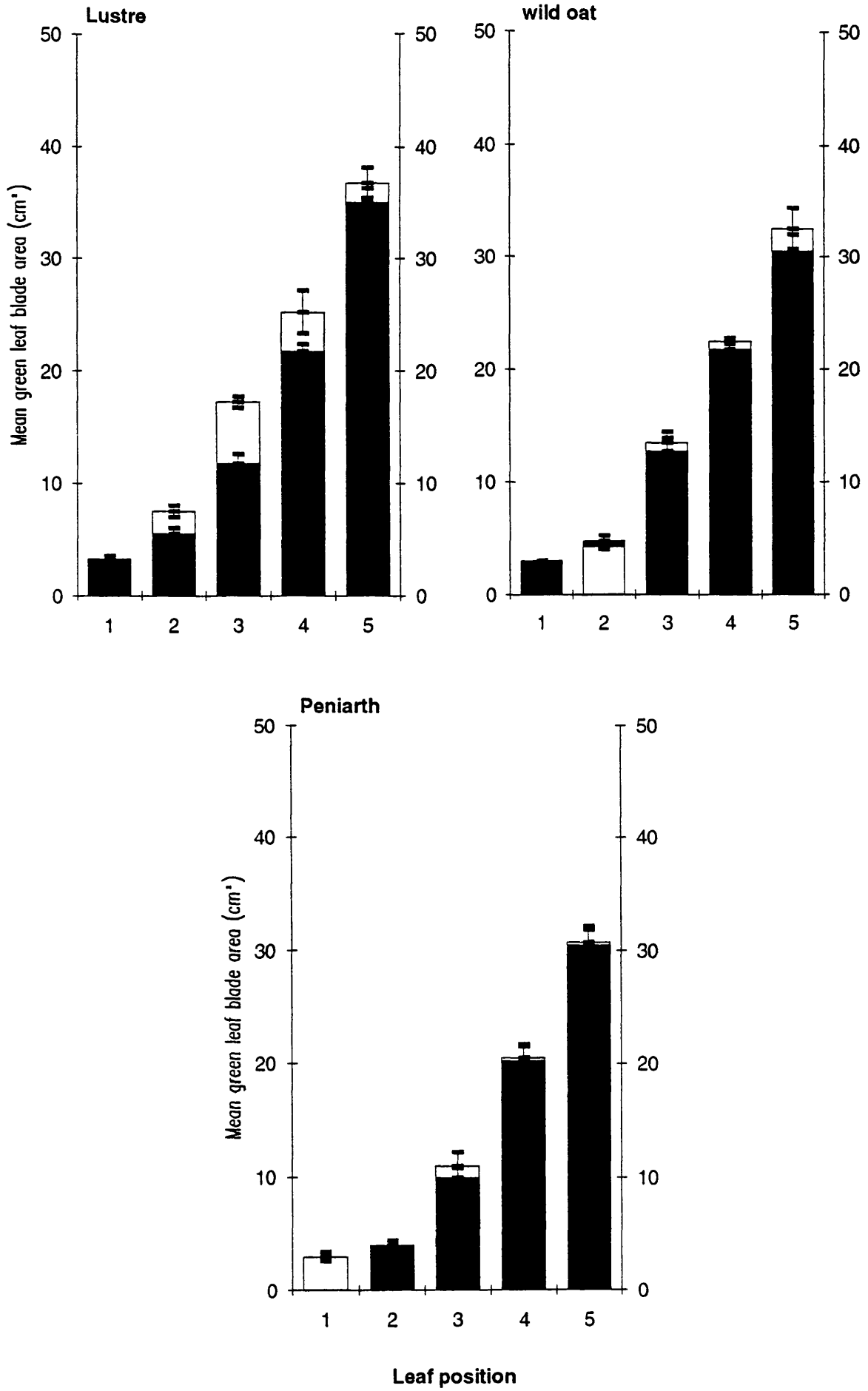
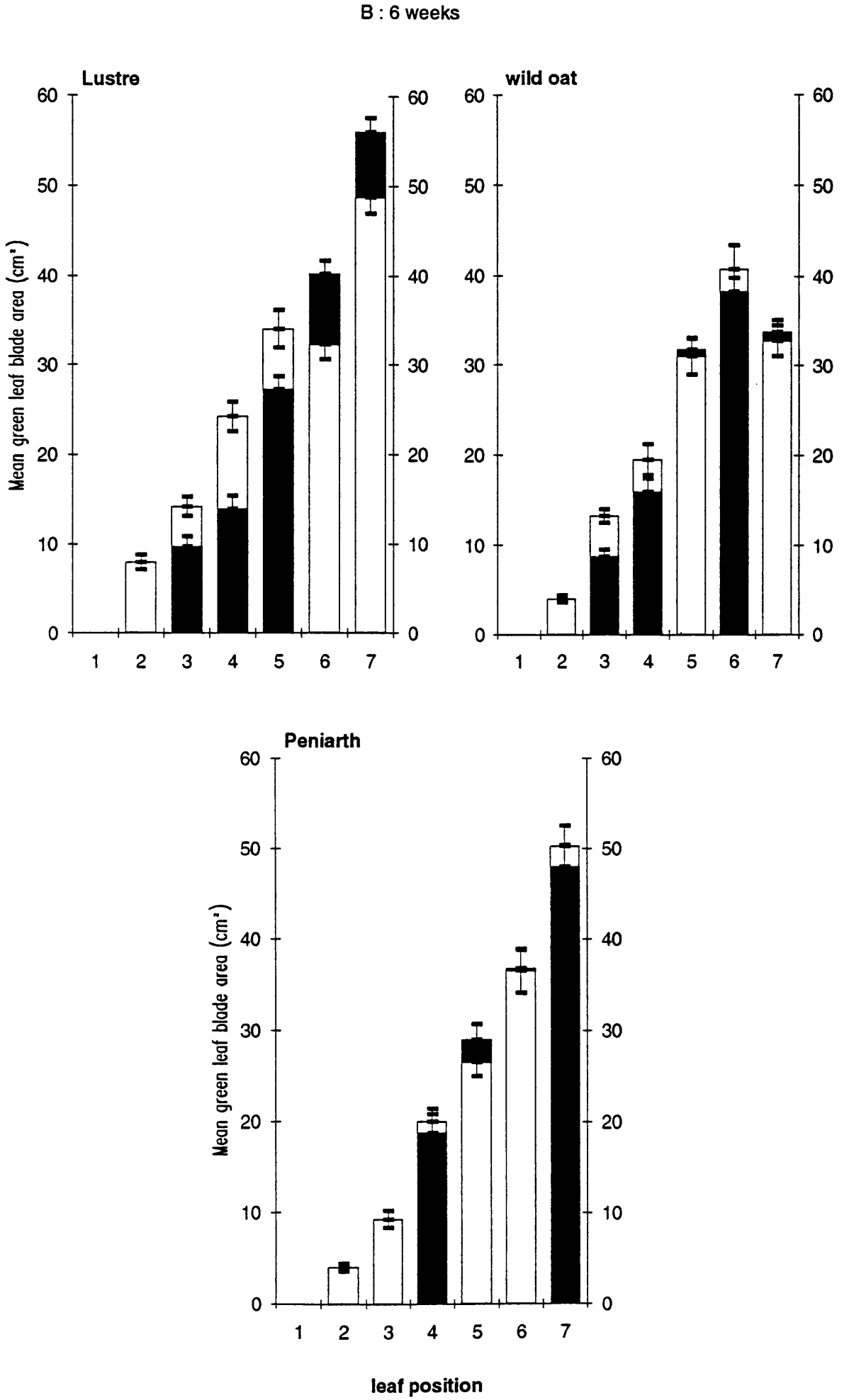


Fig. 8 : Linear relationship between total plant dry weight and total leaf blade area, in cv. Lustre and wild oat.

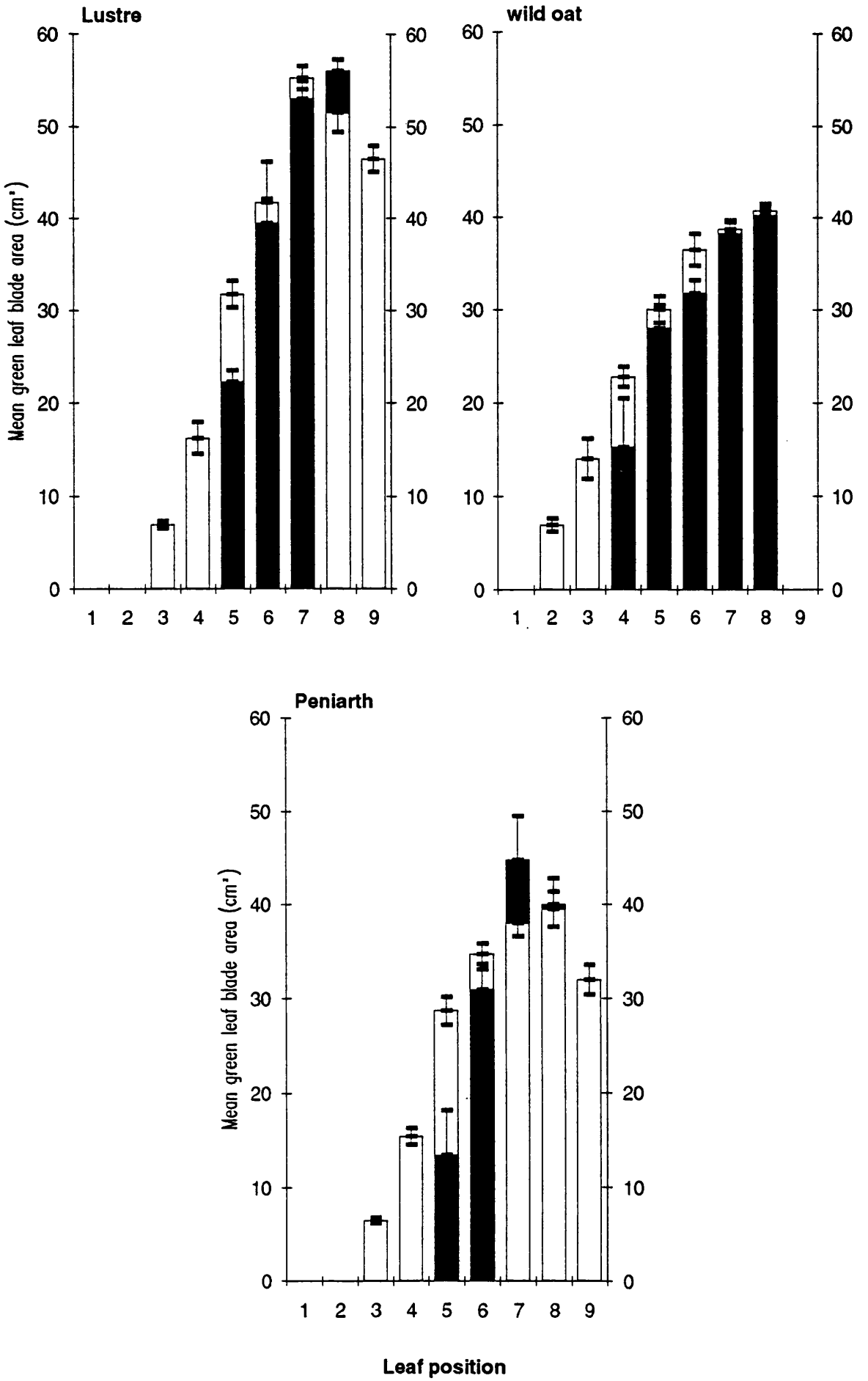
FIGURES 16A-F : Effects of infection on the number and areas of individual green leaf blades, on the main axis, of infected (■) and uninfected (□) plants of wild oat and cvs Lustre, Peniarth during the growth period between the fifth and tenth weeks after sowing.

