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**FUSARIUM DISEASES
OF CEREALS**

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FUSARIUM DISEASES OF CEREALS

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ABSTRACT

Several species of fungi grouped together in the genus Fusarium affect cereals, causing a number of different diseases and also the production of toxins (mycotoxins) which are harmful to humans and animals. The review summarises the information available on both subjects, mainly from UK sources, and defines areas where information is lacking.

The species of Fusarium which cause diseases are F. avenaceum, F. culmorum, F. graminearum, F. nivale and F. poae. All five species cause diseases of the ear and can affect the grain. They (and some others), under suitable conditions, also produce mycotoxins. F. poae causes only a disease of the ear but the other four species also cause diseases of seedlings and stem bases (foot rots, often referred to as brown foot rot). F. nivale can cause a leaf disease, including a disease called snow mould.

Fusarium diseases have been reported since the latter part of the nineteenth century. Ear blight, which is conspicuous as bleached spikelets, has attracted attention periodically. Seedling diseases can result in a thinning of the crop and are usually associated with the use of untreated seed. Foot rots are only rarely severe enough to cause whiteheads. More often the symptoms are lesions on the lowest nodes and internodes and there are reports (not well authenticated) that such infections cause a weakening of the stem which may lead to premature ripening or lodging when the crop is mature.

Infections of seedlings can result in pre- or post-emergence death or may be limited to lesions on the leaf sheaths of plants which survive. Infections by F. avenaceum, F. culmorum and F. graminearum are most severe in warm, dry soils and by F. nivale in cool, dry soils. There are important interactions between the main factors affecting seedling infections, which are soil moisture, soil temperature and spore load on the seed. All the pathogens can be seed-borne and soil-borne. Severe disease is usually associated with high spore loads on the seed (except for F. nivale under otherwise favourable conditions) so that severe seedling disease in the field is usually associated with the use of infected seed which has not been treated with a fungicide. It occurs mainly in winter wheat which is more susceptible than barley. F. nivale, which is best adapted to the environmental conditions in the autumn, is the common cause of seedling disease. A severe thinning of the stand can lead to loss of yield but, in normal growing systems, such losses are rare.

In some soils F. nivale causes little or no disease and this has been associated with the presence of other fungi such as Gliocladium roseum and Trichoderma viride. However, neither of these fungi has provided a very good control of disease when applied artificially to seed or soil.

The seed-borne Fusarium spp. are only partially controlled by organomercury because the chemical does not eradicate deep-seated infections. The triazole fungicides, triadimenol (in "Baytan") and flutriafol (in "Ferrax"), give a similar level of control to organomercury. Both "Baytan" and "Ferrax" contain an MBC-fungicide to improve the control of Fusarium spp., but one of the common seed-borne pathogens, F. nivale, is now mainly resistant to MBC fungicides so that this improved control is unlikely to be maintained. Nevertheless, the level of control given by current seed treatments is sufficient to prevent serious losses of seedlings.

Plants which survive but are infected by Fusarium provide the sources for spread to other parts of the plant. The incidence and severity of these diseases is related to the amount of the pathogen in the crop and the weather.

F. nivale causes snow mould - a disease where leaves, and sometimes the crowns, can be killed under snow cover. Snow mould occurs mainly on winter barley in Scotland and is often found together with snow rot (caused by Typhula incarnata) which is much the more damaging disease at that stage. F. nivale also causes a brown leaf spot which is sometimes prominent on the upper leaves of wheat and especially of triticale but at present is not sufficiently severe to cause loss of yield.

ADAS surveys in recent years have shown that a mean 22-38 per cent of the stem bases of winter wheat and winter barley crops have lesions caused by Fusarium spp., though less than 1 per cent have severe lesions causing a breaking or a softening of the stem tissues. There are ad hoc reports from the field of Fusarium foot rots causing severe damage especially late in the season, but there are no objective accounts of the symptoms, the specific causes or the damage done. There is a need for a thorough investigation of Fusarium foot rots including an assessment of the damage they cause.

There is little information on the interactions of Fusarium stem base diseases and others such as eyespot and sharp eyespot.

In a few trials a spray of the fungicide prochloraz, applied in the spring for the control of eyespot, has reduced the incidence of Fusarium foot rots by about 33 per cent. However, there is no evidence that prochloraz or any other fungicide, as used at present, will consistently give a satisfactory control of the disease. Also there is no information on varietal resistances, if any exist, to Fusarium foot rots.

The earliest signs of ear infections in wheat are water-soaked spots on the glumes. The disease can progress to affect whole spikelets which soon become bleached in contrast to the healthy green spikelets. If the infection progresses into the rachis then all parts above die and also become bleached. All five species, F. avenaceum, F. culmorum, F. graminearum, F. nivale and F. poae, have produced these symptoms following artificial inoculation. F. culmorum is said

to be the most common cause in field crops but no objective surveys have been done. Symptoms caused by F. poae and F. nivale are sometimes limited to lesions with brown margins on the glumes. The disease is favoured by wet weather and a prolonged ripening period. Infections occur mainly in the flowering period and are much stimulated by the presence of pollen.

Spread of disease to the upper parts of the plant and to the ear is by the dissemination of spores from the lower part of the plant. In the case of F. nivale, ascospores produced in the perithecia which occur abundantly on leaf sheaths, are known to be an important means of spread. For this and the other species (which do not produce ascospores) it is assumed that spread is through rain-splashed or air-borne conidia. However, good information on the production and dissemination of spores is lacking.

Severe ear blight occurs erratically and even in 1982, when it was particularly prominent, assessments made in ADAS surveys indicated that national losses were small (less than 1 per cent).

There are varietal differences in susceptibility to ear blight and selection for resistance is now included in plant breeding programmes. There is little information available on the control of ear blight with fungicides.

The conditions which favour ear blight also favour the formation of Fusarium mycotoxins. About twenty mycotoxins occur naturally on cereals but many more related compounds have been identified in laboratory cultures. Fusarium mycotoxicosis is rare in humans but instances in livestock are reported more frequently. In the UK data on the subject has been collected by ADAS, the Flour Milling and Baking Research Association, the Edinburgh School of Agriculture and others. Examination of various species of Fusarium on wheat has shown that many isolates are toxigenic but their potential is rarely realised in the field, presumably because weather conditions are not favourable. Surveys have shown that some mycotoxins are frequently present at very low levels on home-grown grain but there is little

evidence that they pose a widespread threat to man or livestock. One possible exception could be cereals used for livestock feed in parts of Scotland where the prolonged and often late harvest can favour the growth of Fusarium spp. on the grain.

Recommendations for further work

The recommendations for further work can be summarised as follows.

1. A study of the specific causes of Fusarium foot rots, the conditions affecting disease development and the effects of the diseases on the plants, especially in grain yield and quality. (Highest priority).
2. A survey for several years of the incidence and causes of Fusarium foot rots in field crops.
3. Ear blight:
 - i. an assessment of the causes of ear blight in field crops and of the damage done.
 - ii. epidemiology to include sources of infection, the production and dissemination of spores.
 - iii. a study of the factors affecting infection by the five Fusarium species known to cause ear blight symptoms.
4. Monitoring seed-borne disease.
5. Independent tests of new seed treatments and monitoring the effectiveness of chemicals on the market, especially in relation to fungicide resistance.

6. Mycotoxins:

- i. surveillance of home-grown cereals in different parts of the UK for Fusarium mycotoxins, using the most sensitive methods available, in order to establish their incidence. The investigation to be done over several years so as to include different climatic conditions at harvest.
- ii. an assessment of the significance of Fusarium mycotoxins in grain to establish "acceptable" levels of contamination for each mycotoxin or combination of mycotoxins at which no harmful effects on man or livestock would be expected.

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GLOSSARY

anamorph	asexual spore form of a fungus (imperfect state).
anthesis	flowering: appearance of anthers in cereals.
ascospore	fungal spores formed in an ascus.
ascus	fungal spore sac often in a fruit-body, e.g. perithecium; sexual stage.
brown foot rot	common name for foot rot caused by <u>Fusarium</u> spp.
chlamydospore	asexual fungal spore, thick-walled, survives unfavourable conditions, often long-lived.
coleorhiza	sheath surrounding radicle (root in embryo).
coleoptile	protective sheath around shoot in seedling.
conidium	asexual fungal spore.
culm	stem, in cereals stem inside sheath.
endophyte	of a fungus, living within the plant, no symptoms.
endosperm	in the seed, tissues which provide nutrients for embryo.
epidermis	outermost layer of cells of a plant.
etiology	study of causes of disease.
foot rot	a disease of the stem bases and associated leaf tissues.

glume	chaff enclosing spikelet or grain.
hypha	filament (thread-like) of a fungus.
imperfect state	asexual spore form of fungus (anamorph).
inoculum	fungus for inoculating a plant.
internode	part of stem between two successive nodes.
latent	an infection with no obvious symptoms at time of inspection but may show later.
lesion	dead or damaged area of tissue.
macroconidium	larger conidium.
mesocotyl	part where stem (coleoptile) and root tissues merge, adjacent to seed, in germinating cereal seedling.
microconidium	smaller conidium.
MBC fungicides	methyl benzimidazole carbamates, include benomyl, carbendazim, fuberidazole, thiabendazole, thiophanate-methyl.
mycotoxin	poison produced by a fungus on a foodstuff for man or animal.
node	part of stem where a leaf arises; slightly swollen in cereals.
ostiole	pore in a fungal fruiting body (e.g. perithecium, pycnidium) through which spores are released.
perfect state	sexual spore form of fungus (teleomorph).

perithecium	rounded structure containing asci and ascospores.
rachis	main axis of the inflorescence, the ear in cereals.
snow mould	a disease of cereal plants, mainly under snow, caused by <u>Fusarium nivale</u> .
snow rot	a disease of cereal plants, mainly under snow, caused by <u>Typhula incarnata</u> .
spikelet	a unit of the ear within two glumes, consisting of one or a few flowers and, subsequently, grain(s).
teleomorph	sexual spore form of a fungus (perfect state).
toxigenic	capable of producing or inducing (myco)toxin.
triazole	a group of fungicides including flutriafol, propiconazole, triadimefon, triadimenol.
whitehead	premature death of cereal plant after ear emergence, shows as a bleached plant in contrast to green healthy plants.

INTRODUCTION

Several species of fungi grouped together in the genus Fusarium affect cereals causing diseases and the production of toxins (mycotoxins) which are injurious to man and animals. The aim of this review is to provide a summary of the information available on both subjects. The article also defines areas where information is lacking and makes recommendations for further work.

The review is limited to information from the UK though information from other countries has been included where it is relevant. In addition a short section is devoted to Fusarium diseases in some other countries.

Information for the review has been obtained from publications (see the lists of references) and from conversations and correspondence with scientists in the UK and overseas (see acknowledgements). In addition, abstracts on Fusarium subjects in the most recent 20 years of the Review of Plant Pathology have been read, mainly for information from other countries.

Although most of the fungi which cause disease are also involved in the production of mycotoxins, the two subjects have little else in common and they have therefore been reviewed in two separate sections.

The six species of Fusarium associated with diseases have some similar features and cause some similar symptoms, but it is a mistake to regard "Fusarium" as a single entity. The pathogens and the diseases they cause differ in several important respects. In particular, F. nivale and the diseases it causes are significantly different from the others. Thus the commonly recorded "Fusarium sp." as a cause of a disease is not helpful in understanding the nature and cause of that disease.

Despite the specific nature of most of the diseases it has been decided that in this review it would be most appropriate to discuss the diseases, and their causal pathogens, as they affect various stages of the crop - seedling, mature plant and ears. This is then followed with a more general account of other aspects - spread, incidence, severity and crop losses, control measures and a short note on the diseases in other countries.

THE FUSARIA - A NOTE ON NOMENCLATURE

The naming of fungi is governed by a code. Each species has a binominal name - the genus (e.g. Fusarium) and a specific epithet (e.g. culmorum). A fungus may have two names - one for the state bearing the asexual spores (conidial state in Fusarium) which is also called the imperfect state, the anamorph, and one for the state bearing the sexual spores (perithecial state in Fusarium) which is also called the perfect state, the teleomorph.

The perfect name (teleomorph) takes precedence over the imperfect (anamorph) and often is the only name used even when the imperfect state is common. In the case of Fusarium, only some species are known to have a perfect state but all are known and familiar by the name of the imperfect state. A further complication arises in the case of Fusarium nivale which, it is generally agreed, is an anomalous member of the genus Fusarium. It has now been transferred to the genus Microdochium; the perfect state has been named, after many changes, as Monographella nivalis (Samuels & Hallett, 1983).

However, although aware of the above comments, for the sake of convenience and clarity the name Fusarium has been retained in this review for all the species discussed.

The following is a list of names of Fusarium species causing diseases in cereals:

Imperfect (conidial) state
(anamorph)

Fusarium avenaceum (Corda ex Fr)
Sacc.

Fusarium culmorum (W.G. Smith)
Sacc.