A : 5 weeks

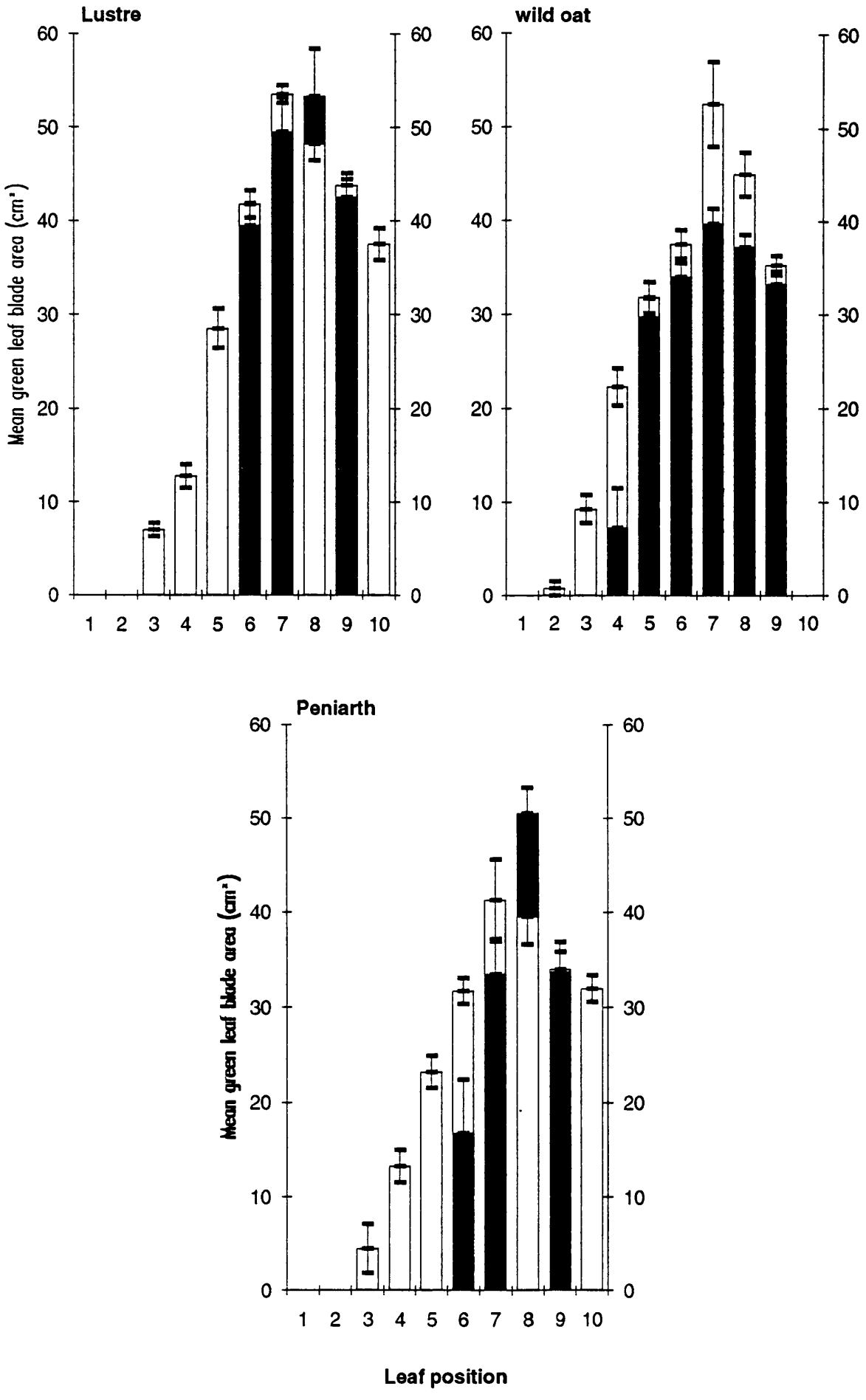

Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
0
10
20
30
40
50
1
2
3
4
5
Leaf position

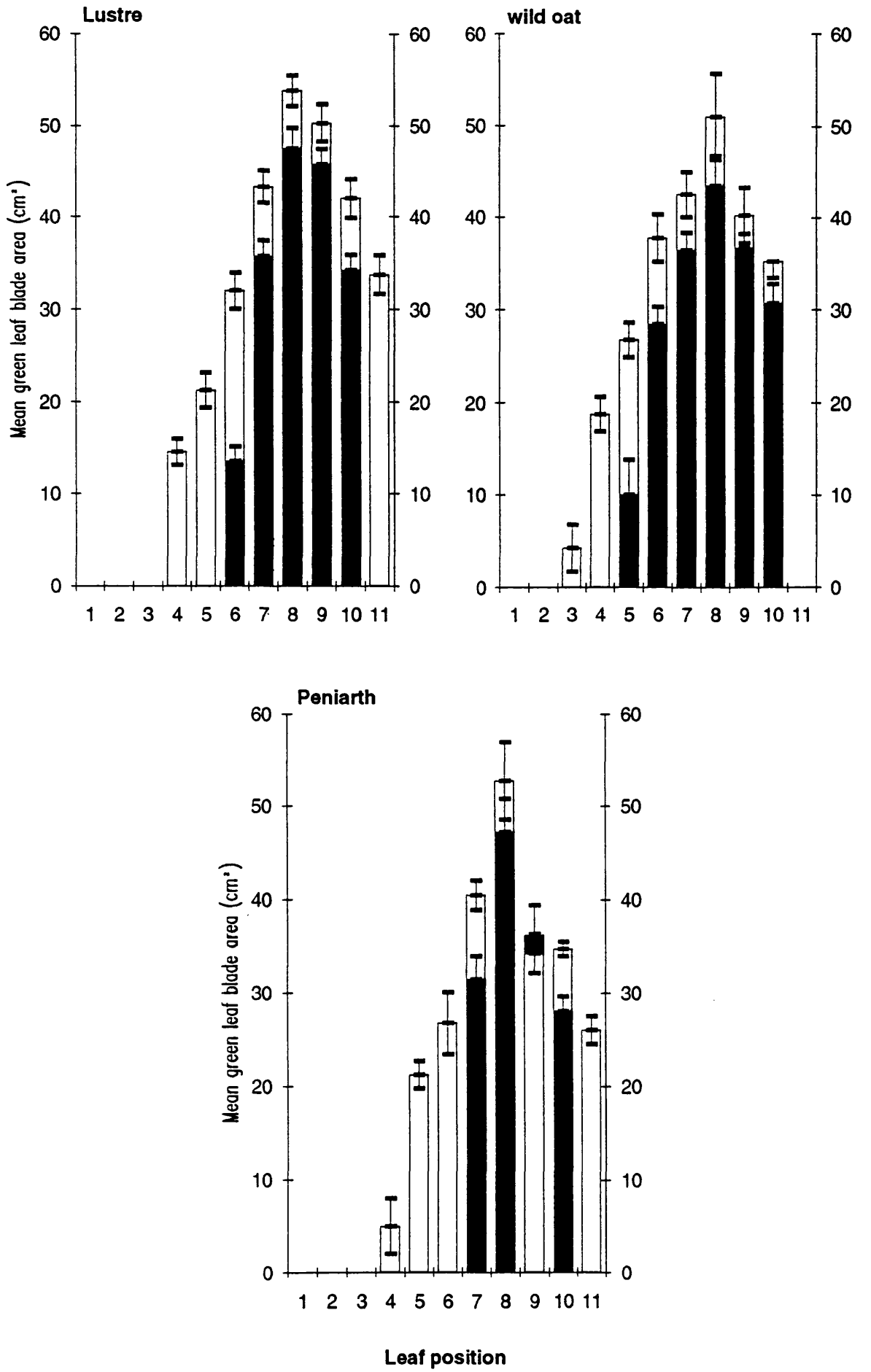

B : 6 weeks
Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
leaf position
0
10
20
30
40
50
60
1
2
3
4
5
6
7

C : 7 weeks

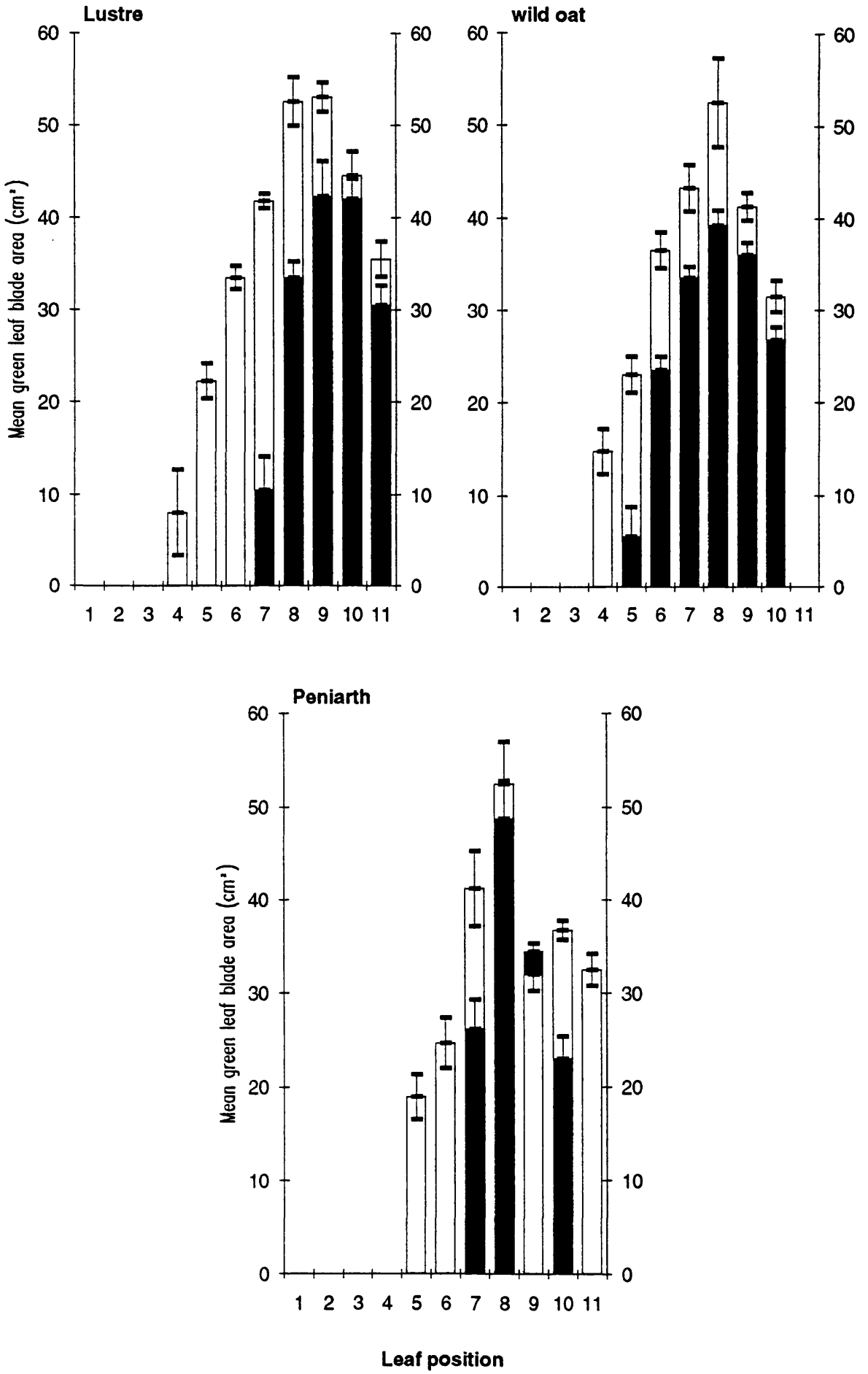

Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
Leaf position
0
10
20
30
40
50
60
1
2
3
4
5
6
7
8
9

D : 8 weeks


Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
0
10
20
30
40
50
60
1 2 3 4 5 6 7 8 9 10
Leaf position


E : 9 weeks
Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
Leaf position
0
10
20
30
40
50
60
1 2 3 4 5 6 7 8 9 10 11

F : 10 weeks


Lustre
wild oat
Peniarth
Mean green leaf blade area (cm²)
0
10
20
30
40
50
60
1 2 3 4 5 6 7 8 9 10 11
Leaf position

Appendix tables 1 to 3

- Data on some primary values of growth of Erysiphe graminis infected and uninfected plants of wild and cultivated oats taken at weekly intervals during the growth period between the fifth and tenth week after sowing.

LS Leaf sheaths + stem tissues

SLB Leaf blades of stem

LT Leaf sheaths + tillers tissues

TLB Leaf blades of tillers

Appendix table 1A. Ist Harvest (Five weeks after sowing)

'Lustre'

Rep.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	6.65	5	0	0.75	1.310	1.273	0.254	0.333	0.609	3.779	0.125	0.165	0.037	0.042	0.069	0.438
2	5.75	5	0	0.80	1.118	1.561	0.211	0.255	0.587	3.732	0.107	0.175	0.036	0.031	0.059	0.408
3	5.97	5	0	0.72	1.412	1.662	0.176	0.101	0.742	4.093	0.126	0.164	0.036	0.045	0.078	0.449
4	4.21	5	0	0.82	1.297	1.750	0.315	0.357	0.833	4.552	0.120	0.184	0.040	0.051	0.086	0.481
Mean	5.65	5	0	0.77	1.284	1.561	0.239	0.261	0.693	4.039	0.119	0.172	0.037	0.042	0.073	0.444
SE	0.45	0	0	0.02	0.053	0.090	0.026	0.050	0.050	0.164	0.004	0.004	0.001	0.004	0.005	0.013
CONTROL																
1	0	5	0	0.81	1.386	1.324	0.238	0.283	0.766	3.997	0.134	0.177	0.038	0.043	0.069	0.461
2	0	5	0	0.95	1.466	1.362	0.359	0.300	0.922	4.409	0.149	0.200	0.040	0.051	0.093	0.533
3	0	5	0	0.89	1.550	1.343	0.200	0.274	1.074	4.441	0.147	0.198	0.032	0.040	0.087	0.504
4	0	5	0	0.94	1.229	1.543	0.199	0.313	0.840	4.124	0.120	0.207	0.033	0.047	0.078	0.485
Mean	0	5	0	0.90	1.408	1.393	0.249	0.292	0.900	4.343	0.137	0.195	0.036	0.045	0.082	0.496
SE	0	0	0	0.03	0.059	0.044	0.033	0.008	0.057	0.094	0.006	0.006	0.002	0.002	0.005	0.013