Fusarium graminearum Schwabe

Fusarium crookwellense Burgess,
Nelson & Toussoun

Fusarium poae (Peck) Wollenweber

Microdochium nivale Samuels &
Hallett
= Gerlachia nivalis (Ces.)
Gams & Muller
= Fusarium nivale (Fr.) Ces.

Perfect (perithecial) state
(teleomorph)

Gibberella avenacea Cook.
(not recorded in UK)

not known

Gibberella zeae (Schw.) Petch.

not known

not known

Monographella nivalis (Schaffnit)
Muller

= Micronectriella nivalis
(Schaffnit) Booth
= Calonectria nivalis Schaffnit
= Griphosphaeria nivalis (Schaffnit)
Muller & Arx
= Calonectria graminicola Wollenw.

Other species recorded on cereals, but regarded as saprophytes or occasionally weak parasites, include Fusarium equiseti, F. oxysporum, F. sambucinum, F. solani, and F. tricinctum.

F. nivale is sometimes said to occur as two varieties - F. nivale var. nivale and a more virulent F. nivale var. majus.

Most of the species named are also involved in the production of mycotoxins but more complete lists are referred to in the section on mycotoxins.

F. avenaceum, F. culmorum, F. graminearum and F. nivale are well established as pathogens of cereals. F. poae is not regarded as a pathogen except on the ears but it is common on cereals and is important for its production of mycotoxins.

F. crookwellense has been identified only recently (Burgess, Nelson & Toussoun, 1982) and it has been recorded rarely in the UK. It is included here to draw attention to the possibility that it has been wrongly identified as F. avenaceum or F. culmorum in the past.

Species identification in Fusarium has presented problems. The first comprehensive classification by Wollenweber described 142 species, varieties and forms and was difficult to use. Subsequently attempts have been made to simplify and clarify the situation with, it is claimed, some success (Nelson, Toussoun & Marasas, 1983). A list of books and papers on Fusarium is in Appendix 1.

THE DISEASES

A note on symptoms and disease cycles

Fusarium culmorum can survive in soil as long-lived spores (chlamydospores), in debris and in organic matter. It is also seed-borne. The fungus can cause death of seedlings pre- and post-emergence. Seedlings which survive may have stem lesions. Later, at the stem base, brown lesions affecting the internode and the node may be noticed. These are usually not severe but very occasionally they cause a stem rot severe enough to cause a whitehead. Such affected stems usually are rotted at one of the lowest nodes where they break easily. More usually the lowest internodes are discoloured and occasionally may develop a soft rot. The effect of this form of the disease on crop yield is not known but is assumed to be not usually serious. F. culmorum does not normally cause a leaf disease. However, it can cause severe ear symptoms called ear blight. Affected florets or spikelets assume a bleached appearance, very obvious when the ear is green, and usually bear the pink growths of the fungus. Sometimes the rachis is attacked and then all parts of the ear above this point are bleached. Infections occur at or soon after flowering and no grain is formed in the affected spikelets. Later attacks cause grain shrivelling and can also infect the grain directly. Seed-borne infections can be partially controlled by seed

treatments and sufficiently well to prevent severe seedling losses. Spread of the disease up the plants and on to ears is assumed to be through rain-splashed or airborne spores (conidia) produced on the base of the plant. The development of disease on the above ground parts, and especially in the ear, is favoured by wet weather.

F. avenaceum causes diseases very similar to those caused by F. culmorum and the disease cycle is the same except that F. avenaceum does not form long lived chlamydospores in the soil. It is regarded as less virulent than F. culmorum.

F. graminearum also causes similar diseases but with a few marked differences. In the ear blight phase, superficial fungal growth is more evident and purple-black perithecia (Gibberella zeae) are embedded in it. The disease is referred to as "scab". Affected grains may be shrivelled and discoloured red. The disease is serious in some of the major wheat growing areas of the world when warm humid conditions prevail and especially when there are prolonged wet periods at harvest. Infected grains are a major source of mycotoxins. The disease is uncommon in the UK but it has caused isolated outbreaks (two severe epidemics in Eire) of severe seedling disease and later in the season foot rots causing a sudden death of mature plants. On such plants perithecia were found grouped together on diseased stem bases. Spread is assumed to occur by conidia as in the case of F. culmorum but perithecia produced on ears and stems may initiate infections in autumn and spring. The fungus sometimes forms chlamydospores in soil but they are said not to be long-lived.

F. nivale is favoured by much cooler temperatures than the three species just described. It does not form chlamydospores in soil but survives in debris and organic matter. It causes the disease snow mould under snow, mainly in Scotland on barley. Snow mould causes a leaf disease and may kill crowns but it is less important than snow rot (caused by Typhula incarnata) with which it may occur. F. nivale is commonly seen on leaf sheaths and can cause a leaf spot disease. It causes a discolouration of the lower nodes and can affect the culm

though probably it does not cause severe foot rot. It can cause an ear blight similar to that described for the other Fusarium species but it also causes less severe symptoms such as a discolouration of the glumes. F. nivale is seed-borne and is the most common cause of pre- and post-emergence seedling disease. The disease may be spread by conidia as with other Fusarium diseases but ascospores from the perithecia (the Monographella nivalis state) formed in large numbers on leaf sheaths are assumed to play a major part in the spread of disease from the lower to the upper parts of the plant and perhaps further afield.

The only disease attributed to F. poae is a lesion on the glumes. The fungus is commonly recorded on grain where, although it causes no loss of yield, it is an important source of mycotoxins.

An historical perspective

The earliest record of Fusarium diseases of cereals in the UK appears to be that by W.G. Smith (1884) in his book "Diseases of Field and Garden Crops". Norfolk farmers complained of a disease attacking the ears and the fungus Fusarium culmorum was identified as the cause. Whole parts of the ear were affected to give a "spurious appearance of ripeness". The disease was probably seasonal and there are no other reports for many years. In Scandinavia and on the Continent, ear blight and snow mould caused by F. nivale were well described (Eriksson, 1912). In 1920 the Ministry of Agriculture's mycologist was quoted (Bennett, 1928) as saying that a Fusarium foot rot was common in cereals but had not been investigated. After 1919 reports on crop diseases were compiled annually and published occasionally (Cotton, 1921, 1922; Pethybridge, 1926, 1929, 1934; Moore, 1947, 1948, 1959; Baker, 1972). There are references to foot rots ("brown foot rot") in most years, and to seedling losses and ear blight in some years. Moore (1959) summed up the period to 1957 by saying that brown foot rot and ear blights (associated mainly with F. avenaceum and F. culmorum) were widely distributed in poorly-drained acid soils but were rarely serious, except in the north of England and Scotland. The

reference to the north of England may be related to the fact that most of the investigational work on the diseases was done there. In the 1920's F.T. Bennett investigated the problem of "deaf-ears" (whiteheads) and "thinning out" (due to the falling over of tillers before harvest). Bennett (1928) associated these symptoms with a foot rot caused by F. avenaceum and F. culmorum and showed that these fungi would also cause poor germination, a seedling blight and ear blight. He also recorded F. graminearum and its perithecial state, Gibberella zeae, for the first time in the UK (Bennett, 1930, 1931, 1933b), investigated diseases caused by F. nivale (Bennett, 1933a) and recorded other Fusarium species, mainly not important parasites, on cereals (Bennett, 1935). Eyespot (caused by the fungus Pseudocercospora herpotrichoides) was not recognised at that time (it was first recorded in the UK in 1935) and it seems likely that some of the field symptoms of foot rot and consequent whiteheads and crop thinning recorded by Bennett may have been caused partially or mainly by eyespot. Nevertheless, the description of some field symptoms (e.g. the nodal rot associated with isolated or small groups of plants) and the fact that the diseases were worst in wheat crops grown every fourth year (which should exclude serious eyespot) suggests that some of his observations and conclusions are valid.

Other reports of Fusarium diseases were based on casual observations in crops except for a two-year survey in Cambridgeshire (Russell, 1932) which showed that seedling blights and ear blights were uncommon and that F. culmorum was the most important cause of foot rots.

Further substantial work on Fusarium diseases was not done until the 1960's when J. Colhoun and his co-workers at Manchester carried out a number of comprehensive investigations, mainly on diseases associated with seedlings (Colhoun, 1970, 1972).

The last crop disease summary covered the period 1957-1968 (Baker, 1972). Ear blights were a feature of the wet seasons and, following a wet harvest, trouble with seedling blights was recorded, though only

if the seed was untreated or treated with an ineffective fungicide. Brown foot rots (mainly associated with F. avenaceum and F. culmorum, sometimes F. nivale) were considered to be of little consequence, except in 1961 when they were widespread and sometimes associated with whiteheads scattered through crops. This high incidence was associated with exceptionally wet soils which persisted into the spring. F. graminearum, usually recognised by its perithecial state Gibberella zeae, was recorded only rarely but was associated with severe outbreaks on oats in mid-Wales in 1942 and in the same year epidemic levels were recorded on wheat in Eire (McKay, 1957). Although F. nivale was recorded in 1933 it was not reported often until Noble & Montgomerie (1956) showed it was common in crops, as well as on seed, in oats in Scotland and Colhoun, Taylor & Millar (1963) reported it to be the most common Fusarium on stem bases in England.

Since 1968 there have been no published crop disease reports and during this period there have been significant changes in methods of growing cereal crops. No significant developments in the seed-borne and seedling aspects of Fusarium diseases have been reported though F. nivale (recognised as the most damaging seed-borne Fusarium) is now regarded as a frequent (previously infrequent) if still erratic seed-borne hazard (Hewett, 1983). Severe disease from this source is still prevented by seed treatments.

Ear blight has attracted more attention in recent years especially after a minor epidemic in some parts of England in 1982. Most attention has been centred on the foot rot phase of the disease. Severe foot rot causing whiteheads has not been reported but ADAS surveys have disclosed a fairly high incidence of Fusarium symptoms on stem bases though most have appeared to be slight and unlikely to be very damaging. There have been several reports of brown foot rot and a weakening of the stem which are said to be associated with premature senescence or lodging. These reports are anecdotal and there appears to be no clear evidence to demonstrate a consistent relationship between foot rots caused by Fusarium spp. and a loss of grain yield.

Seedling diseases

Symptoms and conditions affecting infection

Fusarium avenaceum, F. culmorum, F. graminearum and F. nivale can all cause pre-emergence and post-emergence damage to cereal seedlings. The symptoms which can range from death soon after germination to superficial stem lesions on the emerged plants, are similar for all the pathogens and usually the cause can be established only by isolating and identifying the pathogen.

Fusarium poae is not regarded by Colhoun & Park (1964) as a pathogen of seedlings. Fusarium crookwellense which has been recognised only recently is reported to have caused damage to seedlings in Poland but further studies will be needed to establish its importance in the UK where its identity in the past may have been confused with F. avenaceum and, more particularly, F. culmorum.

Bennett (1928) regarded F. culmorum and F. avenaceum as the predominant pathogens with the former causing the more severe damage and with wheat more severely affected than barley.

Colhoun & Park (1964) found that F. culmorum and F. graminearum caused pre-emergence death of the coleoptile, often just before emergence. Pre-emergence and post-emergence death was most severe in dry soils and increased with increasing temperature (in the range 8-23°C). These marked effects of soil moisture and temperature were not observed with F. avenaceum which they regarded as a much less virulent pathogen. All three species can cause brown lesions on stems without killing them, but under favourable conditions both F. culmorum and F. graminearum caused post-emergence death about 14 days after sowing.

Colhoun, Taylor & Tomlinson (1968) stress the importance of interactions between certain factors to cause seedling diseases. The most influential factors are soil moisture, temperature and (especially) the level of seed inoculum (spore load). For F. culmorum

on wheat there was little pre- or post-emergence death or stem lesion development at any temperature or soil moisture until the spore load reached fairly high levels (e.g. at least 10^5 spores/25 g seed). Although disease was favoured by higher temperatures (up to 23°C), in dry soils high spore load compensated to some extent for lower temperatures (e.g. 8°C) to give severe disease. In wet soils which were also cold there was very little pre- or post-emergence damage even at high seed spore loads. When wet soils were combined with high temperatures then high spore load did result in some increase in stem lesions.

In experiments with barley results were similar except that dry soils and high spore load did not compensate for low temperatures to cause high levels of pre-emergence death. Also the development of lesions was less influenced by soil moisture than in wheat.

F. avenaceum was a much weaker pathogen than F. culmorum although influenced by soil conditions in much the same way. Lesions developed on stems under a range of conditions, but only under favourable conditions of soil moisture and temperature and a much higher seed spore load than for F. culmorum were seedlings killed.

F. nivale caused seedling diseases similar to those just described but with several important differences. Infected seed could result in severe pre-emergence death (up to 80 per cent) often soon after the germination of the seed. Later symptoms included a browning of the coleoptile which may be deformed and (in contrast to the other Fusarium spp.) the first leaf could be attacked so that it lay on the soil surface. It often had a shredded appearance. The first and second leaves could also bear lens-shaped pale brown or auburn lesions (Noble & Montgomerie, 1956; Millar & Colhoun, 1969a). Disease was favoured by dry soils and lower temperatures, e.g. in dry soils there was more disease at 6.1°C than at other temperatures up to 16.4°C . At all soil temperatures the amount and severity of disease was reduced as soil moisture increased. There was more disease at higher seed spore loads but a very low spore load gave considerable disease when

the soil was dry. There was also more disease with increasing sowing depth (Millar & Colhoun, 1969b). Noble & Montgomerie (1956) also found deep sowing, especially on heavy soils, resulted in more attenuated coleoptiles and more pre-emergence death of oat seedlings.