Appendix table 1B. 2nd Harvest (Six weeks after sowing)

'Lustre'

Rep.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	18.27	7	2	1.40	2.012	2.703	0.946	0.885	2.398	8.944	0.178	0.314	0.106	0.162	0.225	0.985
2	16.20	7	2	1.35	2.003	2.537	1.045	0.722	2.115	8.422	0.175	0.232	0.115	0.158	0.222	0.902
3	17.00	7	2	1.66	1.802	2.916	0.859	1.014	2.113	8.704	0.172	0.437	0.097	0.175	0.207	1.088
4	18.05	7	2	1.48	2.019	2.800	1.161	0.618	1.899	8.497	0.198	0.337	0.115	0.154	0.212	1.016
Mean	17.38	7	2	1.47	1.959	2.739	1.003	0.810	2.131	8.642	0.181	0.330	0.108	0.162	0.216	0.998
SE	0.42	0	0	0.07	0.045	0.069	0.056	0.076	0.089	0.101	0.005	0.037	0.004	0.004	0.004	0.033
CONTROL																
1	0	7	1	1.63	2.270	2.909	1.378	0.831	2.182	9.570	0.213	0.344	0.135	0.192	0.225	1.109
2	0	7	1	1.75	1.968	3.065	1.320	1.047	2.649	10.049	0.181	0.374	0.126	0.186	0.242	1.109
3	0	7	1	1.41	1.916	2.715	1.189	0.522	2.543	8.885	0.180	0.325	0.079	0.142	0.239	0.965
4	0	7	1	1.67	2.059	2.932	1.629	1.188	2.504	10.312	0.191	0.349	0.192	0.239	0.238	1.209
Mean	0	7	1	1.62	2.053	2.905	1.379	0.897	2.469	9.704	0.191	0.348	0.133	0.190	0.236	1.098
SE	0	0	0	0.08	0.068	0.062	0.080	0.126	0.087	0.271	0.007	0.009	0.020	0.017	0.003	0.043

Appendix table 1B. 2nd Harvest (Six weeks after sowing)

'Lustre'

Rep.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	18.27	7	2	1.40	2.012	2.703	0.946	0.885	2.398	8.944	0.178	0.314	0.106	0.162	0.225	0.985
2	16.20	7	2	1.35	2.003	2.537	1.045	0.722	2.115	8.422	0.175	0.232	0.115	0.158	0.222	0.902
3	17.00	7	2	1.66	1.802	2.916	0.859	1.014	2.113	8.704	0.172	0.437	0.097	0.175	0.207	1.088
4	18.05	7	2	1.48	2.019	2.800	1.161	0.618	1.899	8.497	0.198	0.337	0.115	0.154	0.212	1.016
Mean	17.38	7	2	1.47	1.959	2.739	1.003	0.810	2.131	8.642	0.181	0.330	0.108	0.162	0.216	0.998
SE	0.42	0	0	0.07	0.045	0.069	0.056	0.076	0.089	0.101	0.005	0.037	0.004	0.004	0.004	0.033
CONTROL																
1	0	7	1	1.63	2.270	2.909	1.378	0.831	2.182	9.570	0.213	0.344	0.135	0.192	0.225	1.109
2	0	7	1	1.75	1.968	3.065	1.320	1.047	2.649	10.049	0.181	0.374	0.126	0.186	0.242	1.109
3	0	7	1	1.41	1.916	2.715	1.189	0.522	2.543	8.885	0.180	0.325	0.079	0.142	0.239	0.965
4	0	7	1	1.67	2.059	2.932	1.629	1.188	2.504	10.312	0.191	0.349	0.192	0.239	0.238	1.209
Mean	0	7	1	1.62	2.053	2.905	1.379	0.897	2.469	9.704	0.191	0.348	0.133	0.190	0.236	1.098
SE	0	0	0	0.08	0.068	0.062	0.080	0.126	0.087	0.271	0.007	0.009	0.020	0.017	0.003	0.043

Appendix table 1C. 3rd Harvest (Seven weeks after sowing)

'Lustre'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	23.14	8	4	1.70	2.361	3.117	3.377	1.683	4.369	14.907	0.263	0.474	0.459	0.282	0.394	1.872
2	25.88	8	4	1.72	2.703	3.194	3.200	1.576	5.763	16.436	0.278	0.488	0.369	0.345	0.451	1.931
3	19.02	8	4	1.61	2.317	2.964	3.068	0.956	4.207	13.512	0.256	0.371	0.349	0.254	0.338	1.568
4	24.43	8	4	1.86	2.840	3.525	1.771	0.895	5.016	14.047	0.294	0.594	0.256	0.236	0.400	1.780
Mean	23.12	8	4	1.72	2.555	3.200	2.854	1.277	4.839	14.725	0.273	0.482	0.358	0.279	0.396	1.788
SE	1.28	0	0	0.05	0.111	0.103	0.317	0.177	0.307	0.553	0.007	0.040	0.036	0.021	0.020	0.069
CONTROL																
1	0	9	2	2.40	3.356	4.930	3.587	0.504	4.901	17.278	0.351	0.629	0.390	0.262	0.598	2.130
2	0	9	2	2.59	3.963	5.370	3.719	2.483	4.675	20.210	0.456	0.741	0.407	0.448	0.572	2.524
3	0	9	2	2.29	4.011	4.851	2.710	3.143	5.411	20.126	0.490	0.506	0.380	0.461	0.602	2.339
4	0	9	2	2.59	5.118	5.159	4.020	3.545	6.218	24.060	0.546	0.654	0.411	0.705	0.648	2.864
Mean	0	9	2	2.47	4.112	5.077	3.509	2.419	5.301	20.418	0.461	0.632	0.397	0.469	0.605	2.564
SE	0	0	0	0.07	0.318	0.102	0.244	0.584	0.296	1.206	0.036	0.042	0.006	0.079	0.014	0.135

Appendix table 1D. 4th Harvest (Eight weeks after sowing)

'Lustre'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	23.43	9	5	2.13	3.472	3.537	1.889	2.026	6.082	17.006	0.448	0.521	0.295	0.441	0.624	2.329
2	26.14	9	5	1.65	3.008	2.272	1.740	1.448	4.460	13.428	0.373	0.481	0.287	0.244	0.483	1.868
3	24.60	9	5	1.74	2.924	2.643	1.556	1.764	3.698	12.585	0.329	0.468	0.218	0.283	0.402	1.700
4	23.05	9	5	1.87	2.912	3.081	2.301	1.847	4.177	14.318	0.257	0.501	0.359	0.387	0.448	1.952
Mean	24.30	9	5	1.85	3.079	3.008	1.871	1.771	4.604	14.334	0.352	0.493	0.290	0.339	0.489	1.962
SE	0.70	0	0	0.10	0.115	0.172	0.137	0.105	0.448	0.830	0.035	0.010	0.025	0.039	0.041	0.115
CONTROL																
1	0	10	4	2.95	5.449	5.489	4.803	4.635	10.054	30.430	0.691	0.743	0.605	0.692	1.015	3.746
2	0	10	4	2.67	4.728	5.328	4.352	4.147	7.740	26.295	0.552	0.684	0.477	0.671	0.923	3.307
3	0	10	4	2.48	6.676	5.241	5.769	4.344	10.021	32.051	0.752	0.662	0.698	0.625	1.238	3.975
4	0	10	4	2.82	4.820	5.355	5.108	4.107	9.141	28.531	0.636	0.735	0.608	0.565	0.978	3.522
Mean	0	10	4	2.73	5.418	5.353	5.008	4.308	9.239	29.327	0.658	0.706	0.597	0.638	1.037	3.637
SE	0	0	0	0.10	0.389	0.044	0.258	0.104	0.470	1.074	0.037	0.017	0.039	0.024	0.060	0.125

Appendix table 1E. 5th Harvest (Nine weeks after sowing)

'Lustre'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	16.75	10	5	1.69	3.452	2.468	2.042	1.998	3.589	13.539	0.891	0.450	0.663	0.531	0.466	3.001
2	15.82	10	5	1.97	3.473	2.821	1.881	2.158	4.040	14.373	1.125	0.536	0.578	0.626	0.587	3.452
3	18.06	10	5	1.82	3.980	2.687	2.442	2.076	3.798	14.983	1.021	0.498	0.751	0.648	0.528	3.446
4	15.25	10	5	1.59	3.282	2.443	1.669	2.001	3.578	12.973	0.810	0.427	0.477	0.379	0.432	2.525
Mean	16.47	10	5	1.77	3.547	2.605	2.008	2.056	3.751	13.967	0.962	0.478	0.617	0.546	0.503	3.106
SE	0.53	0	0	0.08	0.130	0.078	0.142	0.034	0.094	0.385	0.061	0.021	0.051	0.053	0.030	0.191
CONTROL																
1	0	11	3	3.09	7.668	5.005	9.292	7.150	8.092	37.207	2.566	0.756	1.914	1.269	1.354	7.859
2	0	11	3	3.17	6.415	5.116	5.945	6.564	7.353	31.393	1.786	0.832	1.191	1.028	1.078	5.915
3	0	11	3	2.57	6.826	4.684	7.984	6.859	7.492	33.845	1.930	0.615	1.722	1.195	0.134	6.596
4	0	11	3	2.80	7.104	4.830	7.160	7.074	7.384	33.552	1.983	0.712	1.328	1.223	0.106	6.352
Mean	0	11	3	2.91	7.003	4.909	7.595	6.912	7.580	33.999	2.066	0.729	1.539	1.179	1.168	6.680
SE	0	0	0	0.14	0.228	0.083	0.609	0.114	0.150	1.040	0.149	0.039	0.146	0.045	0.055	0.361

Appendix table 1F, 6th Harvest (Ten weeks after sowing)

'Lustre'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total pla (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	16.24	11	7	1.64	6.201	2.345	5.208	2.588	3.812	20.154	1.418	0.356	0.859	0.396	0.542	3.148
2	17.74	11	6	1.70	5.902	2.516	5.639	2.822	3.735	20.614	1.285	0.569	1.228	0.465	0.722	4.402
3	18.61	11	6	1.47	5.911	2.508	4.830	2.040	3.600	18.889	1.287	0.427	0.706	0.339	0.454	3.213
4	23.25	11	6	1.58	5.520	2.558	5.220	2.354	3.424	19.076	0.995	0.475	1.011	0.573	0.598	3.942
Mean	18.96	11	6.25	1.60	5.883	2.482	5.224	2.451	3.643	19.683	1.246	0.457	0.951	0.443	0.579	3.676
SE	1.34	0	0.22	0.05	0.121	0.041	0.143	0.145	0.074	0.361	0.077	0.039	0.096	0.044	0.049	0.261
CONTROL																
1	0	11	4	2.57	13.135	3.505	19.646	6.537	8.021	50.844	3.394	0.629	3.737	1.046	1.224	10.030
2	0	11	3	3.15	11.886	3.875	20.145	6.996	10.38	53.282	3.234	0.794	4.215	1.556	1.626	10.425
3	0	11	3	2.92	10.293	3.664	16.007	6.458	7.213	43.635	2.725	0.728	2.847	0.923	0.905	8.128
4	0	11	4	2.82	10.710	3.661	18.885	6.667	8.406	48.329	2.600	0.689	3.377	1.269	1.342	9.277
Mean	0	11	3.50	2.87	11.506	3.676	18.671	6.664	8.505	49.022	2.738	0.710	3.544	1.198	1.274	9.465
SE	0	0	0.25	0.12	0.554	0.066	0.801	0.103	0.583	1.785	0.210	0.030	0.250	0.120	0.129	0.438

Appendix table 2A. Ist Harvest (Five weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weigh (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	8.35	5	0	0.69	1.692	1.537	0.452	0.231	0.959	4.871	0.142	0.176	0.056	0.033	0.090	0.497
2	7.16	5	0	0.70	1.450	1.246	0.328	0.128	0.671	3.823	0.125	0.157	0.047	0.036	0.065	0.430
3	6.39	5	0	0.80	1.430	1.565	0.593	0.098	0.751	4.437	0.119	0.191	0.061	0.031	0.078	0.480
4	6.62	5	0	0.72	1.528	1.413	0.617	0.257	0.892	4.707	0.129	0.180	0.077	0.035	0.112	0.533
Mean	7.13	5	0	0.73	1.525	1.440	0.497	0.178	0.818	4.459	0.129	0.176	0.060	0.034	0.086	0.485
SE	0.38	0	0	0.03	0.052	0.063	0.058	0.033	0.057	0.199	0.004	0.006	0.005	0.001	0.009	0.019
CONTROL																
1	0	5	0	0.77	1.465	1.529	0.504	0.341	0.910	4.749	0.117	0.184	0.057	0.045	0.107	0.510
2	0	5	0	0.84	1.332	1.634	0.575	0.135	1.126	4.802	0.108	0.193	0.072	0.022	0.115	0.510
3	0	5	0	0.73	1.519	1.426	0.475	0.158	0.853	4.431	0.128	0.174	0.043	0.032	0.085	0.462
4	0	5	0	0.69	1.765	1.338	0.692	0.061	1.090	4.946	0.138	0.169	0.058	0.054	0.112	0.531
Mean	0	5	0	0.76	1.520	1.482	0.561	0.174	0.995	4.732	0.123	0.180	0.057	0.038	0.105	0.503
SE	0	0	0	0.03	0.078	0.055	0.042	0.051	0.058	0.094	0.006	0.005	0.005	0.006	0.006	0.013

Appendix table 2B. 2nd Harvest (Six weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	17.42	7	2	1.25	2.114	2.205	1.351	0.648	2.055	8.373	0.189	0.285	0.164	0.144	0.188	0.970
2	20.12	7	2	1.36	2.459	2.010	1.160	0.529	2.422	8.580	0.203	0.302	0.150	0.133	0.238	1.026
3	17.75	7	2	1.32	2.345	2.267	1.602	0.819	2.021	9.054	0.209	0.293	0.184	0.151	0.211	1.048
4	16.39	7	2	1.21	2.784	2.122	1.322	0.468	1.808	8.504	0.234	0.284	0.123	0.122	0.179	0.942
Mean	17.92	7	2	1.28	2.425	2.151	1.359	0.616	2.076	8.628	0.209	0.291	0.155	0.137	0.204	0.996
SE	0.68	0	0	0.03	0.121	0.048	0.079	0.067	0.110	0.128	0.008	0.004	0.011	0.006	0.011	0.021
CONTROL																
1	0	7	1	1.30	2.694	1.976	1.896	0.871	2.058	9.495	0.208	0.311	0.178	0.160	0.200	1.057
2	0	7	1	1.50	2.336	2.478	1.471	1.290	2.270	9.845	0.199	0.322	0.113	0.166	0.216	1.016
3	0	7	1	1.53	2.828	2.544	2.040	1.250	2.493	11.155	0.232	0.330	0.214	0.207	0.249	1.232
4	0	7	1	1.32	2.057	2.161	1.698	0.501	2.259	8.676	0.172	0.316	0.159	0.131	0.202	0.980
Mean	0	7	1	1.41	2.479	2.290	1.776	0.978	2.270	9.793	0.203	0.320	0.166	0.166	0.217	1.071
SE	0	0	0	0.06	0.151	0.116	0.107	0.160	0.077	0.447	0.011	0.004	0.018	0.014	0.010	0.048

Appendix table 2C. 3rd Harvest (Seven weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	31.07	8	3	1.63	3.250	3.177	2.604	1.501	3.718	14.250	0.395	0.426	0.229	0.297	0.392	1.739
2	21.39	8	4	1.26	3.645	2.480	2.405	1.327	3.534	13.391	0.403	0.403	0.158	0.256	0.378	1.598
3	27.05	8	3	1.56	3.187	3.428	2.987	2.148	4.106	15.586	0.374	0.425	0.328	0.322	0.403	1.852
4	32.48	8	3	1.69	3.094	3.669	2.727	2.059	3.358	14.907	0.345	0.450	0.240	0.335	0.356	1.726
Mean	28.00	8	3.25	1.54	3.294	3.188	2.681	1.759	3.679	14.601	0.379	0.426	0.239	0.302	0.382	1.729
SE	2.15	0	0.22	0.09	0.105	0.222	0.105	0.176	0.139	0.451	0.011	0.008	0.030	0.015	0.009	0.045
CONTROL																
1	0	8	1	1.88	3.484	3.529	3.115	2.402	3.820	16.350	0.480	0.583	0.426	0.444	0.429	2.362
2	0	8	1	1.82	3.205	2.789	2.742	2.033	4.420	15.189	0.359	0.367	0.160	0.254	0.561	1.701
3	0	8	1	1.97	3.219	3.673	2.748	2.129	4.112	15.881	0.407	0.597	0.263	0.327	0.490	2.084
4	0	8	1	1.92	3.512	3.124	2.880	2.220	3.525	15.269	0.529	0.391	0.282	0.369	0.384	1.955
Mean	0	8	1	1.90	3.355	3.279	2.873	2.196	3.969	15.672	0.444	0.484	0.283	0.348	0.466	2.025
SE	0	0	0	0.04	0.072	0.174	0.076	0.068	0.166	0.237	0.033	0.053	0.047	0.034	0.033	0.119

Appendix table 2D, 4th Harvest (Eight weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	28.42	9	4	1.66	3.619	2.711	1.966	2.109	3.811	14.216	0.382	0.388	0.237	0.355	0.398	1.760
2	30.71	9	4	1.87	3.809	3.220	2.336	2.266	4.012	15.643	0.429	0.437	0.291	0.388	0.489	2.034
3	30.29	9	3	1.90	3.855	4.029	2.731	2.172	4.461	17.248	0.509	0.513	0.316	0.380	0.609	2.327
4	28.95	9	3	1.82	3.376	3.330	2.104	2.180	4.184	15.174	0.344	0.466	0.279	0.369	0.516	1.974
Mean	29.59	9	3.50	1.81	3.665	3.322	2.284	2.182	4.117	15.570	0.416	0.451	0.281	0.373	0.503	2.024
SE	0.47	0	0.25	0.05	0.094	0.235	0.145	0.028	0.119	0.548	0.031	0.023	0.014	0.006	0.038	0.101
CONTROL																
1	0	9	1	2.59	4.396	4.502	3.774	3.607	5.427	21.706	0.872	0.730	0.591	0.768	0.868	3.829
2	0	9	2	2.38	4.077	4.085	3.326	3.428	5.289	20.205	0.638	0.606	0.492	0.501	0.808	3.045
3	0	9	2	2.29	3.946	4.389	3.012	2.835	5.067	19.249	0.532	0.604	0.402	0.498	0.744	2.780
4	0	9	2	2.11	3.746	3.882	3.244	3.357	5.121	19.350	0.563	0.532	0.495	0.549	0.710	2.849
Mean	0	9	1.75	2.34	4.041	4.214	3.339	3.307	5.226	20.127	0.651	0.618	0.495	0.579	0.782	3.126
SE	0	0	0.22	0.10	0.118	0.123	0.138	0.144	0.071	0.492	0.067	0.036	0.033	0.055	0.030	0.209

Appendix table 2E. 5th Harvest (Nine weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	22.59	10	5	1.62	4.801	2.541	2.455	1.321	3.756	14.874	1.186	0.457	0.416	0.421	0.458	2.938
2	18.94	10	4	2.20	4.730	2.941	2.945	1.807	4.445	16.868	1.107	0.625	0.674	0.654	0.726	3.786
3	24.21	10	4	1.90	4.540	2.872	3.011	1.660	4.229	16.312	0.842	0.542	0.659	0.636	0.512	3.191
4	26.98	10	4	1.72	4.584	2.777	2.834	1.573	3.634	15.402	0.748	0.530	0.470	0.620	0.430	2.798
Mean	23.18	10	4.25	1.86	4.664	2.783	2.811	1.590	4.016	15.864	0.971	0.538	0.555	0.583	0.531	3.178
SE	1.71	0	0.22	0.13	0.053	0.076	0.108	0.088	0.166	0.387	0.091	0.030	0.058	0.047	0.058	0.189
CONTROL																
1	0	10	3	2.40	6.089	4.306	4.373	4.063	5.825	24.656	1.300	0.578	0.867	0.886	0.727	4.358
2	0	10	3	2.74	6.763	4.338	4.581	3.264	6.483	25.429	1.432	0.704	1.018	0.908	0.862	4.924
3	0	10	2	2.33	6.770	4.295	4.063	3.092	5.048	23.268	1.382	0.523	0.735	0.696	0.688	4.024
4	0	10	2	2.79	6.893	4.500	5.544	3.061	7.258	27.256	1.588	0.834	1.235	1.056	1.167	5.880
Mean	0	10	2.50	2.57	6.629	4.360	4.640	3.370	6.153	25.152	1.425	0.660	0.964	0.886	0.861	4.796
SE	0	0	0.25	0.12	0.158	0.041	0.277	0.204	0.408	0.720	0.052	0.060	0.093	0.064	0.094	0.352

Appendix table 2F, 6th Harvest (Ten weeks after sowing)

'Wild oat'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	28.08	10	5	1.54	5.362	2.276	2.535	2.639	2.103	14.915	1.183	0.424	0.846	0.405	0.458	3.316
2	22.44	10	4	1.85	5.750	2.569	3.657	3.363	2.607	17.946	1.665	0.486	1.243	0.715	0.723	4.832
3	18.84	10	5	1.50	5.034	2.167	2.322	2.417	1.978	13.918	1.040	0.398	0.778	0.333	0.439	2.988
4	24.44	10	4	1.72	5.489	2.349	2.963	3.187	2.355	16.343	1.325	0.438	1.029	0.566	0.616	3.974
Mean	23.45	10	4.50	1.65	5.409	2.340	2.869	2.901	2.261	15.780	1.303	0.436	0.974	0.505	0.559	3.777
SE	1.67	0	0.25	0.09	0.129	0.074	0.255	0.193	0.121	0.759	0.116	0.016	0.090	0.074	0.058	0.352
CONTROL																
1	0	10	3	2.37	9.582	3.424	14.448	5.321	6.029	38.804	2.040	0.569	2.906	0.841	1.018	7.344
2	0	10	3	2.69	9.976	3.756	15.840	5.719	6.174	41.465	2.533	0.704	3.235	1.154	1.287	8.883
3	0	10	3	2.20	8.327	3.192	10.779	3.823	5.565	31.686	1.818	0.512	1.822	0.510	0.779	5.411
4	0	10	3	2.45	8.697	3.545	11.210	4.149	4.158	31.759	1.894	0.592	2.064	0.723	0.819	6.062
Mean	0	10	3	2.43	9.145	3.479	13.069	4.753	5.481	35.928	2.071	0.594	2.507	0.807	0.976	6.925
SE	0	0	0	0.10	0.331	0.102	1.069	0.394	0.398	2.155	0.139	0.035	0.291	0.116	0.101	0.664

Appendix table 3A, Ist Harvest (Five weeks after sowing)

'Peniarth'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	8.69	5	1	0.57	1.663	1.227	0.311	0.097	1.043	4.341	0.152	0.155	0.029	0.034	0.090	0.460
2	9.48	5	1	0.67	1.563	1.451	0.409	0.326	1.076	4.825	0.143	0.177	0.049	0.044	0.101	0.514
3	8.01	5	1	0.75	1.559	1.611	0.386	0.086	0.788	4.430	0.139	0.184	0.034	0.038	0.078	0.473
4	9.67	5	1	0.61	1.660	1.343	0.322	0.414	0.798	4.537	0.142	0.169	0.031	0.040	0.084	0.466
Mean	8.96	5	1	0.65	1.611	1.408	0.357	0.231	0.926	4.533	0.144	0.171	0.036	0.039	0.088	0.478
SE	0.33	0	0	0.04	0.025	0.071	0.021	0.071	0.067	0.091	0.002	0.005	0.004	0.002	0.004	0.011
CONTROL																
1	0	5	0	0.61	1.638	1.375	0.216	0.111	0.925	4.265	0.167	0.172	0.035	0.036	0.096	0.506
2	0	5	0	0.67	1.428	1.482	0.299	0.223	0.878	4.310	0.136	0.190	0.030	0.039	0.083	0.478
3	0	5	0	0.69	1.567	1.634	0.678	0.431	0.781	5.091	0.156	0.213	0.055	0.043	0.086	0.553
4	0	5	0	0.80	1.457	1.660	0.410	0.217	1.118	4.862	0.140	0.216	0.040	0.047	0.106	0.549
Mean	0	5	0	0.69	1.522	1.538	0.401	0.245	0.925	4.632	0.150	0.198	0.040	0.041	0.093	0.521
SE	0	0	0	0.04	0.042	0.058	0.087	0.058	0.061	0.177	0.006	0.009	0.005	0.002	0.005	0.016

Appendix table 3B. 2nd Harvest (Six weeks after sowing)

'Peniarth

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	11.86	7	3	1.42	1.796	2.593	1.821	1.425	3.071	10.706	0.182	0.310	0.189	0.139	0.285	1.105
2	13.94	7	3	1.40	1.618	2.695	1.917	0.945	3.213	10.388	0.168	0.401	0.234	0.137	0.348	1.288
3	13.15	7	3	1.18	2.515	2.371	1.729	0.875	2.644	10.134	0.204	0.208	0.159	0.092	0.236	0.899
4	9.96	7	3	1.30	1.967	2.436	1.859	1.025	2.997	10.284	0.194	0.308	0.193	0.192	0.281	1.168
Mean	12.23	7	3	1.32	1.974	2.524	1.831	1.067	2.981	10.378	0.187	0.307	0.194	0.140	0.287	1.115
SE	0.75	0	0	0.05	0.168	0.064	0.034	1.107	0.105	0.105	0.007	0.034	0.013	0.018	0.020	0.070
CONTROL																
1	0	7	1	1.44	1.944	2.595	2.039	1.261	3.814	11.653	0.183	0.334	0.187	0.110	0.374	1.188
2	0	7	1	1.30	2.980	2.638	2.313	0.795	3.463	12.189	0.211	0.312	0.218	0.197	0.305	1.243
3	0	7	1	1.48	1.819	2.680	1.724	1.589	3.242	11.054	0.170	0.347	0.137	0.248	0.259	1.161
4	0	7	1	1.64	2.421	2.992	2.456	1.780	3.255	12.904	0.189	0.350	0.322	0.275	0.288	1.424
Mean	0	7	1	1.47	2.291	2.726	2.133	1.356	3.443	11.950	0.188	0.336	0.216	0.207	0.306	1.254
SE	0	0	0	0.07	0.228	0.078	0.140	0.187	0.116	0.341	0.007	0.007	0.034	0.031	0.021	0.051