The marked effect of soil moisture (dry soils favouring disease) may be explained by an escape of severe infection in the wetter soils by seedlings growing more rapidly. Shen (1940) showed that germinating seed prior to sowing reduced disease severity. Malalasekera & Colhoun (1968) showed that soaking seed for three hours prior to inoculation or prior to sowing in inoculated soil, reduced disease compared with seeds not soaked or soaked for more than 12 hours. The three-hour soaking increased seedling vigour and the seedlings escaped infection and especially pre-emergence death. Seedlings from unsoaked seed or seed soaked for a long period were exposed to attack for a longer period and hence were more diseased.

Soil reaction (pH) appears to have only a small effect, if any, on seedling diseases and is much less important than seed spore load, soil moisture and temperature (Bennett, 1933; Millar & Colhoun, 1969b).

Rawlinson (1971) found that several Fusarium species were more aggressive to oat seedlings when environmental conditions were adverse, e.g. freezing, though not through the invasion of tissues damaged by frost. Perry (1986) also noted that F. nivale was more pathogenic to spring barley when there were short periods of freezing before the seedlings emerged.

Invasion of seedlings

F. culmorum and F. nivale were similar in the way in which they penetrated and invaded wheat seedlings. Germinating seeds from inoculated seed were quickly attacked, the pathogen being first detected in the coleorhiza and parts of the roots adjacent to it. The apparent entry was where the primary root burst through the

coleorhiza. Both fungi penetrated stomata in the coleoptile. F. culmorum also penetrated between epidermal cells whilst F. nivale grew through cracks in the cuticle. Although roots were invaded, neither species spread downwards in the roots. When seedlings from healthy seeds were grown in inoculated soil, segments of root distant from the coleorhiza were infected but again the fungi did not spread rapidly in the roots (Malalasekera, Sanderson & Colhoun, 1973).

Rawlinson (1971) and Perry (1986) noted earliest attacks in the region of the mesocotyl (the part of the coleoptile nearest the seed). Perry suggested that the fungus preferentially invades that part of the germinating seed where sugars and other nutrients, which diffuse from the endosperm through the scutellum, provide an energy-rich medium. Perry also frequently isolated F. nivale from apparently healthy coleoptiles after inoculation, indicating that the fungus had invaded tissues without causing symptoms. He suggested that the relationship might be described as endophytic (no symptoms) in contrast to latent (symptoms produced later) and that the fungus may persist from crop to crop by this means as well as in soil debris.

Although a root rot is often included as one of the symptoms of Fusarium diseases, none of the descriptions of infection at the seedling stage includes a severe form of root rot. Millar & Colhoun (1969a) suggest that general references to a brown root rot symptom in young seedlings may be derived from observations during seed testing; on wheat plants from field crops root browning was not a conspicuous symptom. However, McKay (1957) describes a severe root rot in wheat and oat seedlings caused by F. graminearum and Cook (1980) says that a crown rot is caused by F. culmorum gaining entry through newly emerging crown roots.

Sources of Fusarium diseases: Soil

All the pathogens which cause seedling disease can survive in the soil though the manner of survival may differ. Snyder & Nash (1968) during one season at Rothamsted, compared the saprophytic and

parasitic Fusarium spp. in fields where only cereals or only roots had been grown for about 100 years or more and also in long-term pasture and woodland. From diseased plants in cereal crops they isolated mainly F. culmorum and F. nivale, F. avenaceum often, F. tricinctum (probably = F. poae) occasionally but not F. graminearum. Isolations from soil (which excluded debris and organic matter) showed that F. culmorum could not be recovered where only roots had been grown or from woodland but was present in soils from permanent pasture and was the dominant pathogen from cereal soils. The largest populations were found in plots receiving complete fertilizer (including high nitrogen) and dung and the lowest in plots not fertilized or where N, P, K were applied alone (i.e. high populations in the plots where good crop growth had been maintained). F. avenaceum and F. tricinctum (= F. poae) were only rarely isolated from soil though F. avenaceum was more common in the surface layers of uncultivated woodland soils. F. nivale was not recovered from any soils even though, in the case of the cereal fields, it was common on the above ground parts of the plants.

These findings reflect the fact that only F. culmorum readily forms chlamydospores, thick-walled spores which can survive in soil. F. graminearum which is not very common in cereals in the UK can also form chlamydospores but these are short lived (Cook, 1981). F. avenaceum rarely produces true chlamydospores though it does form a type of resting spore within macroconidia and hyphae which may enable the fungus to survive in soil for at least 18 months (Hargreaves & Fox, 1977, 1978).

All of the Fusarium pathogens can survive in plant debris and residues. F. culmorum is an aggressive saprophyte of straw in which it can survive in soil (Sadasivan, 1939) and on which it forms masses of macroconidia and the chlamydospores which also survive in soils. However, in observations by Booth & Taylor (1976b) it did not spread through soil by mycelial growth.

F. nivale, although not isolated directly from soils, can be detected by growing bait plants and isolating from roots and mesocotyls even though there are no obvious symptoms (Rawlinson & Colhoun, 1969). The fungus was isolated in this way from soil which had not grown cereals for at least 15 years. It was suggested that the fungus had been introduced on infected straw in farmyard manure or had survived on roots of other plants such as grasses. Snyder & Nash (1968) recorded F. nivale in the field on wheat straw from the previous season. Booth & Taylor (1976a) investigating straw as a source of inoculum found most seedling disease incidence where straw had been ploughed in (97 per cent seedlings affected) and least incidence (47 per cent) where it had been removed, with 70 per cent incidence where straw had been left standing. No seedling death was recorded except where very high levels of inoculum were added. Booth & Taylor (1976b) regarded F. nivale as a saprophyte capable of colonising straw in soil though not as aggressively as F. culmorum and not as a competitor when other organisms were established in the straw. Al-Hashimi & Perry (1986a) found that F. nivale did not survive in soil in inoculated straw nor did it colonise sterilised straws. The fungus had no significant cellulytic ability, a characteristic of fungi which survive well as saprophytes in straw. They concluded that F. nivale is a weak saprophytic competitor and is only likely to survive in straw which has been colonised in the parasitic phase. F. nivale is inhibited in some soils so that it causes little or no disease. This has been associated with the presence of other fungi such as Gliocladium roseum (Millar & Colhoun, 1969b) and Trichoderma viride (Al-Hashimi & Perry, 1986b). Mycelial growth of F. nivale from a nutrient base (agar, seed, straw) has been recorded in both sterile and non-sterile soils. This spread (up to 95 mm) could initiate disease in seedlings provided they grew through soil which had been invaded. There was no infection when mycelial spread occurred after seedling emergence (Booth & Taylor, 1976b). Al-Hashimi & Perry (1986a) also noted mycelial growth in the same field soils in which F. nivale had failed to colonise straw, i.e. antagonists which prevented colonisation did not prevent active growth from a food base. Once the mycelium was deprived of a food base (e.g. an infected seedling), lysis was rapid and infectivity to bait plants was lost in eight weeks.

Al-Hashimi & Perry (1986a) also found that when conidia of F. nivale were added to the soil, infectivity to bait plants persisted longer than was expected. This was unexpected because conidia are assumed to be short-lived and there is no evidence that F. nivale produces more persistent propagules such as chlamydospores.

Severe seedling disease is associated with high levels of inoculum and this is more likely to be associated with a seed source than a soil source. High levels of inoculum on seed, however, occur erratically and it seems likely that soil-borne sources provide a more consistent source of the pathogens to initiate diseases in seedlings and subsequently on other parts of the older plant. The severity of these diseases is then more related to other factors (host, environment) than to the source.

Sources of Fusarium diseases: Seed

Surveys have shown that F. nivale is one of the most common of the Fusarium seed-borne diseases. (Hewett, 1983; Rennie, Richardson & Noble, 1983). However, the incidence is low (usually less than one per cent) except for some isolated samples and in seasons when the summer weather, particularly at harvest, is wet. Even in wet seasons the incidence of F. nivale in a sample rarely exceeds 30 per cent and this may be due to the inhibitory effects of some components of the microflora on the ear. For example, Alternaria alternata, the most abundant seed-borne fungus in crops, is an inhibitor of F. nivale (Bateman, 1979).

F. avenaceum and F. culmorum are usually at a low incidence except in wet seasons and especially when seed is harvested from lodged crops (Hewett, 1966). Malalasekera & Colhoun (1969) pointed out that severe seedling disease is associated with high spore load and this is not measured by most seed testing methods which only indicate presence or absence. They describe a test which is more quantitative and can therefore provide a better forecast of the performance of a seed sample.

Severe seedling disease occurs most commonly in winter wheat when untreated seed is sown. Because F. nivale is fairly commonly seed-borne and can attack young seedlings at the prevailing low soil temperature, it is the most likely cause of the disease. Winter barley and spring-sown cereals are less affected.

Seed-borne Fusarium diseases are controlled by seed treatments. Organomercury does not control deep-seated infections (Bateman, 1983) but the partial control obtained is sufficient to prevent significant seedling losses (Hewett, 1983; Richardson, 1974). However, such control does not prevent diseases becoming established in the crop nor does it influence the subsequent development of disease on leaves, stems, ears and seed. Some of the more modern fungicides gave a better control of seed-borne diseases but they contain an MBC-fungicide specifically to improve the control of seed-borne Fusaria. The current widespread occurrence of resistance to MBC in F. nivale (Locke, Moon & Evans, 1987) will reduce the effectiveness of these fungicides.

More information on seed-borne diseases is included in the sections 'Incidence and severity' and 'Control'.

Foot rots, stem and leaf diseases

Snow mould caused by Fusarium nivale is widespread and often damaging in areas where crops are covered with snow during winter (e.g. northern continental Europe and Scandinavia). Considerable spread occurs under cover of melting snow when humidity is 90-100 per cent and air temperatures are above freezing, particularly in years with heavy snow cover and slow melting in the spring. Leaves and, in severe attacks, the crowns are killed; diseased tissues are usually covered by grey or pink coloured mycelium. The disease is common on turf grass in the UK but was virtually unrecorded on cereals until relatively recently when it has been found, mainly in Scotland, on winter wheat and much more commonly on winter barley. The disease usually occurs in barley together with snow rot (caused by the fungus Typhula incarnata) which is regarded as much more important. In the north of Scotland the diseases tend to occur separately when snow mould usually causes limited damage in small (30 cms) patches (J. Gilmour, S.J. Wale, personal communication).

Plants infected with F. nivale but not affected by the snow mould symptoms usually exhibit in spring a brown decay of the lowest leaf sheaths and sometimes a shredded first leaf blade lies on the soil surface. Fungal hyphae and soil particles may be associated with the diseased tissues. The oldest two leaves may bear buff or auburn lens-shaped spots. Later in wheat, usually after stem elongation, perithecia appear in brown areas on the lower leaf sheaths and then extend, with the browning, all over the sheaths and sometimes on to the adjacent parts of the leaf blade (though the leaf blade remains largely unaffected). Usually under the diseased leaf sheaths the culm (stem) remains healthy. Symptoms are usually associated at first with the bottom three nodes but subsequently there may be spread by ascospores and conidia to leaf sheaths higher up the plant and to ears. The perithecia on the sheaths are black, very numerous and in rows with the ostioles (openings) corresponding to the stomata of the leaf sheaths. They are formed only on those parts of the sheath which are exposed, i.e. they are absent on the covered part where the sheath overlaps itself. The disease spreads from the sheath to the nodes and at the junction there is a characteristic purple-brown colour. The fungus can be isolated readily from the affected nodes but, in the early stages at least, not from the internodes (Millar & Colhoun, 1969a). Noble & Montgomerie (1956) recorded that, in oats, perithecia were formed as early as the five to six leaf stage and that the ascospores they contained ripened and were shed as the stem elongated.

Later in the season, usually after anthesis, brown or black streaks can occur on the lowest internodes and sometimes merge to affect most of the internode. Other symptoms include a pale brown water-soaked appearance of the lowest internode, immediately above the crown roots. These symptoms are grouped and usually ill-defined as 'foot rots', 'brown foot rot' or 'stem base diseases'.

Rarely there is a severe stem rot which causes the premature death of the tiller and shows as a whitehead occurring at random in an otherwise green crop. The stem breaks off easily at a rotted node which usually bears the pink mould of the fungus.

F. nivale can be isolated from the brown and black streaks and sometimes is associated with the other symptoms but there does not appear to be any evidence that F. nivale causes a severe stem rot and Fehrman (1988b) states that, in contrast to other foot-rot pathogens, it does not cause lodging.