Appendix table 3C. 3rd Harvest (Seven weeks after sowing)

'Peniarth'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	16.59	8	4	1.51	2.987	2.286	3.471	1.899	4.997	15.640	0.226	0.478	0.215	0.253	0.459	1.631
2	19.23	8	4	1.30	2.945	2.207	2.510	2.249	4.679	14.590	0.303	0.388	0.269	0.411	0.331	1.702
3	31.59	8	4	1.26	2.904	1.888	1.833	1.590	3.921	12.136	0.312	0.358	0.320	0.309	0.249	1.548
4	14.51	8	5	1.10	2.864	2.279	2.632	1.989	4.464	14.228	0.417	0.357	0.281	0.347	0.337	1.739
Mean	20.48	8	4.25	1.29	2.925	2.165	2.611	1.932	4.515	14.148	0.314	0.395	0.271	0.330	0.344	1.655
SE	3.31	0	0.22	0.08	0.023	0.081	0.291	0.118	0.196	0.636	0.034	0.025	0.019	0.029	0.037	0.036
CONTROL																
1	0	9	2	2.14	3.560	3.564	4.633	3.148	5.297	20.202	0.452	0.545	0.450	0.328	0.419	2.194
2	0	9	2	1.93	3.768	4.201	4.995	3.321	6.862	23.147	0.446	0.537	0.306	0.529	0.791	2.609
3	0	9	2	1.86	4.209	3.680	6.216	4.842	6.017	24.964	0.511	0.514	0.249	0.649	0.588	2.511
4	0	9	2	1.87	5.006	4.126	6.121	4.031	5.831	25.115	0.398	0.544	0.337	0.510	0.570	2.359
Mean	0	9	2	1.95	4.136	3.893	5.491	3.835	6.002	23.357	0.452	0.535	0.335	0.504	0.592	2.418
SE	0	0	0	0.06	0.277	0.138	0.345	0.334	0.281	0.990	0.020	0.006	0.037	0.057	0.066	0.079

Appendix table 3D. 4th Harvest (Eight weeks after sowing)

'Peniarth'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	22.54	9	5	1.38	3.140	1.855	1.312	1.626	3.159	11.092	0.443	0.311	0.364	0.494	0.401	2.013
2	18.85	9	5	1.56	3.260	2.691	2.483	1.528	3.689	13.651	0.432	0.584	0.584	0.573	0.527	2.800
3	12.40	9	6	1.02	2.722	1.615	1.373	1.259	2.683	9.652	0.217	0.287	0.418	0.381	0.321	1.624
4	17.89	9	5	1.42	3.050	2.138	1.732	1.086	3.554	11.560	0.320	0.375	0.439	0.350	0.444	1.928
Mean	17.92	9	5.25	1.34	3.043	2.075	1.725	1.375	3.271	11.489	0.353	0.389	0.476	0.449	0.423	2.091
SE	1.81	0	0.22	0.11	0.100	0.201	0.233	0.107	0.196	0.716	0.046	0.058	0.062	0.045	0.037	0.217
CONTROL																
1	0	10	3	2.24	5.756	4.317	4.501	5.371	7.960	27.905	0.659	0.562	0.527	0.805	1.175	3.728
2	0	10	2	2.59	5.395	4.349	6.127	3.704	6.030	25.605	0.566	0.588	0.611	0.488	0.678	2.931
3	0	10	2	2.05	5.553	3.862	6.267	3.780	6.142	25.604	0.619	0.551	0.703	0.549	0.766	3.188
4	0	10	3	1.90	6.161	3.629	6.402	4.334	7.233	27.759	0.733	0.530	0.737	0.745	0.968	3.713
Mean	0	10	2.50	2.20	5.716	4.039	5.824	4.297	6.841	26.718	0.644	0.558	0.644	0.647	0.897	3.390
SE	0	0	0.25	0.15	0.143	0.153	0.385	0.333	0.399	0.557	0.030	0.010	0.041	0.066	0.096	0.171

Appendix table 3E. 5th Harvest (Nine weeks after sowing)

'Peniarth'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	14.02	10	6	1.58	4.316	2.084	4.412	1.963	3.772	16.547	0.907	0.458	0.827	0.625	0.512	3.329
2	11.47	10	6	1.33	3.701	1.853	3.764	2.071	3.454	14.843	0.825	0.395	0.658	0.486	0.387	2.751
3	17.68	10	6	1.56	4.451	1.432	3.569	1.892	3.236	14.580	0.718	0.434	0.548	0.453	0.459	2.612
4	13.49	10	6	1.25	3.126	1.681	3.646	2.000	3.049	13.502	0.597	0.319	0.676	0.577	0.367	2.536
Mean	14.16	10	6	1.43	3.898	1.762	3.848	1.981	3.378	14.868	0.762	0.401	0.677	0.535	0.431	2.807
SE	1.12	0	0	0.08	0.264	0.119	0.167	0.032	0.134	0.546	0.058	0.026	0.050	0.043	0.079	0.156
CONTROL																
1	0	11	4	2.69	6.725	3.855	7.938	5.113	6.844	30.475	1.578	0.611	1.703	0.868	1.254	6.014
2	0	11	4	2.31	6.265	3.542	7.888	5.077	6.699	29.471	1.439	0.587	1.681	0.678	0.984	5.369
3	0	11	3	2.22	5.933	3.652	7.534	4.346	6.479	27.944	1.269	0.584	1.462	0.687	0.962	4.964
4	0	11	3	2.43	5.943	3.864	6.215	3.694	5.936	25.652	1.373	0.605	1.420	0.690	0.622	4.710
Mean	0	11	3.50	2.41	6.216	3.728	7.394	4.557	6.489	28.385	1.415	0.597	1.566	0.731	0.955	5.264
SE	0	0	0.25	0.10	0.161	0.068	0.349	0.292	0.172	0.909	0.056	0.006	0.063	0.040	0.112	0.246

Appendix table 3F. 6th Harvest (Ten weeks after sowing)

'Peniarth'

Repl.	% Infection	Total No of Leaves	No of Senesc Leaves	Leaf Area (dm^2)	Fresh Weight (g)					Total plant (g)	Dry Weight (g)					Total plant (g)
					LS	SLB	LT	TLB	Root		LS	SLB	LT	TLB	Root	
INFECTED																
1	10.90	10	6	1.42	7.105	1.662	7.060	3.802	3.336	22.965	1.291	0.374	1.104	0.327	0.480	3.576
2	16.15	10	6	1.52	7.345	1.700	8.550	4.036	3.589	25.220	1.420	0.454	1.280	0.504	0.650	4.308
3	12.70	10	6	1.28	7.086	1.633	6.382	2.219	2.957	20.277	0.952	0.406	0.928	0.269	0.338	2.893
4	12.40	10	6	1.08	6.804	1.554	4.966	1.519	2.519	17.362	0.881	0.325	0.821	0.206	0.315	2.548
Mean	13.04	10	6	1.33	7.085	1.637	6.739	2.894	3.100	21.456	1.136	0.390	1.033	0.326	0.446	3.331
SE	0.96	0	0	0.10	0.096	0.027	0.645	0.529	0.202	1.470	0.113	0.023	0.087	0.056	0.067	0.337
CONTROL																
1	0	11	4	2.34	11.497	2.726	17.641	4.279	6.492	42.635	2.376	0.674	3.108	0.719	1.302	8.179
2	0	11	4	2.79	10.179	3.120	19.517	6.316	6.826	45.958	2.085	0.701	3.574	1.036	1.545	8.871
3	0	11	4	2.13	9.574	2.722	16.272	3.975	6.280	38.823	1.783	0.609	2.598	0.508	0.895	6.423
4	0	11	4	2.29	9.960	2.918	16.607	3.320	5.850	38.655	1.872	0.574	2.763	0.561	0.962	6.732
Mean	0	11	4	2.38	10.302	2.871	17.509	4.472	6.362	41.518	2.029	0.639	3.001	0.706	1.176	7.551
SE	0	0	0	0.14	0.361	0.082	0.632	0.560	0.177	1.509	0.114	0.025	0.170	0.103	0.131	0.505

Appendix tables 4 to 6

- Data on the effects of infection on the development of individual leaves on the main axis, on the stem height, on the number of tillers and on total leaf blade area on tillers. Measurements taken at weekly intervals during the growth period between the fifth and tenth week after sowing.

Appendix table 4A, Ist harvest (5 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	0.030	20	0.040	20	0.030	20	0.030	15	0.032	0.25	18.85
2	0.050	15	0.070	15	0.050	12	0.050	10	0.055	0.50	13.18
3	0.120	15	0.110	15	0.100	15	0.140	10	0.117	0.85	13.51
4	0.230	8	0.220	5	0.200	8	0.220	5	0.217	0.63	6.48
5	0.320	0	0.360	0	0.340	0	0.380	0	0.350	1.29	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	6.65	12.00	1.00	0.390
2	5.75	10.00	1.00	0.340
3	5.97	10.00	2.00	0.430
4	4.21	10.00	2.00	0.510
Mean	5.65	10.50	1.50	0.417
SE	0.45	0.43	0.25	0.031

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	0.030	0.030	0.030	0.030	0.030	0.00
2	0.060	0.080	0.080	0.080	0.075	0.50
3	0.180	0.180	0.170	0.160	0.172	0.48
4	0.200	0.260	0.260	0.290	0.252	1.89
5	0.340	0.400	0.350	0.380	0.367	1.38

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	11.00	1.00	0.460
2	0.00	12.00	2.00	0.600
3	0.00	12.00	1.00	0.300
4	0.00	10.00	2.00	0.490
Mean	0.00	11.25	1.50	0.462
SE	0.00	0.41	0.25	0.107

CONTROL PLANTS

Appendix table 4B , 2nd harvest (6 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	0.090	50	0.080	50	0.130	40	0.090	35	0.097	1.11	43.20
4	0.140	35	0.110	40	0.180	40	0.130	45	0.140	1.47	40.00
5	0.250	25	0.250	20	0.310	20	0.280	25	0.272	1.44	22.43
6	0.390	15	0.370	15	0.440	15	0.410	15	0.402	1.49	15.00
7	0.530	8	0.540	5	0.600	5	0.570	8	0.560	1.58	6.47

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	18.38	15.00	3.00	0.480
2	16.03	15.00	2.00	0.730
3	17.00	12.00	3.00	0.430
4	18.05	16.00	1.00	0.860
Mean	17.36	14.50	2.25	0.625
SE	0.42	0.75	0.41	0.089

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	-
2	0.100	0.080	0.060	0.080	0.080	0.82
3	0.130	0.170	0.120	0.150	0.142	1.11
4	0.250	0.270	0.210	0.240	0.242	1.65
5	0.350	0.350	0.280	0.380	0.340	2.12
6	0.320	0.370	0.300	0.300	0.322	1.65
7	0.480	0.510	0.440	0.520	0.487	1.80

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	16.50	3.00	0.740
2	0.00	13.00	2.00	0.710
3	0.00	13.00	4.00	0.500
4	0.00	15.00	2.00	0.970
Mean	0.00	14.38	2.75	0.730
SE	0.00	0.74	0.41	0.083

CONTROL PLANTS

Appendix table 4C , 3rd harvest (7 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	0.200	60	0.230	80	0.220	70	0.240	70	0.222	1.25	70.34
6	0.430	50	0.400	35	0.360	30	0.450	50	0.410	1.96	41.95
7	0.530	10	0.520	20	0.490	12	0.580	8	0.530	1.87	11.90
8	0.540	1	0.570	3	0.540	2	0.590	2	0.560	1.22	1.77

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	23.14	18.50	4.00	1.270
2	25.88	19.00	3.00	1.830
3	19.65	17.00	2.00	1.130
4	24.26	22.00	2.00	0.770
Mean	23.23	19.12	2.75	1.250
SE	1.28	0.91	0.41	0.191

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	0.070	0.060	0.080	0.070	0.070	0.41
4	0.150	0.210	0.130	0.160	0.162	1.70
5	0.300	0.360	0.300	0.310	0.318	1.44
6	0.370	0.490	0.360	0.420	0.385	1.32
7	0.550	0.640	0.520	0.560	0.552	1.25
8	0.500	0.520	0.470	0.570	0.515	2.10
9	0.460	0.470	0.430	0.500	0.465	1.44

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	27.00	3.00	1.340
2	0.00	30.00	4.00	1.930
3	0.00	32.50	3.00	1.450
4	0.00	35.50	5.00	2.190
Mean	0.00	31.25	3.75	1.728
SE	0.00	1.57	0.41	0.174

CONTROL PLANTS

Appendix table 4D , 4th harvest (8 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	0.460	70	0.380	50	0.350	70	0.390	60	0.395	2.33	62.72
7	0.530	30	0.440	50	0.430	40	0.580	30	0.495	3.62	37.70
8	0.670	2	0.450	5	0.550	2	0.460	5	0.532	5.11	3.28
9	0.470	0	0.380	1	0.410	0	0.440	0	0.425	1.94	0.23

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	23.21	33.00	2.00	1.320
2	26.44	27.00	3.00	0.670
3	24.60	22.00	2.00	0.990
4	23.05	22.00	3.00	1.060
Mean	24.32	26.00	2.50	1.010
SE	0.70	2.26	0.25	0.116

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	SE	Mean Individual leaf area (dm^2)
1	-	-	-	-		-
2	-	-	-	-		-
3	0.080	0.080	0.050	0.070	0.71	0.070
4	0.160	0.130	0.100	0.120	1.25	0.128
5	0.330	0.280	0.230	0.300	2.10	0.285
6	0.420	0.420	0.380	0.450	1.44	0.417
7	0.560	0.520	0.520	0.540	0.96	0.535
8	0.530	0.450	0.460	0.490	1.80	0.482
9	0.460	0.420	0.410	0.460	1.31	0.438
10	0.410	0.370	0.330	0.340	1.71	0.375

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	40.00	4.00	3.180
2	0.00	34.00	3.00	2.720
3	0.00	48.00	5.00	2.410
4	0.00	39.00	5.00	1.610
Mean	0.00	40.25	4.25	2.480
SE	0.00	2.51	0.41	0.286

CONTROL PLANTS

Appendix table 4E , 5th harvest (9 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	0.120	80	0.170	70	0.150	70	0.100	80	0.135	1.55	74.07
7	0.350	40	0.400	35	0.360	30	0.320	35	0.357	1.65	36.08
8	0.460	10	0.520	10	0.500	15	0.420	10	0.475	2.22	11.32
9	0.420	1	0.500	1	0.460	1	0.450	0	0.457	1.65	0.75
10	0.340	0	0.380	0	0.350	0	0.300	0	0.342	1.65	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	16.93	34.50	3.00	1.630
2	15.02	52.00	3.00	1.120
3	18.05	38.00	4.00	1.780
4	15.25	29.00	2.00	0.950
Mean	16.31	38.37	3.00	1.370
SE	0.53	4.25	0.35	0.172

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	-	-	-	-	
4	0.180	0.150	0.110	0.140	0.145	1.44
5	0.220	0.260	0.170	0.200	0.212	1.89
6	0.330	0.370	0.300	0.280	0.320	1.96
7	0.450	0.470	0.390	0.420	0.433	1.75
8	0.560	0.570	0.500	0.520	0.538	1.65
9	0.520	0.550	0.460	0.480	0.502	2.02
10	0.450	0.450	0.360	0.420	0.420	2.12
11	0.380	0.350	0.280	0.340	0.338	2.10

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	83.00	5.00	4.050
2	0.00	77.00	5.00	2.280
3	0.00	56.00	5.00	3.170
4	0.00	54.00	4.00	3.690
Mean	0.00	67.50	4.75	3.298
SE	0.00	6.35	0.22	0.333

CONTROL PLANTS

Appendix table 4F , 6th harvest (10 weeks after sowing)

'Lustre'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	-	-	-	-	-	-	-	-	-		-
7	-	-	0.160	70	0.140	50	0.120	80	0.105	3.59	74.52
8	0.380	60	0.320	50	0.340	45	0.300	40	0.335	1.71	49.33
9	0.530	5	0.420	10	0.380	1	0.360	20	0.422	3.79	8.54
10	0.380	1	0.480	1	0.420	0	0.400	1	0.420	2.16	0.75
11	0.350	0	0.320	0	0.300	0	0.250	0	0.305	2.10	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	15.75	59.00	3.00	1.430
2	18.75	35.00	3.00	1.570
3	14.37	57.00	2.00	0.690
4	20.42	82.00	3.00	0.860
Mean	17.32	58.25	2.75	1.138
SE	1.34	8.32	0.22	0.185

INFECTED PLANTS

'Lustre'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	SE	Mean Individual leaf area (dm^2)
1	-	-	-	-		-
2	-	-	-	-		-
3	-	-	-	-		-
4	-	0.180	0.140	-	4.69	0.080
5	0.170	0.230	0.230	0.260	1.89	0.223
6	0.340	0.360	0.300	0.340	1.26	0.335
7	0.410	0.430	0.430	0.400	0.75	0.417
8	0.480	0.600	0.520	0.500	2.63	0.525
9	0.490	0.550	0.520	0.560	1.58	0.530
10	0.520	0.420	0.400	0.440	2.83	0.445
11	0.300	0.380	0.380	0.360	1.89	0.355

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	118.00	6.00	3.190
2	0.00	130.00	4.00	4.330
3	0.00	95.00	4.00	2.260
4	0.00	83.00	5.00	3.280
Mean	0.00	106.50	4.75	3.265
SE	0.00	9.25	0.41	0.423

CONTROL PLANTS

Appendix table 5A. Ist harvest (5 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	0.030	25	0.030	25	0.030	25	0.030	25	0.030	0.00	25.00
2	0.040	20	0.060	20	0.040	20	0.050	20	0.047	0.48	20.00
3	0.130	20	0.110	25	0.160	15	0.110	15	0.127	1.18	18.43
4	0.210	5	0.220	2	0.230	3	0.210	5	0.217	0.48	3.71
5	0.280	2	0.280	0	0.340	1	0.320	1	0.305	1.50	1.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	8.35	16.50	2.00	0.330
2	7.16	15.00	1.00	0.480
3	6.39	12.50	2.00	0.260
4	6.62	15.00	2.00	0.570
Mean	7.13	14.75	1.75	0.410
SE	0.38	0.72	0.22	0.061

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	0.030	0.030	0.030	0.030	0.030	0.00
2	0.040	0.050	0.040	0.040	0.042	0.25
3	0.140	0.160	0.120	0.120	0.135	0.96
4	0.230	0.230	0.220	0.220	0.225	0.29
5	0.330	0.370	0.320	0.280	0.325	1.85

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	15.00	2.00	0.510
2	0.00	14.00	2.00	0.220
3	0.00	16.00	1.00	0.300
4	0.00	17.00	1.00	0.620
Mean	0.00	15.50	1.50	0.412
SE	0.00	0.56	0.25	0.080

CONTROL PLANTS

Appendix table 5B , 2nd harvest (6 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	0.070	50	0.100	60	0.080	50	0.100	50	0.087	0.75	52.86
4	0.160	30	0.200	30	0.130	40	0.150	30	0.160	1.47	32.03
5	0.300	30	0.300	30	0.350	20	0.320	20	0.318	1.18	24.72
6	0.380	10	0.400	15	0.410	15	0.340	10	0.382	1.55	12.65
7	0.340	2	0.360	1	0.350	3	0.300	2	0.338	1.31	1.99

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	17.42	17.00	3.00	0.530
2	20.12	22.00	2.00	0.480
3	17.75	20.00	3.00	0.580
4	16.39	25.00	1.00	0.350
Mean	17.92	21.00	2.25	0.485
SE	0.68	1.46	0.41	0.043

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	0.050	0.040	0.040	0.030	0.040	0.41
3	0.120	0.140	0.150	0.120	0.133	0.75
4	0.180	0.240	0.200	0.160	0.195	1.71
5	0.250	0.330	0.340	0.320	0.310	2.04
6	0.400	0.480	0.400	0.350	0.408	2.69
7	0.300	0.300	0.370	0.340	0.327	1.70

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	22.50	3.00	0.600
2	0.00	22.00	2.00	0.720
3	0.00	26.00	4.00	0.890
4	0.00	18.00	2.00	0.490
Mean	0.00	22.12	2.75	0.675
SE	0.00	1.42	0.41	0.074

CONTROL PLANTS

Appendix table 4C , 3rd harvest (7 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	0.180	80	-	-	0.200	75	0.230	75	0.152	5.19	76.47
5	0.280	60	0.220	55	0.280	50	0.340	50	0.280	2.45	52.50
6	0.360	30	0.310	30	0.300	30	0.300	40	0.317	1.44	32.44
7	0.380	20	0.350	15	0.380	10	0.420	20	0.382	1.44	16.37
8	0.430	2	0.380	2	0.400	1	0.400	2	0.402	1.03	1.50

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	30.96	36.00	2.00	1.280
2	21.75	42.00	2.00	0.880
3	27.05	33.00	3.00	1.510
4	32.48	30.00	3.00	0.840
Mean	28.06	35.25	2.50	1.127
SE	2.15	2.22	0.25	0.140

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	0.070	0.060	0.090	0.060	0.070	0.71
3	0.100	0.140	0.200	0.120	0.140	2.16
4	0.250	0.200	0.240	0.220	0.228	1.11
5	0.280	0.280	0.300	0.340	0.300	1.44
6	0.360	0.380	0.320	0.400	0.365	1.71
7	0.400	0.370	0.400	0.380	0.387	0.75
8	0.420	0.390	0.420	0.400	0.407	0.75

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	42.00	4.00	1.850
2	0.00	28.50	3.00	1.270
3	0.00	32.00	3.00	1.650
4	0.00	47.00	3.00	1.560
Mean	0.00	37.38	3.25	1.582
SE	0.00	3.72	0.22	0.104

CONTROL PLANTS

Appendix table 4D , 4th harvest (8 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	0.160	80	0.130	80	0.072	4.23	80.00
5	0.260	70	0.350	70	0.280	60	0.300	70	0.297	1.93	67.65
6	0.340	50	0.370	50	0.300	40	0.350	30	0.340	1.47	42.50
7	0.400	25	0.380	30	0.440	30	0.370	25	0.397	1.55	27.52
8	0.360	5	0.410	5	0.370	5	0.350	2	0.372	1.31	4.30
9	0.300	2	0.360	2	0.350	2	0.320	2	0.332	1.38	2.25

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	28.67	36.00	2.00	1.140
2	30.57	41.00	3.00	1.590
3	30.18	50.00	3.00	1.300
4	28.95	28.00	3.00	1.010
Mean	29.59	38.75	2.75	1.260
SE	0.47	3.99	0.22	0.108

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	SE	Mean Individual leaf area (dm^2)
1	-	-	-	-		-
2	0.030	-	-	-	0.75	0.008
3	0.130	0.060	0.100	0.080	1.49	0.090
4	0.260	0.250	0.200	0.180	1.93	0.222
5	0.340	0.350	0.300	0.280	1.65	0.317
6	0.400	0.400	0.360	0.340	1.50	0.375
7	0.580	0.520	0.600	0.400	4.50	0.525
8	0.500	0.420	0.400	0.480	2.38	0.450
9	0.350	0.380	0.330	0.350	1.03	0.353

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	51.00	5.00	2.520
2	0.00	45.00	4.00	2.260
3	0.00	40.50	4.00	1.890
4	0.00	39.00	4.00	2.000
Mean	0.00	43.87	4.00	2.168
SE	0.00	2.33	0.22	0.122

CONRTOL PLANTS

Appendix table 4E , 5th harvest (9 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	0.180	70	0.100	75	0.120	80	0.100	3.74	74.25
6	0.260	60	0.330	50	0.300	50	0.250	50	0.285	1.85	52.28
7	0.340	30	0.420	15	0.350	30	0.350	40	0.365	1.85	28.08
8	0.360	20	0.510	10	0.460	20	0.410	20	0.435	3.23	17.07
9	0.360	10	0.400	2	0.380	10	0.330	8	0.368	1.49	7.37
10	0.300	0	0.360	0	0.310	0	0.260	0	0.307	2.06	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	22.59	75.00	2.00	1.090
2	18.94	66.00	4.00	1.340
3	24.21	52.00	3.00	1.930
4	26.98	43.00	3.00	1.680
Mean	23.18	59.00	3.00	1.510
SE	1.71	6.17	0.35	0.160

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	-	0.070	0.100	0.042	2.53
4	0.180	0.240	0.150	0.180	0.187	1.89
5	0.230	0.300	0.240	0.300	0.268	1.89
6	0.350	0.420	0.320	0.420	0.377	2.53
7	0.370	0.400	0.480	0.450	0.425	2.47
8	0.600	0.540	0.380	0.520	0.510	4.65
9	0.350	0.480	0.360	0.420	0.402	3.01
10	0.320	0.360	0.330	0.400	0.353	1.80

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	73.00	4.00	2.250
2	0.00	96.00	5.00	2.530
3	0.00	75.00	4.00	2.060
4	0.00	109.00	5.00	3.470
Mean	0.00	88.25	4.50	2.578
SE	0.00	7.49	0.25	0.271

CONTROL PLANTS

Appendix table 4F , 6th harvest (10 weeks after sowing)

'wild oat'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	0.130	50	-	-	0.090	80	0.055	3.28	62.27
6	0.200	70	0.240	50	0.230	50	0.270	50	0.235	1.44	54.46
7	0.340	50	0.350	40	0.300	30	0.350	30	0.335	1.19	37.69
8	0.370	20	0.420	10	0.380	15	0.380	20	0.392	1.60	16.12
9	0.340	10	0.400	10	0.350	5	0.350	10	0.360	1.35	8.79
10	0.250	1	0.300	0	0.240	2	0.280	1	0.267	1.38	0.96