Other Fusarium species can be isolated from the stem bases but there appears to be no good description of foot rots caused by F. culmorum and F. avenaceum as they occur in the UK or in other parts of Europe. The best descriptions are by Bennett (1928) but his observations have to be treated with caution because eyespot was not recognised at the time and may have been involved in the disease he studied. The stem-base infections by F. culmorum are said to originate through an infection of the roots and the disease then develops under warm dry conditions though symptoms are rarely seen until after flowering (Fehrman, 1988a). Cassini (1981) says that F. culmorum and F. graminearum are important in France as causes of ear blight but they also cause a foot-rot similar to that described by Cook in the Pacific Northwest (USA). The foot rot is held to be responsible for "scald", a premature death of plants following a period of hot dry weather just before the crop matures. No evidence is offered and it is acknowledged that "this point of view is not often shared".

The most comprehensive investigation of a foot rot disease is that carried out by Cook (1980, 1981) in the Pacific Northwest where F. culmorum causes a severe disease. The primary sources are soil-borne chlamydospores and infected plant debris. The fungus enters the plant 2-3 cms below soil level through openings around emerging roots or by infecting newly emerged crown roots. The fungus grows into the crown and remains latent unless the plant is subject to water stress in which case the fungus then progresses one to three internodes up the culm during or shortly after heading. The diseased internodes become chocolate brown though they can be seen only by removing the healthy-appearing sheaths enclosing them. The diseased crowns are spongy and brown with abundant mycelium, often pink or burgundy coloured, in the stem cavities. The wheat grows in an area of

Mediterranean-like climate where rainfall is negligible from heading onwards. The crop matures on water stored in the soil to depths of 180 cm and plants develop water potentials (a measure of water stress) of -50 bars. Severe foot rot is initiated when plant water potentials are about -32 to -35 bars at the beginning of heading. Plants with water potentials between -15 and -25 bars (considered typical of wheat with ample soil moisture) may become infected but do not develop severe foot rots. The critical nature of the water stress has been emphasised by Cook (1980) who pointed out that it proved impossible to reproduce the conditions necessary to cause symptoms in plants away from the field (e.g. in large pots) and this restricted research on the problem.

This description of severe foot rot in the Pacific Northwest is necessary so that it can be compared with symptoms of foot rots in the UK and elsewhere in Europe. Clearly the water stress conditions described are unlikely to occur under normal conditions on good wheat soils in northern Europe. However, it has been suggested by Cassini (1981) that a similar foot rot could occur under certain conditions in Europe. A key factor in identifying the cause of the death of the plants is the distribution of affected plants in the crop. A distinction should be drawn (R.J. Cook, personal communication) between plants which are killed by lack of water and occur in patches associated with some adverse soil condition, and a pattern of scattered bleached dead plants, amongst unaffected plants, which occurs when they are killed by Fusarium. In the UK and elsewhere in northern Europe scattered whiteheads associated with Fusarium foot rot are rarely recorded (one of the authors has noticed it only once in twenty years). It is therefore very unlikely that the disease described in the Pacific Northwest occurs commonly in northern Europe.

Premature death of cereals, including a bleached appearance is not uncommon in the UK, especially in barley, but usually occurs in small or large patches and can often be related to adverse soil conditions (light soils, poor soil structure etc) which restrict water supply to

the plant. Fusarium spp. may also occur on stem bases in such areas but the symptoms are not the same as those described by Cook. Colhoun (1970 and personal communication) also states that the disease described by Cook is not common in the UK.

Colhoun (1970) noted that seedlings with lesions caused by F. culmorum or F. nivale did not develop foot rots later unless the air humidity at the base of the plant was high (irrespective of whether the soil was wet or dry). When the humidity around the stem bases was maintained at a high level by artificial means for four weeks or more a severe foot rot developed. There is, however, no evidence that such environmental conditions are similar to those which could occur in the field.

F. graminearum is only rarely reported causing severe disease but in one report of the disease in oats, initiated by infected seed, (Richardson, 1970), a severe foot rot and root rot caused a whiteheads symptom in plants distributed at random among normal green plants in the crop during August. Affected plants could be lifted from the ground with little effort and came away without roots. The bases of the stems were covered by dark purple-black perithecia.

Cereal foot rots often occur as a complex of several diseases but there is very little published work on the interaction of the pathogens involved. Hoare (1987) in greenhouse tests found that F. culmorum in combination with Gaeumannomyces graminis (the take-all fungus) caused significantly more damage than G. graminis alone. However, in combination with Pseudocercospora herpotrichoides (eyespot) and Rhizoctonia cerealis (sharp eyespot), F. culmorum did not cause any additional damage. Parry (1987) reported that when field plots of winter wheat were inoculated with F. culmorum and P. herpotrichoides singly and in combination, the proportion of plants infected with F. culmorum increased from 60% in plots inoculated with F. culmorum alone to 90% in plots inoculated with both fungi, i.e. infection by F. culmorum was enhanced by the presence of the eyespot fungus. Furthermore the plants affected by both diseases had the most

severe symptoms and were probably the most likely to lodge. In contrast, some work in West Germany indicated that as the eyespot disease indices decreased there was a slight increase in Fusarium diseases (Fehrmann & Duben, 1980).

Periodically eyespot lesions are observed to have a pink or orange colour and this has been associated with the presence of Fusarium sp. and in fact F. culmorum has been isolated from such lesions (J.D.S. Clarkson, personal communication). However, at the Plant Breeding International (formerly Plant Breeding Institute), Cambridge a comprehensive investigation came to the conclusion that the lesions were caused by the eyespot fungus only, though there was no explanation for the colour. (T.W. Hollins, personal communication).

Some isolated cases of other forms of stem disease have been reported. Hunter, Best & Jordan (1984) reported whiteheads in one crop of winter wheat due to a rot of the terminal (top) node caused by Fusarium avenaceum. The disease showed also as dark brown lesions immediately above and girdling the flag leaf node so that the upper stem portions were easily detached.

Recent observations indicate that some Fusarium spp. may move and/or grow systemically (internally) in stem tissues. Jordan & Fielding (1988) inoculated spring wheat just below soil level with F. avenaceum, F. culmorum, F. graminearum and F. nivale, singly and in combinations. F. culmorum was isolated from all internodes and some ears and it was concluded that the fungus had progressed internally. With the other species there was less internal colonisation of stem tissues.

Systemic infection by F. culmorum has also been investigated in the Netherlands (C.H.A. Snijders, personal communication). Inoculated plants grown in nutrient perlite (a non-soil medium) produced a foot rot symptom consisting of mycelial growth and brown longitudinal lesions at the base of the culm. At flowering F. culmorum was detected (on agar plates) up as far as the fifth internode,

i.e. towards the top of the stem. It is suggested that such systemic infections might be an important way of infecting sheaths and ears and, at least, facilitate infection by water splash. In maize, where Fusarium spp. cause severe stem rots, systemic infections high in the stem have been recorded in the UK (R.J. Cook, ADAS, personal communication).

Much earlier Bennett (1928) examined the height to which F. culmorum and/or F. avenaceum extended in stems of plants grown in pots or in the field. From 66 plants he isolated pathogens 44 times from the internode immediately above soil level, 15 times from the second internode but not once above this. He concluded that neither fungus grew internally up the stems into the ears. Colhoun & Park (1964) frequently isolated F. culmorum and F. nivale from discoloured nodes of wheat and barley and up to the fifth node, but not from internodes. Duben & Fehrmann (1980) observed that F. avenaceum colonised the whole of the plant, including artificially inoculated plants and field-grown plants, during a very wet summer. They concluded that there was no convincing evidence for systemic growth within the plant and that experience suggested this was not important. They suggest that field infections followed an attack by barley yellow dwarf virus.

In addition to attacking leaves as snow mould, F. nivale can also affect leaves higher up the plant. The lesions on the upper leaf blades are often large and are greyish green and water soaked, though later they become brown, with the fungus usually sporulating abundantly through the stomata. The disease was first recorded on wheat in Scotland where similar lesions on barley leaves were also observed (Richardson & Zillinsky, 1972). The disease has been noticed more commonly in recent years, mainly on wheat, in all parts of the UK. This phase of the disease is usually unimportant though some triticale varieties are sometimes fairly severely affected. This leaf disease has become prominent in Germany, The Netherlands and Switzerland in the 1980's (H. Fehrmann, personal communication). Both F. nivale and F. culmorum can enter leaves through lesions caused by powdery mildew (Erysiphe graminis) (Forrer, Rijsdijk & Zadoks, 1982; Mathis & Forrer, 1986) though the incidence and severity of such attacks is not significant.

Ear blight and grain diseases

Fusarium avenaceum, F. culmorum, F. graminearum, F. poae and F. nivale occur on the ears of cereals and produce similar symptoms which are most common on wheat. Infections may occur at any time from ear emergence to maturity but ears are most susceptible, and most damage is done, in the flowering period. The earliest signs of infection in wheat are small brown water-soaked spots on the outer glumes. The infection may remain limited to the glume but under favourable conditions affects a floret or more usually the whole spikelet. The affected tissues take on a bleached appearance in marked contrast to the green healthy spikelets. The glumes of an infected spikelet can become bound together by the growth of the fungus within the spikelet, and a pink mass of sporulating fungus is often seen at the base of the glumes. Under moist conditions the affected spikelet can be covered by a weft of white or pink mycelium. The disease may spread to adjacent spikelets but the most severe effect is when the pathogen grows into the central axis of the ear, the rachis, and cuts off supplies of water and food materials to the parts above, which also soon become bleached. Affected ears take on the appearance of premature ripening though later they become darker in colour than the healthy ears due to the growth of saprophytic moulds on them (McKay, 1957).

The development of symptoms depends on weather conditions and also, probably, on the specific pathogen though the evidence for the latter is very limited. In artificial inoculations all five fungi listed above can cause the typical ear blight symptoms (Scott & Benedikz, 1987). No objective surveys have been done to determine the cause of ear blight in the field but F. culmorum is regarded as the most common together with F. avenaceum (Parry, Bayles & Priestley, 1984, 1985) (see page 44). F. nivale has been isolated from bleached spikelets in the UK (P.D. Hewett, personal communication) though it is probably more common as a seed-borne infection without conspicuous ear blight symptoms. In France it has been described as causing a brown glume spot with a dark brown margin (Rapilly, Lemaire & Cassini, 1973)

though Cassini (1981) states that it can also cause more severe symptoms. In Germany (Fehrmann, 1988a), severe attacks cause the brown glume spot symptom but the bleached spikelet symptom as caused by F. culmorum is rare and seed infection remains latent. However, in southern Germany, F. nivale is a common cause of severe ear blight symptoms (A. Obst, personal communication).

F. poae differs from the others listed above in causing only ear symptoms and in the field the symptoms are limited to glume spots very similar to those described for F. nivale (and also similar to those attributed to Botrytis cinerea). F. poae is often isolated from wheat ears at all stages from before ear emergence to maturity though it may not cause symptoms and the method by which it is transmitted is not understood (Sturz & Johnston, 1983; J. Lacey, personal communication) (see also page 37).

In the case of F. graminearum, in wet warm weather the disease progresses with blue-black perithecia (Gibberella zeae) developing on the affected tissue to give the ears a "scabbed" appearance. This disease is relatively uncommon in the UK but is the common cause of ear blight in Canada and the USA, and in parts of southern Europe. Epidemics in Eire have been described by McKay (1957).

The effect of ear disease on the grain depends on the time of infection. Early infections in the flowering period can cause bleached spikelets which contain no grain or very shrivelled grains. Later infections, or the death of spikelets above an infected rachis, are associated with symptoms ranging from severe grain shrivelling to no symptoms but sometimes with latent infection of the grain.

Grains infected by F. graminearum are described (Dickson, 1947) as more or less shrivelled with a scabby appearance due to tufty mycelial outgrowths from the seed coat and white, pink or light brown in colour depending on the time of infection and the prevailing weather. Grains infected with F. culmorum may be shrivelled; the fungus rarely, if ever, penetrates as far as the embryo though hyphae can be found on

the outer and inner surfaces of the seed coat (Colhoun, 1972). Millar & Colhoun (1969a) found grains naturally infected by F. nivale to be shrivelled with grey-brown patches around the embryos though such seed germinated well. Bateman (1983) isolated F. nivale from the seedcoat, endosperm and embryo but infections were concentrated in the space beneath the epidermis and in the crease of wheat grains. (The effects of seed infection are discussed in the section on seedling diseases; see also page 38 and page 46).

Conditions affecting ear blight

Sutton (1982) reviewing the epidemiology of wheat ear blight caused by F. graminearum found that epidemics in Canada were associated with above average rainfall in the summer and especially when harvests were consequently delayed. Similarly the incidence of Fusarium ear diseases in the UK is highest in Scotland, especially the north of Scotland, where there is a long ripening period. Sutton regards a substantial growth of F. graminearum on the anthers as prerequisite to natural infection of wheat spikelets. Ear blight epidemics are completed in one infection cycle. The ears are highly receptive for 10-20 days and then rapidly become resistant. Infections occur when weather favours the production and dissemination of spores to coincide with host receptivity (anther production) and then warm wet weather persists. Both ascospores and conidia play a part in the spread of the disease but there is no evidence available on the sources or dispersal of the inoculum. In the UK, F. nivale can spread through the dispersal of ascospores produced in perithecia on leaf sheaths but there is very little information on the dissemination of conidia or on the means of spread of other Fusarium pathogens (see page 36). The systemic infection by F. culmorum recorded by Snijders (personal communication) and Jordan & Fielding (1988) (see also page 30) may cause, or provide a source of, infection but the significance of this in the field is not yet known.

Anderson (1948) confirmed that the severity of symptoms was linked with anthesis. When wheat ears were inoculated during flowering, only those spikelets which produced anthers became infected. Spikelets were susceptible from the beginning of anthesis (but were resistant before) until the soft dough stage. Strange & Smith (1971) provided convincing evidence that anthers (pollen) promote infection. When extruded anthers were inoculated with F. graminearum massive growth of the fungus occurred followed by infection of other parts of the spikelet to produce typical ear blight symptoms. Very few spikelets were so affected following inoculations of ears from which the anthers had been removed. Further, later inoculations of anthers after pollen had been shed produced few infected spikelets. Pollen was therefore considered to contain all, or at least most, of the stimulant. Later (see Strange & Smith, 1978) choline and betaine were identified as constituents of pollen which exert a strong stimulatory effect on F. graminearum, promoting hyphal extension. They similarly affected F. culmorum and F. avenaceum, which are also known causes of ear blight, but not F. nivale which, Strange & Smith state, is not a cause of ear blight. However, as pointed out above, F. nivale has been associated with typical ear blight symptoms in the field and following artificial inoculations.

Although some workers contend that infection can occur wherever the fungus comes in contact with the spikelet or rachis (Schroeder & Christensen, 1963), it is generally accepted that anthers/pollen play a very significant role in the infection of ears.

For F. graminearum persistent wetness or high humidity and temperatures above 15°C are necessary for infection and disease progress; there is no progress below 15°C (Sutton, 1982). Anderson (1948) demonstrated that germination of conidia occurred in 3 hours at 28-30°C, in 6 hours at 20-24°C and up to 72 hours below 20°C. Pugh, Johann & Dickson (1933) showed that at 25°C ears inoculated and exposed to 36 and 48 hours continuous wetness produced 18 and 77 per cent infected ears, respectively. At 20°C, 60 hours of continuous wetness was required to produce 30 per cent infected ears and at 15°C only a trace of infection occurred.

Most of the work on F. graminearum as a cause of ear blight has been done in North America. Similar work on this or on other species of Fusarium causing ear blight has not been reported from Europe except for the work of Strange & Smith and observations following artificial inoculations (see page 52).

Spread

The principal sources of Fusarium diseases, seed and soil, were discussed in the section on seedling diseases. Seed is commonly affected by Fusarium, albeit normally at fairly low levels, and is an obvious means of spreading the disease from one place to another. Occasionally this can be seen clearly as in the outbreaks of F. graminearum (Gibberella zeae) in oats in Scotland which were attributed to infected imported seed stocks (Richardson, 1970). In the soil, spread is limited but there could be some spread through the transfer of infected straw from one field to another.

Fusarium diseases are initiated by sources of inoculum on seed or in the soil (including crop debris) within the crop. Some infection which occurs at an early stage may remain latent and cause obvious symptoms later (e.g. on stem bases). However, the spread of disease to the upper parts of the plant is assumed to be by the transfer of spores (but see also systemic infections, page 30).

All Fusarium species produce spores in the asexual state, conidia, and some produce spores in the sexual state, ascospores.

Millar & Colhoun (1969a) noted that the black perithecia of Monographella nivalis (anamorph Fusarium nivale), which develop first on the lowest leaf sheaths, did not ripen simultaneously on the same sheath. When the sheath was wetted asci were protruded beyond the ostiole and ascospores were discharged 15-20 minutes later (Sanderson, 1970). According to Miller & Colhoun (1969a), after being moistened with water, perithecia liberated ascospores in a salmon-coloured gelatinous substance which formed over the ostiole. Ascospores were released into the air as this dried. Sanderson (1970) states that this occurs only when the tissues dry out during the

period between ascus elongation and ascospore discharge. Normally ascospores are liberated forcefully into the atmosphere. Millar & Colhoun (1969a) trapped few spores in dry weather but many in the hour immediately after rain. Ascospores were trapped readily at 15 cm above soil level but none at ear height. Sanderson (1970) noted a diurnal periodicity with most spore discharge in the evening, 18.00-24.00 hours, and maximum discharge about 22.00 hours.

Leaf sheath infections and the occurrence of perithecia were associated first with sheaths at the bottom three nodes. Sometimes spread occurred later to the upper leaf sheaths and then there was usually one intermediate sheath free from perithecia, suggesting that the two groups of perithecia resulted from different infections. Snyder & Nash (1968) noted the very rapid cycle of ascospore to ascospore which can occur when light and temperature are favourable; this happened in 5-10 days during their one-year study.

Gibberella zeae forms perithecia on diseased ears ('scab') and stem bases and these mature to produce ascospores which can infect crops in the autumn and spring. Mature ascospores have not been observed in summer (McKay, 1957, Richardson & Cook, 1983),

All Fusarium species produce conidia, sometimes in large numbers and especially on dead tissues. It is assumed that these spores are rain splashed and perhaps carried in air currents from the basal parts to upper parts. There appears to be no information on the production and dissemination of these spores though Millar & Colhoun (1969a) comment that conidia (of F. nivale) were trapped mainly after rain.

F. poae is regarded as a weak parasite or saprophyte and the only disease symptom with which it is associated in cereals is a lesion on wheat glumes (except in artificial inoculations, see page 52). The same fungus is found in a bud rot of carnation and chrysanthemum and in these cases the condition is thought to be caused by a mutualistic association of the mite Siteroptes graminum and the fungus. There is no published record of these mites or any other pest

being involved with the spread and infection of F. poae in cereals though in one case in winter wheat in south west England Siteroptes mites and F. poae were associated with a rot of the node and adjacent stem tissue within the sheath of the flag leaf (M.J. Griffin, personal communication). Also Sturz & Johnston (1983) in Canada, reporting on early colonisation of ears by F. poae noted that thrips (Limothrips denticornis) were present in large numbers.

Damage by Fusarium diseases (after the seedling stage) is usually associated with wet weather. Ear and grain diseases, especially, are worst in wet seasons and especially when harvest is delayed or crops are lodged (Baker, 1972). In the latter case not only are the ears more likely to be wet for longer periods but they are close to sources of inoculum near soil level. In Canada, Sutton (1982) reviewing epidemics of wheat head (ear) blight found that in a 55 year period to 1980 the disease (caused by F. graminearum) was severe in six seasons when there was above average rain in June-August and in the severe epidemic of 1980 (when mycotoxins in wheat first became prominent) frequent rains delayed harvest for 2-4 weeks. He also comments on the lack of information on the dispersal of inoculum which causes ear disease, both the sources and the means of movement. Sturz & Johnston (1985), also in Canada, emphasise the latter point recording that although F. poae is one of the most common fungi on the ears it is rarely isolated from soil, organic soil debris or crown tissues.

Incidence, severity and losses

Seed-borne diseases

Cereal seed-borne diseases have been monitored by Hewett since 1959, with some particular attention paid to F. nivale which had been recorded as the most common of the seed-borne Fusaria in Scotland (Noble & Montgomerie, 1956).

In surveys of winter wheat seed in the five year period 1959-63 (sowing dates), seed infection with F. nivale was generally low except in 1963. During the first four years about one sample in seven showed infection, mostly 1-2 per cent, with average infection levels of about 0.3 per cent. In 1963 most samples contained infected grain. The cultivar Cappelle Desprez had 78 per cent of samples infected with an average seed infection of 5.3 per cent and a maximum of 37 per cent. Other cultivars showed fairly high infections but less than Cappelle Desprez (Hewett, 1965). Of the other Fusaria, F. avenaceum was the most common with about half the samples infected and an average seed infection of 1-2 per cent. In 1960 infections were well above average with 6 per cent of samples showing 10 per cent or more infection whereas in other years there were only occasional infections of more than 5 per cent. F. culmorum caused an average seed infection of 0.3 per cent. Of nearly 1000 samples examined in the five year period only 24 had 3 per cent or more seed infection (mainly in 1960). The incidence of F. poae was similar each year with about 1-1.5 per cent seeds infected and only very few samples with more than 5 per cent (Hewett, 1967).

Commercial seed samples are selected and screened, so to obtain data on infection levels in the field, Hewett (1966) examined seed as harvested from variety trials. In 1965 seed infections were high with a mean seed infection by F. nivale of 20 per cent (4 and 7 per cent in 1963 and 1964). F. culmorum and F. avenaceum (together) were also abundant with a mean seed infection of 39 per cent (10 and 9 per cent in 1963 and 1964) with particularly high incidences in lodged plots. F. nivale was more common on winter than on spring cultivars and one cultivar, Hybrid 46, was consistently heavily infected with both F. nivale and F. culmorum. Similar material harvested in 1969, 1970 and 1973 showed mean seed infections by F. nivale of 1-2 per cent. However, Hewett points out that very few samples from seed analysts of seed intended for sowing bore appreciable levels of F. nivale (Septoria nodorum was much more common). Up to this time the data supported the view that the seed-borne phase was only occasionally and erratically important. However, subsequent experience, including 1980 when levels were high, led Hewett (1983) to a modified view that F. nivale should be regarded as a frequent if still erratic hazard.

Hewett (1983) found that the high levels of seed-borne F. nivale in winter wheat harvested in 1980 were associated with low emergence when untreated seed was sown. He also points out a difficulty in testing naturally infected seed samples, in the field for emergence, since both Fusarium spp. and Septoria nodorum are usually present (the latter often the more common) and cause similar emergence problems. Colhoun (1970) recorded yield losses of up to 18 and 20 per cent in plots when plants were grown from seed inoculated with F. nivale and F. culmorum respectively. Losses occurred only when a sufficient number of seedlings were killed and when the surviving plants, though they tillered more, did not compensate for the plants killed.

Richardson (1974) in field trials with oats, also found a good relationship between seed infection with F. nivale and seedling emergence and also yield. Again the effect was related to a reduction in ear number because of a low seedling population. However, Colhoun points out that losses of the kind described are unlikely to occur frequently in commercial crops. In his experiments when conditions were not favourable for seedling disease (this would include use of treated seed) little or no loss of yield occurred.

In spring barley, F. nivale on seed appears to be less damaging than it is on winter wheat. Hewett (1975) examined over 200 samples from five cultivars in two seasons and found 39-52 per cent were affected with 1.0-2.5 per cent of grains infected though there were no adverse effects in germination tests. In Scotland in 1980 when high levels of F. nivale were recorded on seed, the pathogen had no effect on either laboratory germination or seedling emergence in pot trials (Rennie & Tomlin, quoted by Rennie, Richardson & Noble, 1983). In two years of experiments with artificially infected seeds, Perry (1986) found no effect on emergence, crop growth or grain yield. However, in a trial Rennie et al., (1983) found that seed with high levels of F. nivale infection had higher levels of seedling infection than untreated seed, though emergence was not affected.

Seedling diseases

In surveys in Scotland, F. nivale was the pathogen most often isolated from seedlings prior to tillering though whether the source was seed or soil could not be determined. Results for 1970-74 were as follows (Rennie et al., 1983):-

	Winter wheat		Spring barley	
	diseased*	healthy	diseased*	healthy
Number of seedlings	2657	13823	3300	15635
Per cent <u>F. nivale</u>	42	4	39	2
Per cent <u>F. culmorum</u> and other pathogens	5	2	3	1

* seedlings showing symptoms

The intensity and frequency of Fusarium spp. on Scottish crops was also recorded on seedlings (S) and adult plants (A):-

	<u>F. nivale</u>				<u>F. culmorum</u> / <u>F. avenaceum</u>			
	% infected:							
	Plants		Crops		Plants		Crops	
	S	A	S	A	S	A	S	A
Winter wheat (339 crops, 1971-74)	5-10	15-25	80-90	75-95	1	25-45	25-45	90-94
Spring barley (467 crops, 1971-73)	5-7	20-40	55-65	80-95	1	20-35	15-20	80-95
Spring oats (452 crops, 1974-76)	2-4	12-20	30-40	60-85	1-3	40-55	25-40	95-100

There are no equivalent survey data for England and Wales.

Foot rots (stem base diseases, brown foot rot)

In the Scottish surveys (see table above) when adult plant bases were examined in July the intensity and frequency of F. nivale and F. culmorum/F. avenaceum were higher than in the spring especially for the latter two pathogens. The authors state "it is difficult to assess the significance of such infections at that stage of crop growth, when there is much dead tissue for colonisation".

In the 1960's when F. nivale was first recognised as a prevalent pathogen in cereals in England and Wales, Colhoun, Taylor & Millar (1963) reported on a number of limited surveys. In most samples some plants showed discolouration of the basal leaf sheaths and nodes and the culms were sometimes discoloured. The proportion of plants affected ranged from 0 to 80 per cent and only few crops had no symptoms. Isolations from the stems revealed mainly F. nivale with small numbers of F. culmorum and F. avenaceum.