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	28.08	72.00	2.00	1.120
2	21.89	100.00	3.00	1.670
3	18.95	79.00	3.00	0.830
4	24.76	84.00	3.00	1.410
Mean	23.40	83.75	2.75	1.258
SE	1.67	5.15	0.22	0.157

INFECTED PLANTS

'wild oat'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	-	-	-	-	
4	0.130	0.200	0.090	0.170	0.147	2.39
5	0.220	0.250	0.180	0.270	0.230	1.96
6	0.350	0.420	0.330	0.360	0.365	1.94
7	0.400	0.470	0.480	0.380	0.432	2.50
8	0.610	0.590	0.400	0.500	0.525	4.80
9	0.380	0.400	0.420	0.450	0.413	1.49
10	0.280	0.360	0.300	0.320	0.315	1.71

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	129.00	4.00	2.670
2	0.00	144.00	5.00	3.520
3	0.00	117.00	4.00	1.610
4	0.00	123.00	4.00	2.390
Mean	0.00	128.25	4.25	2.548
SE	0.00	5.02	0.22	0.341

CONTROL PLANTS

Appendix table 6A, Ist harvest (5 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	0.030	5	0.050	2	0.040	5	0.040	5	0.040	0.41	4.06
3	0.080	8	0.100	5	0.120	5	0.100	5	0.100	0.82	5.60
4	0.180	15	0.210	20	0.240	15	0.190	20	0.202	1.44	17.47
5	0.280	5	0.310	5	0.350	5	0.280	5	0.305	1.66	5.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	8.69	12.50	1.00	0.350
2	9.48	9.00	2.00	0.460
3	8.01	9.50	2.00	0.370
4	9.67	10.00	2.00	0.430
Mean	8.96	10.25	1.75	0.402
SE	0.33	0.67	0.22	0.022

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	0.020	0.030	0.030	0.040	0.030	0.41
2	0.030	0.040	0.040	0.050	0.040	0.41
3	0.090	0.090	0.120	0.140	0.110	1.22
4	0.180	0.210	0.200	0.230	0.205	1.04
5	0.290	0.300	0.300	0.340	0.307	1.11

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	11.00	2.00	0.350
2	0.00	10.00	2.00	0.390
3	0.00	10.00	3.00	0.430
4	0.00	9.00	2.00	0.510
Mean	0.00	10.00	2.25	0.420
SE	0.00	0.35	0.22	0.030

CONTROL PLANTS

Appendix table 6B , 2nd harvest (6 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	0.240	35	0.200	40	0.150	50	0.160	30	0.188	2.06	38.26
5	0.280	15	0.330	20	0.300	15	0.250	15	0.290	1.68	16.42
6	0.370	10	0.400	10	0.310	10	0.390	10	0.367	2.02	10.00
7	0.530	1	0.470	2	0.420	1	0.500	1	0.480	2.35	1.25

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	11.86	16.00	3.00	0.500
2	13.94	14.00	3.00	0.480
3	13.15	18.00	2.00	0.360
4	9.96	16.00	3.00	0.750
Mean	12.23	16.00	2.75	0.523
SE	0.75	0.71	0.22	0.071

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	0.040	0.040	0.030	0.050	0.040	0.41
3	0.090	0.080	0.080	0.120	0.092	0.95
4	0.180	0.180	0.200	0.240	0.200	1.41
5	0.250	0.230	0.300	0.280	0.265	1.55
6	0.330	0.320	0.390	0.420	0.365	2.40
7	0.550	0.450	0.480	0.530	0.502	2.29

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	16.00	3.00	0.810
2	0.00	18.50	4.00	0.950
3	0.00	14.50	2.00	0.980
4	0.00	16.00	4.00	1.140
Mean	0.00	16.25	3.25	0.970
SE	0.00	0.72	0.41	0.059

CONTROL PLANTS

Appendix table 6C , 3rd harvest (7 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	0.220	50	0.170	60	0.150	75	-	-	0.135	4.73	60.09
6	0.370	30	0.300	40	0.270	85	0.300	50	0.310	2.12	49.23
7	0.540	5	0.440	5	0.490	10	0.320	3	0.448	4.71	6.01
8	0.380	1	0.390	1	0.350	1	0.480	0	0.400	2.80	0.92

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	16.59	17.00	3.00	0.750
2	19.23	22.00	3.00	2.110
3	31.59	23.00	4.00	1.730
4	14.51	28.00	3.00	1.880
Mean	20.48	22.50	3.25	1.618
SE	3.31	1.95	0.22	0.259

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	0.060	0.060	0.070	0.070	0.065	0.29
4	0.150	0.150	0.140	0.180	0.155	0.87
5	0.330	0.280	0.260	0.280	0.287	1.49
6	0.370	0.340	0.360	0.320	0.347	1.11
7	0.420	0.380	0.360	0.360	0.380	1.41
8	0.450	0.390	0.370	0.370	0.395	1.89
9	0.360	0.330	0.300	0.290	0.320	1.58

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	34.00	5.00	1.690
2	0.00	30.00	4.00	1.940
3	0.00	44.00	4.00	2.280
4	0.00	25.00	4.00	2.050
Mean	0.00	33.25	4.25	1.990
SE	0.00	3.49	0.22	0.106

CONTROL PLANTS

Appendix table 6D , 4th harvest (8 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	0.220	60	0.250	70	-	-	0.200	60	0.167	5.68	63.73
7	0.380	35	0.300	30	0.250	40	0.410	25	0.335	3.66	31.75
8	0.460	10	0.580	5	0.470	5	0.510	5	0.505	2.72	6.14
9	0.320	0	0.430	0	0.300	1	0.300	2	0.338	3.12	0.67

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	22.54	29.00	2.00	1.610
2	18.85	33.00	4.00	1.970
3	12.40	22.00	3.00	1.230
4	17.89	26.00	3.00	1.420
Mean	17.92	27.50	3.00	1.558
SE	1.81	2.02	0.35	0.137

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	0.080	0.100	-	0.045	2.63
4	0.160	0.160	0.090	0.120	0.132	1.70
5	0.230	0.280	0.220	0.200	0.232	1.70
6	0.350	0.330	0.290	0.300	0.318	1.38
7	0.460	0.510	0.350	0.330	0.412	4.33
8	0.380	0.480	0.370	0.350	0.395	2.90
9	0.340	0.390	0.330	0.300	0.340	1.87
10	0.320	0.360	0.300	0.300	0.320	1.41

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	50.00	4.00	3.160
2	0.00	37.00	4.00	1.790
3	0.00	52.00	5.00	1.860
4	0.00	58.00	6.00	2.630
Mean	0.00	49.25	4.75	2.360
SE	0.00	3.83	0.41	0.284

CONTROL PLANTS

Appendix table 6E , 5th harvest (9 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	-	-	-	-	-	-	-	-	-		-
7	0.380	50	0.260	50	0.300	75	0.320	40	0.315	2.50	55.48
8	0.560	5	0.450	5	0.490	10	0.390	10	0.472	3.57	9.52
9	0.360	1	0.340	0	0.450	0	0.300	1	0.363	3.17	0.69
10	0.280	0	0.280	0	0.320	0	0.240	0	0.280	1.63	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	14.03	59.00	4.00	1.460
2	11.47	45.00	3.00	1.740
3	17.68	36.00	3.00	1.270
4	13.49	30.00	3.00	1.390
Mean	14.16	42.50	3.25	1.465
SE	1.12	5.46	0.22	0.086

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	1st plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	-	-	-	-	
4	-	-	0.080	0.120	0.050	3.00
5	0.200	0.250	0.180	0.220	0.212	1.49
6	0.350	0.200	0.230	0.290	0.267	3.33
7	0.440	0.420	0.390	0.370	0.405	1.55
8	0.640	0.480	0.450	0.540	0.528	4.19
9	0.400	0.340	0.300	0.330	0.342	2.10
10	0.360	0.360	0.340	0.330	0.348	0.75
11	0.300	0.260	0.250	0.230	0.260	1.47

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	96.00	6.00	3.450
2	0.00	75.00	6.00	1.850
3	0.00	52.00	5.00	2.260
4	0.00	61.00	5.00	2.530
Mean	0.00	71.00	5.50	2.643
SE	0.00	8.30	0.25	0.290

CONTROL PLANTS

Appendix table 6F , 6th harvest (10 weeks after sowing)

'Peniarth'

Leaf No on main axis	Ist plant		2nd plant		3rd plant		4th plant		Mean Individual leaf area (dm^2)	SE	Mean % Infection
	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect	Indiv La(dm^2)	% Infect			
1	-	-	-	-	-	-	-	-	-		-
2	-	-	-	-	-	-	-	-	-		-
3	-	-	-	-	-	-	-	-	-		-
4	-	-	-	-	-	-	-	-	-		-
5	-	-	-	-	-	-	-	-	-		-
6	-	-	-	-	-	-	-	-	-		-
7	0.320	45	0.300	70	0.250	60	0.180	60	0.262	3.12	58.28
8	0.540	2	0.570	5	0.450	2	0.390	5	0.487	4.13	3.47
9	0.350	0	0.350	2	0.360	1	0.320	2	0.345	0.87	1.23
10	0.210	0	0.300	0	0.220	0	0.190	0	0.230	2.42	0.00

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	10.90	70.00	3.00	1.060
2	16.15	84.00	2.00	1.550
3	12.70	51.00	4.00	0.880
4	12.40	57.00	3.00	0.690
Mean	13.04	65.50	3.00	1.045
SE	0.96	6.35	0.35	0.160

INFECTED PLANTS

'Peniarth'

Leaf No on main axis	Ist plant Individual Leaf area (dm^2)	2nd plant Individual Leaf area (dm^2)	3rd plant Individual Leaf area (dm^2)	4th plant Individual Leaf area (dm^2)	Mean Individual leaf area (dm^2)	SE
1	-	-	-	-	-	
2	-	-	-	-	-	
3	-	-	-	-	-	
4	-	-	-	-	-	
5	0.180	0.260	0.170	0.150	0.190	2.42
6	0.240	0.320	0.190	0.240	0.247	2.69
7	0.400	0.530	0.350	0.370	0.412	2.69
8	0.490	0.650	0.440	0.520	0.525	4.48
9	0.320	0.370	0.290	0.300	0.320	1.78
10	0.390	0.380	0.350	0.350	0.368	1.03
11	0.320	0.280	0.340	0.360	0.325	1.71

Replicates	% Infection	Stem Height (cm)	Tillers No / plant	La. on tillers (dm^2)
1	0.00	136.00	6.00	2.570
2	0.00	104.00	5.00	2.940
3	0.00	92.00	5.00	1.950
4	0.00	108.00	6.00	2.230
Mean	0.00	110.00	5.50	2.423
SE	0.00	8.06	0.25	0.185

CONTROL PLANTS

Appendix table 7

Primary values on the effects of Erysiphe graminis on yield components and final grain yield of infected (I) and uninfected (C) plants of cv. Lustre.

Replic.	Number of tillers		Number of fertile tillers		Number of grain per panicle[1]		Number of grain per plant		Hundred grain weight (g)		Total grain yield (g)		Total plant d. wt (g)		Harvest Index	
	I	C	I	C	I	C	I	C	I	C	I	C	I	C	I	C
1	3	5	2	4	53	38	73	166	1.889	2.562	1.379	4.253	7.147	13.193	16.17	24.38
2	4	5	2	4	46	45	101	164	2.141	2.701	2.162	4.429	5.429	11.585	28.48	27.66
3	2	3	3	3	37	68	71	146	1.777	2.266	1.262	3.308	4.149	7.538	23.32	30.50
4	4	4	3	3	28	52	80	163	1.748	1.901	1.398	3.099	4.664	8.402	23.06	26.94
5	3	2	2	2	39	54	96	137	1.903	1.950	1.827	2.671	5.172	9.334	26.10	22.25
6	3	3	2	2	52	77	104	132	1.565	2.025	1.628	2.673	6.488	10.454	20.06	20.36
7	4	2	2	3	33	49	58	176	1.736	2.492	1.007	4.386	2.733	8.732	26.92	33.43
8	2	5	3	4	38	37	74	129	1.893	1.926	1.401	2.484	2.965	7.449	32.09	25.01
9	3	3	3	3	21	44	119	141	1.618	2.479	1.925	3.495	4.234	7.772	31.25	31.02
10	4	4	3	3	45	51	63	148	2.035	2.233	1.282	3.305	5.750	9.124	18.23	26.59
Mean	3.2	3.6	2.5	3.1	39.2	51.5	85.2	150.2	1.830	2.253	1.527	3.410	4.873	9.358	24.57	26.81
SE	0.2	0.3	0.2	0.2	3.1	3.8	8.1	4.8	0.053	0.088	0.106	0.218	0.426	0.563	1.70	1.28

1 main axis panicle.

Appendix table 8

Primary values on the effects of Erysiphe graminis on yield components and final grain yield of infected (I) and uninfected (C) plants of wild oat.