Foot rot (referred to as brown foot rot) has been recorded in some long-term trials at Rothamsted Experimental Station. In the period 1969-1976, in winter wheat cultivar trials on land which had grown at least one wheat crop immediately before, the disease had been in the range of 10-40 per cent tillers slightly affected, and severe symptoms (lesions extending around the circumference of the stem) in the range 5-20 per cent, but only in some seasons. From 1977 to 1986 records show a much lower incidence, usually less than 5 per cent tillers affected. In some winter barley multi-factorial experiments, 1981-87, brown foot rot has been recorded at low levels of 1-2 per cent tillers affected and only slightly higher in winter wheat straw residue experiments. Thus the disease is not considered to be very important though there are no objective assessments of the relationship between foot rot and yield loss (R.J. Gutteridge, J.F. Jenkyn, and R.D. Prew, personal communications).

Clark (1977) reported a three-year survey of spring barley crops in the west of Scotland where 34 and 27 per cent of stem bases were affected by F. nivale and F. roseum (= F. culmorum + F. avenaceum) respectively. Isolations from various tissues showed the following incidences of F. nivale and F. roseum respectively (per cent tillers affected): crown nodes 20 and 18, stem nodes 23 and 16, internodes 13 and 14, roots 7 and 6.

ADAS surveys of cereal diseases in England and Wales are based on a stratified sample of about 350 randomly selected crops and therefore give an objective assessment of the national incidence and severity of

diseases. Assessments of Fusarium diseases of the stem base have been done only in recent years. The assessments, made at the milky-ripe growth stage, are of symptoms (black or brown discolourations of the stem (culm)) on the node, internode and both. In 1987 stems were assessed as 'severe' when there was an indication that lesions were weakening the stem.

Nearly all the samples contained some stems affected by Fusarium. The following table summarises the results:

		mean per cent stems affected			
		nodal	internodal	both	severe
winter wheat	1985	27.91	5.65	4.63	-
	1986	5.16	21.34	9.26	0.26
	1987	5.30	11.17	4.51	0.51
winter barley	1986	12.87	5.30	11.02	-
	1987	9.11	13.65	10.23	1.02

There is no apparent explanation for the change in the incidence of nodal and internodal levels between 1985 and 1986. Further analyses of the data showed there were no obvious consistent effects on the incidence of the symptoms by any of the following factors: cultivar, previous crop, monoculture, sowing date and fungicide use (R.W. Polley, M.R. Thomas, personal communications).

In these surveys no attempt was made to identify the Fusarium spp. associated with lesions. This would have necessitated the time-consuming process of isolating the fungi on agar plates because the causal pathogen cannot be identified from the symptoms. However, Locke, Moon & Evans (1987) used 109 samples from the 1986 survey of winter wheat to make 704 isolates. Of these 82 per cent were F. nivale, 12 per cent F. avenaceum, 6 per cent F. culmorum and one isolate was F. poae.

It is not possible to use the survey data to assess the damage done nationally by Fusarium stem base diseases because there is no reliable information to relate the symptom categories to yield loss.

Clarkson & Polley (1981) reported some preliminary results using a method which involves the comparison of the mean yields of large numbers of single tillers in the different disease assessment categories with those of healthy tillers. The loss of grain yield per ear was 0.6, 6.0 and 12.5 per cent for the categories nodal, internodal and both infections, respectively. However, the yields of the affected tillers were not statistically significantly different from yields of healthy tillers. Assessed losses associated with the disease categories varied from 0-15 per cent (nodal) 0-47 per cent (internodal) and 0-41 per cent (both). The yield loss assessments are therefore considered to be not sufficiently reliable to be applied to survey data (J.D.S. Clarkson, personal communication).

Richardson, Whittle & Jacks (1976) also used the single tiller method in winter wheat to relate dark lesions on sheaths just above the nodes, normally associated with F. nivale, to yield. The disease assessments were made just after the ear emerged from the sheath (GS 10.5 = GS59). Later it was found, making isolations from stem base tissues, that F. avenaceum and/or F. culmorum were also present in affected stems. Some of the "healthy" stems yielded Fusarium spp. indicating that some infections apparently were not causing symptoms at the time of assessment. No lodging or whiteheads were associated with the lesions but the affected tillers yielded 20 per cent less than the unaffected tillers, with 75% of the loss due to a reduction in seed number and 25% to seed size. The same authors also report that in spring barley they found evidence of interactions between mercury seed treatment, F. nivale infection and mildew in which F. nivale (as recorded on the seed) intensified the loss due to mildew.

Ear diseases

Ear blight is said to be caused primarily by F. culmorum but F. avenaceum, F. graminearum, F. nivale and F. poae can also cause similar symptoms (Parry, Bayles & Priestley, 1984). Parry (personal

communication) isolated 60 per cent F. culmorum and 40 per cent F. avenaceum from a field sample. Dale & Heaney (personal communication) obtained 68 isolations from a sample of 30 ears derived from the ADAS national survey of which 34 per cent were F. nivale, 24 per cent F. poae, 21 per cent F. culmorum and 21 per cent F. avenaceum.

In a recent survey in Minnesota (Wilcoxson, Kommendahl, Ozmon & Windels, 1988) F. graminearum accounted for 75 per cent of isolates from grains from infected ears, F. poae 17 per cent and each of 13 other Fusarium species not more than 2 per cent. In pathogenicity tests F. graminearum and F. culmorum caused a disease of inoculated spikelets which spread to other spikelets. Most spikelets inoculated with the other 11 Fusarium species became necrotic but the disease usually did not spread.

Ear diseases have been assessed as part of the ADAS national surveys for many years but separate assessments of damage by Fusarium spp. were not done, because the incidence was very low. However, in 1982 when ear blight was very noticeable in crops, J.E. King (personal communication) commented that "the most unusual feature of the survey was the high frequency of crops showing ear discolouration on which Botrytis cinerea or Fusarium spp. could be seen to sporulate. This condition was observed in 65 per cent of the crops but, although the symptoms were the most severe of any of the ear diseases, they never covered a substantial proportion of the area of the glumes, infection levels being above 5 per cent in only 5 of the 320 crops surveyed". The highest incidences were in the East, South East and West Midlands Regions of the Ministry of Agriculture and least in the South West and Yorkshire and Lancashire Regions. The sample for the survey was taken at the milky ripe stage and may therefore underestimate both incidence and severity in the mature crops, though it is likely to give a reasonably good assessment of the amount of severe damage caused.

There appear to be no estimates of direct loss of yield due to ear diseases for the UK except that Colhoun (1970) records yield reductions of up to 13 per cent in experiments when ears were inoculated with F. culmorum or F. nivale. In most European countries this phase of the disease is considered to be the most damaging. Rapilly, Lemaire & Cassini (1973) reported losses of up to 50 per cent in France. In Canada, Sutton (1982) and Martin & Johnston (1982) reported high losses in some years and emphasise losses due to the associated production of mycotoxins. In Minnesota head blight is important at approximately 5-year intervals with the incidence of infected ears within a field ranging from a trace to 100 per cent (Wilcoxson et al., 1988).

Control

Seed treatments

Seed treatments give some control of seed-borne and soil-borne Fusaria. In pot experiments ethyl mercury chloride was more effective than phenyl mercury acetate (PMA, the organomercury in products currently marketed) in controlling seed-borne disease (Bateman, 1976). PMA was more effective against F. nivale on wheat than on barley probably because the fungus was more accessible to the chemical; in barley the "hulls" (the chaff adhering to the grain) act as a barrier. PMA was more effective against Septoria nodorum than against F. nivale probably because infections of the latter are more deep seated. Later Bateman (1983) established that in wheat seed treated with PMA there was considerably less viable infection in the outer epidermis but the fungus could still be isolated from the inner seedcoat, the endosperm and the embryo. He also found that most fungus in naturally infected seed was in the space beneath the epidermis and in the crease. Thus the partial control by organomercury seed treatment can be explained by the depth of infection and the presence of the fungus in the crease, an area not effectively covered by the fungicide. Nevertheless, the marked improvement in emergence afforded by the seed

treatment suggests that the part of the seed infection eradicated by the treatment is that which most readily causes seedling disease. The importance of the seed treatment is demonstrated in the field occasionally when heavily infected seed is sown untreated leading to a thin stand and to yield losses (Richardson, 1974, Colhoun, 1970; see also page 40).

Some alternatives to organomercury have been introduced. A newly introduced seed treatment containing quintozone was associated with many reports of seedling blight in 1958/59 (Baker, 1972). The chemical, although effective against some seed-borne diseases, was much less effective than organomercury against both Fusarium and Septoria seed-borne diseases. The position was probably exacerbated by a higher than average incidence of Fusarium ear blight during the 1958 harvest resulting in a higher than usual inoculum on seeds. This case is quoted as a classic example of what can happen when a fully effective seed treatment is not used regularly. However, Richardson (1986) following trials over a 5-year period in Scotland, considered that seed treatment is not necessary for certified seed sown to produce a non-seed crop, except when loose smut is known to be present (the seed he used without benefit of organomercury treatment was the final generation of certified seed, all seed in the earlier multiplication stages having been treated). In a separate single trial over a 7-year period even with repeated sowing of untreated seed of winter wheat and spring barley on the same land, most diseases, including those caused by Fusarium spp. did not increase (Davis, Jacks, Thomas, Young & Richardson, 1987). In commenting on these results the authors point out that the system could not be recommended without more evidence from a comprehensive series of trials.

The introduction of the product 'Baytan' in the late 1970's gave an improved control of Fusarium seed-borne diseases. 'Baytan' contains the triazole fungicide, triadimenol and the MBC fungicide, fuberidazole. Wainwright, Rollett & Morris (1979) reported the control of seed-borne F. nivale as follows:

Per cent control F. nivale

treatment, g a.i./100 kg seed

triadimenol	triadimenol + (fuberidazole)	PMA** + carboxin	% infected tillers untreated
30	30+ (4.0-4.8)	*37.5+ (4.5-6.0)	
<u>w. wheat</u>			
58	99	99	66
<u>w. rye</u>			
76	99	98	31

* as in 'Baytan'

** PMA = phenyl mercury acetate

Initially triadimenol was considered to be insufficiently active against Fusarium spp. and the MBC fungicide fuberidazole was included in 'Baytan' to improve the control (Frohberger, 1978). Subsequently F. nivale was reported as resistant to MBC fungicides in Germany and in some other countries including, recently, the UK. In 1986 Locke, Moon & Evans (1987) obtained 704 isolates from 109 winter wheat crops in England and Wales. Of these isolates, obtained mainly from superficial nodal or internodal lesions, 82 per cent were F. nivale and 92.1 per cent of these were resistant to the MBC fungicide benomyl. Of the remaining isolates 12 per cent were F. avenaceum, 6 per cent F. culmorum and one isolate was F. poae; none of these was resistant to benomyl. In 1987 from a small sample of 68 infected ears collected from winter wheat crops (S. Dale & S.P. Heaney, personal communication) 34 per cent of the isolates were F. nivale and all were carbendazim (MBC) resistant; 24 per cent were F. poae (56 per cent resistant), 21 per cent F. culmorum (14 per cent resistant) and 21 per cent F. avenaceum (7 per cent resistant).

Despite the decision to complement the activity of triadimenol against Fusarium spp., the fungicide on its own gave a control somewhat similar to that given by organomercury, a finding confirmed in recent

trials (T.J. Martin, personal communication). The use of triadimenol is therefore likely to prevent serious losses of seedlings through seed-borne infections of Fusarium spp. A similar conclusion has been reached in the case of the other triazole fungicide, flutriafol, used together with the MBC fungicide, thiabendazole (and ethirimol) in the seed treatment product 'Ferrax' (R.A. Noon, personal communication). Chemical companies are seeking a replacement for MBC fungicides to improve the control given by the triazole fungicides and to obtain diversity in the mode of action of the chemicals. One chemical which has a different mode of action and has been found to be effective against Fusarium is guazatine (Bartlett & Ballard, 1975; Clark, 1977) but it is not included in any product currently marketed.

Seed treatments may also have some effects on soil-borne sources of infection. Rawlinson (1971) found that organomercury reduced seedling infections from soil sources from 25 per cent to 12 per cent. In growth room studies, Bateman (1977) showed that phenyl mercury acetate significantly reduced seedling disease (resulting from inoculum mixed with the soil) caused by F. avenaceum, F. culmorum and F. nivale. The protection of the coleoptiles indicated movement of the fungicide from the seed either within the plant or into the soil to form a protective zone. The effect was not reduced by any of the factors which would favour disease such as deep sowing, high inoculum and dry soil.

Biological control

Biological control in this context refers to the control of Fusarium disease by other micro-organisms. Tveit & Wood (1955) obtained a good control of seedling blight of oats with some isolates of Chaetomium cochlioides and C. globosum added to the seed or to the soil in which seeds naturally infected with Fusarium spp. (mainly F. nivale) were sown. Control in greenhouse tests was about equivalent to that given by organomercury seed treatment but in the field control was less satisfactory. Millar & Colhoun (1969b) found that in some soils little or no disease developed when seed inoculated with F. nivale was used, and concluded that this was due to the presence of inhibiting or competitive micro-organisms. One fungus they isolated, Gliocladium roseum, when added to artificially contaminated seed reduced the

incidence of seedling disease. However it did not control disease initiated by naturally infected seed and it was assumed that it inhibited only infections on the seed surface. Bateman (1979, 1983) showed that common saprophytes, especially Alternaria reduced the severity of seedling infections and also the colonisation of the seed in the ear by F. nivale. The latter may explain why the incidence of seeds infected by F. nivale rarely exceeds 30 per cent. Al-Hashimi & Perry (1986b) showed that Trichoderma viride, a common and aggressive saprophyte of straw, completely suppressed the growth of F. nivale in agar culture and also reduced the number of diseased seedlings when added to soil in which seed, with low inoculum levels, was grown. The treatment may not be so effective against deep seated infections (as noted by Millar & Colhoun above) but they point out that soil is a more consistent source of infection and that if infections by F. nivale occur during the early stages of germination then the activity of an antagonist would be needed only during germination to control effectively the seedling phase of the disease.

Fungicide sprays

The important stem base disease eyespot (caused by Pseudocercospora herpotrichoides) can be partially controlled by fungicidal sprays applied mainly in the stem erect to second node growth stages (GS30-32). The fungicides used were the MBC's (until MBC-resistance became widespread) and, currently, prochloraz.

There has been much speculation about the effects of the latter on stem base disease caused by Fusarium spp. Prochloraz is contained in the products Sportak and Sportak Alpha and their label recommendation states "....sprayed for the control of eyespot will often give good suppression of sharp eyespot and Fusarium if these diseases are developing at the time of application, but it is not yet known when this effect is likely to be of benefit". The results from seven years of trials have been inconsistent though with a tendency for sprays to reduce disease levels (W. Griffiths, personal communication). In four ADAS trials, in 1987, a spray of prochloraz at GS 31 (first node) was associated with a significant reduction in the mean proportion of stems affected by Fusarium at the milky ripe stage from 27 per cent to 18 per cent and in a few other ADAS experiments some similar reductions have been noted. Dr M.R. Thomas (personal communication)

has analysed the ADAS national survey data for an effect of fungicides applied at GS 31 (first node) on Fusarium stem base diseases assessed at GS 75 (milky ripe). The following selected data is for winter wheat in 1985 and 1987:-

Winter wheat 1985	no. of samples	Per cent stems affected by <u>Fusarium</u>		
		nodal	internodal	both
fungicide group				
MBC's (excluding prochloraz)	109	30.88	5.96	5.12
prochloraz (excluding MBC's)	16	22.00	6.24	7.76
prochloraz + MBC	68	25.60	3.84	3.04
Other DMI's (excluding MBC's)	19	30.12	7.80	1.88
unsprayed at GS 31	83	27.44	5.60	3.84

Winter wheat 1987

fungicide group				
MBC's (excluding prochloraz)	84	4.96	11.32	4.84
prochloraz (excluding MBC's)	65	4.48	13.04	4.36
prochloraz + MBC	59	5.68	10.84	4.56
Other DMI's (excluding MBC's)	14	3.44	10.00	2.56
unsprayed at GS 31	135	6.00	10.56	4.28

Data for winter wheat 1986 and for winter barley 1986 and 1987 are very similar and none provide evidence that sprays of prochloraz reduce disease.

In one ADAS trial there was evidence that mixtures of MBC and dithiocarbamate fungicides applied at the beginning of flowering significantly reduced the numbers of spikelets affected by Fusarium culmorum and F. avenaceum whereas fungicides containing prochloraz + MBC, propiconazole, triadimefon and prochloraz + MBC did not. No other information on this subject has been discovered.

The general conclusion, therefore, is that for stem base diseases caused by Fusarium there is no substantial evidence that sprays containing prochloraz (or, in fact, any other fungicide), as currently used, give an effective and consistent control. The evidence for a control of ear diseases is very limited. The incidence of significant levels of such diseases is erratic and infrequent and since outbreaks cannot be forecast sprays cannot be applied selectively and in time to be effective.

Varietal resistance

The only varietal resistance at present available to farmers is to the ear blight phase of the disease. Disease ratings were first published for winter wheat varieties in the National Institute of Agricultural Botany's Farmers Leaflet No 8 - 'Recommended Varieties of Cereals' in 1987. The ratings are based on a combination of data from natural and artificial infections over a 3-year period. Data for naturally infected trials in 1982 (the year when the disease was particularly prominent) and inoculated plots in 1983 showed that there were significant differences in varietal reactions. Data from the two sets of observations were significantly correlated, though the agreement is not particularly good ($r = 0.56$, $p = 0.05$) (Parry, Bayles & Priestley, 1984). The fungus used in the inoculation tests was F. culmorum, said to be the most common cause of ear blight. F. avenaceum was also found to be a common cause in 1983 and subsequently the two species were compared by inoculating 18 cultivars of winter wheat (Parry, Bayles & Priestley, 1985). F. culmorum caused more severe disease in the earlier (GS 80, 85, early-soft dough stages) but not in the later (GS86, 88, soft-hard dough stages) assessments. Significant differences in varietal resistances to ear blight were again demonstrated but with no significant interaction between cultivars and the pathogens inoculated. It was therefore concluded that either species of Fusarium could be used for varietal testing but F. culmorum was chosen because it is more common and more pathogenic.

At Plant Breeding International (formerly the Plant Breeding Institute), Cambridge, breeding material is now routinely screened for resistance to Fusarium ear blight. Resistant cultivars are aimed partly at the home market but mainly at continental Europe where ear blight is considered to be a major disease (R. Summers, personal communication). Tests with five species, F. culmorum, F. graminearum, F. avenaceum, F. nivale and F. poae showed that all caused similar symptoms and ranked cultivars in the same order (Scott & Benedikz, 1987) (though not in entirely the same order as in the results reported by Parry et al., 1984, 1985). Symptoms developed well when spore suspensions were sprayed on to the ears, the plot was covered with a polythene bag for 48 hours and subsequently mist irrigated for several weeks. Variation in inoculum concentration

(1×10^2 to 5×10^5 spores ml^{-1}) had little effect on disease severity. In one season inoculating just before anthesis resulted in higher levels of disease than during and at the end of anthesis but in a second season there were only small differences between inoculation dates (Scott & Benedikz, 1986).

In the Netherlands, Snijders (1987) found significant interactions between wheat genotypes and isolates of F. culmorum causing ear blight. He therefore advocated the use of several pure isolates in screening genotypes for resistance.

Screening breeding material for resistance to foot rots at the Plant Breeding International has not been possible because there is no method for reproducing severe disease. For two seasons the introduction of additional inoculum to the soil and to the growing crop failed to increase the severity of symptoms on stem bases over the slight-moderate disease on the uninoculated plots (T.W. Hollins, personal communication).

Colhoun (1972) refers to some earlier work in the USA and Scandinavia on resistance to F. nivale causing snow mould and also to the screening at Manchester of wheat cultivars and species for resistance to F. nivale in seedlings, leaf sheaths and stem bases. Some genotypes showed some resistance in the field but none had a high level of resistance.

There appears to be no evidence from UK sources (e.g. variety trials) of differential varietal reactions to Fusarium stem base diseases.

Fusarium diseases of cereals in some other countries

The book "Fusarium: Diseases, Biology and Taxonomy" (Ed. Nelson, Toussoun & Cook, 1981) includes chapters on Fusarium diseases of cereals, mainly wheat, in North America (R J Cook, pp. 39-52), China (R J Cook, pp. 53-55), Western Europe (R Cassini, pp. 56-63), Eastern

Australia (L W Burgess, R L Dodman, W Pont & P Mayers, pp. 64-76) and Eastern Europe and the Soviet Union (A Maric, pp. 77-93).

In North America ear blight and scab are of major importance in the main wheat-growing areas (see also page 33). Snow mould, caused by F. nivale occurs in some northwest and northeast states. Seedling diseases are largely associated with seed-borne inoculum and dry soils at the time of germination. Of most interest to the UK reader is the foot rot disease described by Cook (1980, 1981) in the Pacific northwest which has been discussed previously (page 27).

In France ear blight associated with F. roseum (= F. culmorum, F. avenaceum and/or F. graminearum) and to a lesser extent seed-borne disease are considered to be the most important Fusarium diseases. A working party from state-sponsored institutes, I.T.C.F. and chemical companies has been set up to standardise methods and plan co-operative experiments mainly aimed at the control of ear diseases especially with fungicides. Some coded chemicals have given promising results. (G. Daguenet, personal communication). Foot rots are not common on good land but are associated with premature ripening on poorer land (Cassini, 1981; see also page 27 and 28).

In the Netherlands, ear diseases are regarded as the most damaging though seedling blight and foot rots are reported. Yield losses from ear diseases may have averaged 5 per cent during the past 10 years with losses in some fields as high as 25-40 per cent. During the same period F. nivale has become more troublesome than the group including F. culmorum, F. avenaceum and F. graminearum (C.J. Langerak, personal communication). Investigations with F. culmorum have indicated that there are factors for resistance at all stages of plant development which could be used in breeding resistant winter wheat cultivars. (C.H.A. Snijders, personal communication).

In northern West Germany a comprehensive investigation of Fusarium diseases of winter wheat was carried out by Duben & Fehrmann (1979a, 1979b, 1980). F. avenaceum was the species most frequently isolated from stem bases though it is not considered to be an aggressive pathogen. Following artificial inoculations which caused stem base symptoms, only F. culmorum caused significant crop losses. Since that time seed-borne F. nivale caused some serious losses after organomercury seed treatments were banned. Seed infections are sometimes high but there are no obvious direct losses in yield, the serious damage being caused when infected seed initiates attacks of snow mould. Since 1981 leaf diseases in the latter part of the season caused by F. nivale have become prominent. F. culmorum has been associated with foot rots in the warmer regions but they are never as important as eyespot. Ear blight attacks associated with F. culmorum are sometimes very severe but occur erratically (H. Fehrmann, personal communication). In Bavaria foot rots are the least important of the Fusarium diseases, though eyespot is prevalent. Seed-borne F. nivale can cause emergence problems in winter wheat and initiate snow mould in winter barley and rye. Usually the seedling losses are well controlled by seed treatments. The most damaging disease is ear blight caused by F. graminearum and F. nivale. F. avenaceum and F. poae also sometimes cause ear blight but F. culmorum is seldom found on the ears. Yield losses are sometimes as high as 20 per cent. (A. Obst, personal communication).

In Eire, Fusarium diseases have not received much attention since McKay's investigations of the outbreaks of severe disease caused by F. graminearum (see McKay, 1957). In the mid-1970's foot rots were prevalent and more recently, especially in the wet season of 1986, ear diseases were prominent. Winter wheat grain samples in 1986 showed 58 per cent grain affected by all Fusarium spp. with 35 per cent affected by F. nivale. (P.C. Cunningham, personal communication).

THE MYCOTOXINS

Introduction

Fusarium spoilage of cereals in the field causes the grain to become shrivelled and discoloured pink or red. Grain can be rejected by feed compounders because of this discolouration which occurs mainly in years when crops have been lodged by heavy rain and the harvest delayed. Apprehension about using Fusarium-infested grain in animal feeds arises from the risk that chemical toxins (mycotoxins) produced by Fusarium species could be present. The presence of mycotoxins in affected cereals is well documented, e.g. on wheat in Canada (Trenholm, et al., 1981) and the USA (Hagler, Tyczkowska & Hamilton, 1984). Some of them are extremely toxic to man and livestock, causing a variety of disorders. Hence the feed trade takes precautions to guard against problems that could arise from using grain showing any sign of Fusarium damage. This caution is well justified but it does not follow that colonisation by Fusarium necessarily indicates the presence of Fusarium mycotoxins. Although Fusarium mycotoxins are mostly species specific some can be produced by more than one species. The following is a list of some of the more commonly described Fusarium mycotoxins and the Fusarium species known to produce them:-

<u>Mycotoxin</u>	<u>Producing species</u>
<u>Trichothecenes</u>	
Diacetoxyscirpenol	<u>F. poae</u> , <u>F. tricinctum</u>
Deoxynivalenol	<u>F. culmorum</u> , <u>F. graminearum</u>
Fusarenon-X	<u>F. nivale</u>
HT-2 toxin	<u>F. poae</u> , <u>F. tricinctum</u>
Neosolaniol	<u>F. poae</u>
Nivalenol	<u>F. nivale</u>
T-2 toxin	<u>F. poae</u> , <u>F. tricinctum</u>
<u>Others</u>	
Butenolide	<u>F. nivale</u>
Moniliformin	<u>F. moniliforme</u>
Zearalenone	<u>F. culmorum</u> , <u>F. graminearum</u> <u>F. tricinctum</u>

It is also possible for one species to produce more than one mycotoxin. Within a species, many strains exist which may differ in their ability to produce mycotoxins. The potential of a strain to produce a mycotoxin will not be achieved until the necessary critical combination of moisture and temperature over a period of time is attained. This is best illustrated in the field by the production of certain mycotoxins on grain only after the crop has remained lodged in a cold and wet condition for several weeks.