Replic.	Number of tillers		Number of fertile tillers		Number of grain per panicle[1]		Number of grain per plant		Hundred grain weight (g)		Total grain yield (g)		Total plant d. wt (g)		Harvest Index	
	I	C	I	C	I	C	I	C	I	C	I	C	I	C	I	C
1	3	5	3	4	55	63	86	183	1.074	1.294	0.924	2.368	3.586	7.368	20.49	24.32
2	5	4	3	4	39	58	79	164	1.025	1.366	0.810	2.240	4.904	8.883	14.17	20.14
3	4	4	3	4	41	86	98	205	1.228	1.272	1.203	2.608	4.566	5.409	20.85	32.53
4	4	6	3	5	66	60	139	224	1.265	1.389	1.758	3.111	4.065	6.039	30.19	34.00
5	5	4	4	3	54	74	121	171	1.393	1.249	1.685	2.136	5.267	8.557	24.24	19.98
6	2	2	2	3	38	52	70	151	1.114	1.474	0.780	2.226	2.783	8.349	21.89	21.05
7	3	4	3	3	47	55	94	139	1.094	1.477	1.028	2.053	4.313	6.444	19.24	24.16
8	3	5	4	4	64	77	108	155	1.393	1.287	1.504	1.995	5.195	7.218	22.45	21.65
9	2	2	3	3	48	65	114	146	1.167	1.274	1.330	1.860	3.037	7.161	30.45	20.62
10	4	4	2	4	65	51	68	181	1.011	1.173	0.688	2.123	2.977	5.594	18.77	27.51
Mean	3.5	4.0	3.0	3.7	51.7	64.1	97.7	171.9	1.176	1.325	1.171	2.272	4.069	7.102	22.27	24.60
SE	0.3	0.4	0.2	0.3	3.2	3.5	6.9	8.1	0.042	0.030	0.116	0.108	0.280	0.369	1.58	1.63

1 main axis panicle.

Appendix table 9

Primary values of the effects of Erysiphe graminis on yield components and final grain yield of infected (I) and uninfected (C) plants of cv. Peniarth.

Replic.	Number of tillers		Number of fertile tillers		Number of grain per panicle[1]		Number of grain per plant		Hundred grain weight (g)		Total grain yield (g)		Total plant d. wt (g)		Harvest Index	
	I	C	I	C	I	C	I	C	I	C	I	C	I	C	I	C
1	4	4	3	3	39	42	78	126	1.651	1.833	1.288	2.310	3.474	7.688	27.05	23.10
2	4	3	3	3	44	54	89	145	1.865	1.945	1.660	2.820	4.836	9.148	25.55	23.56
3	3	3	3	4	52	52	95	161	1.487	2.120	1.413	3.413	3.867	6.424	26.76	34.69
4	2	5	3	3	34	40	64	138	1.741	1.864	1.114	2.572	2.547	6.744	30.42	27.61
5	5	4	4	5	50	68	102	164	1.640	2.394	1.673	3.926	5.751	11.233	22.53	35.18
6	3	3	3	4	23	40	72	136	1.564	1.903	1.126	2.588	4.667	10.327	19.44	20.04
7	4	5	2	4	37	43	68	147	1.921	2.131	1.306	3.133	4.162	8.263	23.88	27.49
8	5	6	3	3	34	48	55	124	1.798	2.032	0.989	2.520	3.328	7.767	22.91	24.50
9	3	6	2	4	29	45	45	138	1.527	1.909	0.687	2.634	2.773	7.592	19.86	25.76
10	4	3	2	3	47	46	77	118	1.535	2.544	1.182	3.002	3.734	6.619	24.04	31.20
Mean	3.7	4.2	2.8	3.6	38.9	47.8	74.5	139.7	1.673	2.067	1.244	2.892	3.914	8.180	24.25	27.31
SE	0.3	0.4	0.2	0.3	2.8	2.6	5.3	4.5	0.046	0.071	0.089	0.147	0.293	0.482	1.06	1.59

1 main axis panicle.

Appendix table 10

Measurements in ***the light*** of diffusive resistances of infected and uninfected third leaf of cvs Lustre, Peniath and wild oat .

Days after inocul.	Replicates	Lustre				wild oat				Peniarth			
		Infected		Control		Infected		Control		Infected		Control	
		M	T	M	T	M	T	M	T	M	T	M	T
1	1	4.0	4.2	4.0	4.1	3.3	3.3	3.6	3.6	2.8	2.8	3.2	3.3
	2	3.8	4.2	4.0	4.1	3.3	3.5	3.4	3.5	3.2	3.2	3.5	3.5
	3	4.0	4.5	5.0	5.2	3.6	3.5	3.6	3.6	2.9	3.2	3.3	3.5
	4	4.2	5.2	5.2	5.2	3.6	3.8	3.3	3.5	3.3	3.0	3.2	3.3
	Mean	**4.00**	**4.53**	**4.55**	**4.65**	**3.45**	**3.53**	**3.48**	**3.55**	**3.05**	**3.05**	**3.30**	**3.40**
	Std	**0.16**	**0.47**	**0.64**	**0.64**	**0.17**	**0.21**	**0.15**	**0.06**	**0.24**	**0.19**	**0.14**	**0.12**
3	1	2.9	3.0	4.0	4.0	3.0	3.1	2.9	3.2	2.2	2.7	2.9	2.9
	2	3.2	2.9	3.9	3.9	2.9	2.9	3.0	3.2	2.3	2.9	2.8	3.0
	3	3.0	3.3	3.5	4.0	3.0	3.4	3.3	3.4	2.3	2.9	2.8	2.8
	4	3.0	3.4	3.5	3.5	2.9	3.0	3.2	3.5	2.7	3.0	2.7	2.9
	Mean	**3.03**	**3.15**	**3.73**	**3.85**	**2.95**	**3.10**	**3.10**	**3.32**	**2.38**	**2.88**	**2.80**	**2.92**
	Std	**0.13**	**0.24**	**0.26**	**0.24**	**0.06**	**0.22**	**0.18**	**0.15**	**0.22**	**0.13**	**0.08**	**0.08**
5	1	3.6	3.0	3.0	3.3	2.9	3.2	2.6	2.6	3.0	2.9	2.6	2.9
	2	3.5	3.2	3.3	3.3	3.2	3.2	2.5	2.9	3.5	3.4	3.0	2.9
	3	3.6	3.5	3.7	3.7	2.6	2.8	2.8	2.8	3.4	3.1	2.8	3.0
	4	3.7	3.7	2.9	3.0	3.2	3.7	2.3	2.6	3.3	3.2	2.8	3.2
	Mean	**3.60**	**3.35**	**3.23**	**3.33**	**2.98**	**3.23**	**2.55**	**2.72**	**3.30**	**3.15**	**2.80**	**3.00**
	Std	**0.08**	**0.31**	**0.36**	**0.29**	**0.29**	**0.37**	**0.21**	**0.15**	**0.22**	**0.21**	**0.16**	**0.14**
7	1	4.7	5.5	4.2	4.3	3.7	3.3	2.8	2.5	3.4	3.3	2.8	2.7
	2	5.8	4.1	3.7	4.0	2.9	3.0	2.3	2.9	3.2	2.9	2.6	2.9
	3	4.9	4.9	3.8	4.6	2.9	3.2	2.6	2.9	3.0	3.5	3.1	2.9
	4	4.8	4.7	4.4	4.5	3.3	2.8	2.6	2.7	3.5	3.8	2.9	2.8
	Mean	**5.05**	**4.80**	**4.03**	**4.35**	**3.20**	**3.08**	**2.58**	**2.75**	**3.28**	**3.38**	**2.85**	**2.83**
	Std	**0.51**	**0.58**	**0.33**	**0.26**	**0.38**	**0.22**	**0.21**	**0.19**	**0.22**	**0.38**	**0.21**	**0.10**
9	1	4.0	4.7	3.1	3.3	3.2	3.5	2.6	2.6	3.4	3.6	2.3	2.4
	2	4.4	3.6	3.5	3.8	3.2	2.6	2.6	2.8	3.3	3.2	2.4	2.6
	3	3.8	3.8	3.5	3.5	3.9	4.2	2.2	2.6	2.7	2.6	2.5	2.6
	4	3.8	4.0	3.4	3.6	2.7	2.7	2.4	2.5	3.6	3.8	2.6	2.6
	Mean	**4.00**	**4.03**	**3.37**	**3.55**	**3.25**	**3.25**	**2.45**	**2.62**	**3.25**	**3.30**	**2.45**	**2.56**
	Std	**0.28**	**0.48**	**0.19**	**0.21**	**0.49**	**0.75**	**0.19**	**0.13**	**0.39**	**0.53**	**0.13**	**0.10**

M = middle region of the leaf , T = tip of the leaf .

Appendix table 11

Measurements in ***the dark*** of diffusive resistances of infected and uninfected third leaf of cvs Lustre, Peniarth and wild oat .

Days after inocul.	Replicates	Lustre				wild oat				Peniarth			
		Infected		Control		Infected		Control		Infected		Control	
		M	T	M	T	M	T	M	T	M	T	M	T
1	1	8.4	8.8	8.9	9.0	7.1	7.3	8 .0	8.0	7.3	7.6	8.0	8.3
	2	8.6	8.8	9.2	9.4	7.5	7.6	8.1	8.3	7.2	7.6	8.1	8.1
	3	8.9	9.2	9.8	9.9	7.6	8.0	7.5	7.5	7.4	7.6	7.8	8.1
	4	9.0	9.2	8.2	9.4	7.5	8.0	7.8	8.1	7.1	7.3	8.1	8.3
	Mean	**8.73**	**9.00**	**9.03**	**9.43**	**7.43**	**7.73**	**7.85**	**7.98**	**7.25**	**7.53**	**8.00**	**8.20**
	Std	**0.28**	**0.23**	**0.67**	**0.37**	**0.22**	**0.34**	**0.26**	**0.34**	**0.13**	**0.15**	**0.14**	**0.12**
3	1	6.9	7.0	8.4	8.6	6.7	7.4	7.5	7.7	6.4	6.8	8.1	8.5
	2	7.5	7.2	8.1	8.3	6.2	6.8	7.5	7.7	6.8	7.7	8.0	8.5
	3	6.8	7.0	8.1	8.5	6.6	7.0	7.2	7.5	6.2	7.7	8.1	8.5
	4	7.0	7.2	8.5	8.7	6.6	6.8	7.4	7.6	6.1	6.7	7.9	8.0
	Mean	**7.05**	**7.10**	**8.28**	**8.53**	**6.53**	**7.00**	**7.40**	**7.63**	**6.38**	**7.23**	**8.03**	**8.37**
	Std	**0.31**	**0.12**	**0.21**	**0.17**	**0.22**	**0.28**	**0.14**	**0.10**	**0.31**	**0.55**	**0.10**	**0.25**
5	1	6.1	6.1	7.2	7.6	5.2	5.2	7.7	7.7	5.0	5.6	7.8	8.0
	2	6.0	5.6	6.8	7.0	5.0	4.9	7.3	7.8	5.4	5.6	8.0	8.2
	3	5.5	5.3	6.8	7.3	4.7	5.0	7.6	7.8	6.6	7.0	7.5	8.0
	4	6.1	6.0	7.3	7.0	5.2	5.3	7.8	7.8	5.2	6.5	7.7	7.8
	Mean	**5.93**	**5.75**	**7.03**	**7.23**	**5.03**	**5.10**	**7.60**	**7.78**	**5.55**	**6.18**	**7.75**	**8.00**
	Std	**0.29**	**0.37**	**0.26**	**0.29**	**0.24**	**0.18**	**0.22**	**0.05**	**0.72**	**0.69**	**0.21**	**0.16**
7	1	6.8	5.5	7.7	8.0	3.6	3.5	6.5	6.5	4.1	4.2	7.0	7.2
	2	5.8	6.5	7.5	7.7	3.5	3.5	6.5	6.7	4.3	4.3	6.6	6.8
	3	5.3	5.5	7.3	7.5	6.2	3.6	6.9	7.0	3.8	3.8	6.8	7.0
	4	5.5	5.8	7.6	7.8	4.2	4.3	6.6	7.0	4.2	4.6	6.6	6.8
	Mean	**5.85**	**5.83**	**7.53**	**7.75**	**4.38**	**3.73**	**6.63**	**6.80**	**4.10**	**4.23**	**6.75**	**6.95**
	Std	**0.67**	**0.47**	**0.17**	**0.21**	**1.26**	**0.39**	**0.19**	**0.24**	**0.22**	**0.33**	**0.19**	**0.19**
9	1	4.5	4.5	6.9	6.7	4.3	4.5	7.2	7.3	3.4	4.1	6.1	6.3
	2	4.4	4.9	7.2	6.9	4.1	4.0	7.5	7.7	4.1	4.1	6.9	6.7
	3	5.4	5.3	6.7	6.7	4.4	5.0	7.5	7.7	4.1	3.8	6.6	6.7
	4	4.3	4.3	7.3	7.1	4.0	4.3	7.7	7.8	4.0	3.8	5.9	6.1
	Mean	**4.65**	**4.75**	**7.02**	**6.85**	**4.20**	**4.45**	**7.47**	**7.62**	**3.90**	**3.95**	**6.37**	**6.45**
	Std	**0.51**	**0.44**	**0.28**	**0.19**	**0.18**	**0.42**	**0.21**	**0.22**	**0.34**	**0.17**	**0.46**	**0.30**

M = middle region of the leaf , T = tip of the leaf.

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