The Fusarium mycotoxins

The number of known Fusarium mycotoxins increases annually as new compounds and their derivatives are identified. To date, about twenty compounds have been found to occur naturally on cereals, but many more related compounds have been identified in laboratory cultures of Fusarium. These mycotoxins are conveniently grouped according to their chemical structure (see table on previous page).

The largest group of compounds is comprised of the trichothecenes which include diacetoxyscirpenol, deoxynivalenol and nivalenol. Some of the trichothecenes e.g. diacetoxyscirpenol are highly toxic causing haemorrhagic disease, others e.g. deoxynivalenol are less toxic, but can cause feed rejection or vomiting in pigs (Vesonder, Ellis & Rohwedder, 1981). The effect of trichothecenes on man first became widely apparent between 1942 and 1947 in the population of the Oreburch district of the USSR. A previously undescribed disease whose symptoms included leukopenia (reduction in white cell count), multiple haemorrhage, sepsis and depletion of the bone marrow, affected thousands of victims. This disease became known as alimentary toxic aleukia and was attributed to the consumption of grain which had overwintered under snow and been invaded by Fusarium species leading to the production of trichothecenes (Joffe, 1978).

Instances of Fusarium mycotoxicosis in humans are rare, but they are more frequently reported in livestock. The mycotoxin zearalenone has a chemical structure closely resembling that of the hormone oestrogen. When gilts are fed cereals contaminated with zearalenone they display

hyperoestrogenic symptoms which include vaginal prolapse and vulva vaginitis (Long et al., 1982). This condition is usually associated with feeding contaminated maize in North America. The frequency of zearalenone contamination of maize grown in Canada is highest in those years having a heavy rainfall in August (Sutton, Baliko & Funnell, 1980).

Other naturally occurring Fusarium mycotoxins in cereals include butenolide, found in Japanese wheat (Yoshizawa, 1984) and moniliformin in Canadian maize (Scott & Lawrence, 1987).

Occurrence of Fusarium mycotoxins in home-grown cereals fed to livestock

In view of the possibility that Fusarium mycotoxins could be contributing to livestock disease problems in England and Wales, ADAS Microbiology Laboratories have undertaken the analysis of suspect feeding stuffs and home-grown cereals for mycotoxins from 1975 to the present day. The method used (Patterson & Roberts, 1979) screens for eight mycotoxins including three produced by Fusarium species, namely diacetoxyscirpenol, T-2 toxin and zearalenone. The method uses thin-layer chromatography (TLC) which is relatively insensitive for these mycotoxins, having detection levels of about 200 ug/kg for diacetoxyscirpenol and T-2 toxin and 50 ug/kg for zearalenone. This analysis has been applied to over 1600 samples of home-grown cereals. Neither diacetoxyscirpenol nor T-2 toxin have been detected in any sample whereas about 1.5% were positive for zearalenone. In one instance a more sensitive analysis was applied to a suspect sample of oats which proved positive for T-2 toxin (Buckle, 1983).

In Scotland animal feeding stuffs have also been examined for Fusarium toxins, using the TLC method, at the Edinburgh School of Agriculture. In 1980 it was applied to poultry feed in a particular case when broilers displayed symptoms of poor feathering and stunted growth which could not be attributed to bacterial or viral infections. The suspect feed contained a large proportion of wheat produced in Scotland in 1980. No Fusarium toxins were detected, by the limited

TLC technique, but positive results were obtained in a cytotoxicity test in which an extract of the feed was applied to malignant human epithelial cells cultured in the laboratory. This technique is particularly suitable for detecting trichothecenes because some of them are highly toxic to certain cell cultures. When the feed extract was examined by a more sensitive chemical method both diacetoxyscirpenol and deoxynivalenol were detected (Robb, Kirkpatrick & Norval, 1982). The symptoms observed in the affected birds were consistent with the reported effects of diacetoxyscirpenol in experiments on poultry (Hoerr, Carlton, Yagen & Joffe, 1982). It is significant that wheat from the 1980 harvest which was used in these feeds was of extremely poor quality because of Fusarium damage which had occurred following an exceptionally wet and delayed harvest. However, this was not an isolated incident and problems continue to occur in poultry and pig production where it is suspected that Fusarium mycotoxins are responsible (J. Robb, personal communication). Great care is now taken by some poultry producers to screen cereals locally produced in Scotland to avoid using lots contaminated with Fusarium mycotoxins.

Surveys of home-grown cereals for Fusarium moulds and Fusarium mycotoxins.

The sporadic detection of Fusarium mycotoxins in cereals used in animal feeding stuffs, implicated in causing disease in livestock, provides no accurate indication of their real incidence. More representative data are provided by surveys. These have been undertaken by MAFF and others for a limited number of Fusarium mycotoxins on home-grown barley and wheat since 1980. In addition, four surveys for Fusarium moulds on cereals at harvest have been carried out since 1971.

Barley

The occurrence of Fusarium moulds on barley in 1971-74 has been investigated by Hacking, Rosser & Dervish (1976). In 1971 barley samples from only two out of six sites in the East Midlands yielded Fusarium isolates, which were F. culmorum and F. poae. One of the

F. culmorum isolates was able to produce zearalenone in the laboratory. In 1973 barley was sampled from 36 sites in England and Wales. Out of 63 isolates of F. culmorum, 62 were able to produce zearalenone. A larger survey for Fusarium species on barley was conducted in England and Wales in 1974 when 787 isolates were obtained from 57 sites. The most commonly isolated species was F. culmorum; out of 431 isolates 270 were able to produce zearalenone. Other species also isolated and able to produce zearalenone include F. graminearum and F. avenaceum. The complete list of species isolated is given in Appendix 2. Despite the presence of toxigenic species none of the barley samples collected in 1974 was found to contain zearalenone using a method having a detection level of 50 ug/kg.

The 1980 barley harvest was surveyed for deoxynivalenol using gas chromatography combined with mass spectrometry having a detection level of 10 ug/kg (Gilbert, Shepherd & Startin, 1983). A total of 85 samples was analysed comprising 43 of feed barley and 42 of malting barley obtained from the main growing areas in England and Scotland. The number of samples analysed from each area was in proportion to the production from that area relative to the total UK production. Deoxynivalenol was detected in 51 and 31 per cent respectively of the feed and malting barley samples. The levels present are given in the following table:-

	Number of samples	Number of samples in range deoxynivalenol mg kg ⁻¹			
		<0.01	0.01-0.02	0.02-0.1	>0.1
Feed barley	43	21	11	9	2
Malt barley	42	29	5	8	0

There was no geographical trend evident in the results giving no indication that the colder/wetter climatic conditions in the north of England and Scotland produced any significant differences in the level of contamination compared with southern England.

In a limited survey of barley grown in Scotland reported by Tanaka et al., (1986) on the 1984 harvest, 8 samples were examined for deoxynivalenol, nivalenol and zearalenone using a method having detection limits of respectively 2, 2 and 1 ug/kg. Deoxynivalenol, nivalenol and zearalenone were detected respectively in 4, 2 and 8 samples.

The results of all the surveys are presented in Appendix 3.

Wheat

The occurrence of Fusarium species on wheat at harvest was surveyed in 1982 when 214 samples, mostly of winter wheat, were collected in England and Wales (MAFF 1987). The numbers of samples from different regions of the country were approximately proportional to their estimated wheat production. Grains were individually cultured to determine the incidence of Fusarium infections and species present.

The relative proportion of each species present was determined both as the percentage of grains infected with a particular species and as the percentage of samples in which a particular species predominated. The results, which were very similar, are given in below:-

Species	% of samples on which a species was predominant	% of grains infected
<u>F. poae</u>	42	34
<u>F. tricinatum</u>	18	18
<u>F. culmorum</u>	17	18
<u>F. avenaceum</u>	13	17
<u>F. nivale</u>	9	10
<u>F. graminearum</u>	1	2

Fusarium poae was the most commonly isolated species being predominant in 42 per cent of samples examined. In view of results previously obtained from barley (Appendix 2) it was surprising that F. graminearum occurred so infrequently; it was dominant in only 1% of samples. F. culmorum and F. tricinctum, which are both able to produce zearalenone, were predominant on 17 and 18 per cent samples respectively.

Analysis for zearalenone of 10 samples having the highest incidence of contamination with these species was negative (the method used had a detection level of 50 ug/kg). Analysis for five trichothecenes was also carried out on 55 samples using gas chromatography. Six samples contained deoxynivalenol and three contained fusarenon-X (Appendix 3).

The toxigenic potential of 149 cultures of Fusarium species isolated in this survey was determined by culturing them on Vogel's medium at 20°C for 3 weeks (Niles et al., 1984). Culture extracts were examined for several Fusarium toxins using thin-layer chromatography. They showed that a significant proportion of the isolates were able to produce one or more toxins. Many of the F. culmorum isolates produced both deoxynivalenol and zearalenone; similarly most of the F. poae isolates which produced diacetoxyscirpenol were able to produce T-2 toxin. It is interesting that although these toxigenic species were present the climatic conditions had not resulted in production of all these mycotoxins in the grain.

Home-grown wheat has been examined for Fusarium mycotoxins at harvest in several surveys by the Flour Milling & Baking Research Association over the period 1980-82 (Osborne & Willis, 1984). Using gas liquid chromatography 199 samples were examined for seven trichothecenes. Deoxynivalenol was the only mycotoxin detected; it was found in 16 per cent of the samples at levels ranging between 20-40 ug/kg. The results are presented in Appendix 3.

The most recently conducted and published survey of wheat for Fusarium toxins was done in 1984 (Tanaka et al., 1986). The method employed gas chromatography-mass spectrometry (selected ion monitoring) and was more sensitive than those used in previous surveys. The detection limits for deoxynivalenol, nivalenol and zearalenone were respectively 2, 2 and 1 ug/kg. A total of 33 wheat samples was examined from 31 farms throughout England and 2 in Scotland. Deoxynivalenol, nivalenol and zearalenone were detected in 64, 52 and 18 per cent of samples respectively. The detailed quantitative results are presented in Appendix 4.

Conclusions

The data obtained from the limited number of surveys carried out show that toxigenic species of Fusarium are widely found on home-grown cereals and that Fusarium mycotoxins are relatively common in some years, albeit at relatively low levels. The detection frequency for mycotoxins has risen with the application of increasingly sensitive analytical techniques. However, it is not conclusively established that these mycotoxins would pose a hazard to man or livestock at the low level at which they can now be detected. The formation of Fusarium mycotoxins is weather dependent at harvest with wet weather and a prolonged harvest increasing the risk. There are some indications that for this reason cereals produced in Scotland are more at risk, though, at present, there is insufficient data from Scotland to determine if Fusarium mycotoxins occur more frequently and at higher levels than in England.

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RECOMMENDATIONS FOR FURTHER WORK

A good deal of information is available about the seed-borne and the seedling phases of Fusarium diseases. However the seed-borne diseases should be monitored periodically, possibly as part of a survey of all cereal seed-borne diseases, so that any changes can be detected in time to allow for any action which may need to be taken. It is also important to maintain independent assessments of fungicide seed treatments - tests for effectiveness and monitoring to ensure the effectiveness is maintained, particularly in relation to fungicide resistance.

Snow mould caused by F. nivale is a minor national problem though it can be important in Scotland where it occurs mainly together with the more important snow rot (Typhula incarnata). It may be feasible to include some studies on snow mould in any investigations of snow rot.

Very little information is available on Fusarium diseases affecting the stem bases (foot rots, brown foot rot). These are attracting much attention (partly as a result of the attention paid to eyespot) as possible causes of premature ripening and of lodging late in the season, with adverse consequences for yield and grain quality. As pointed out in the review there is no evidence to substantiate general statements made about damage caused by the foot rot phase of Fusarium diseases nor is much known about the nature and specific causes of these diseases.

A comprehensive study of the etiology (causes) of Fusarium foot rots is recommended together with an investigation of the conditions which favour the development of the disease and an assessment of the damage to grain yield and quality. This is likely to be a difficult project. Cook (1980, 1981) in the U.S.A. has pointed out the difficulties associated with studying a foot rot which could not be produced under laboratory conditions and at Plant Breeding International, Cambridge, attempts to devise a method of screening varieties in the field for resistance to foot rots have not been successful (T.W. Hollins, personal communication). The project is not, therefore likely to be

successful as a short term or part-time study. However, it could be considered as part of a larger project on cereal foot rots at one specialist centre. Because of the difficulty of the project there is merit in considering some short term aspects (e.g. surveys, loss assessment) at other centres where different approaches may shed new light on the subject.

Some useful quick information may be obtained by a survey to study the incidence and cause(s) of foot rot symptoms. However, great care would have to be exercised in planning the work (e.g. regarding descriptions of symptoms, timing of observations, method of isolating pathogens, etc.) so that the methods employed are likely to give an objective view of the problem.

Some information is available on ear diseases but there are important gaps. No objective assessment has been made of the causal pathogens in the UK, nor of the amount of damage caused. Also little is known about the production and dissemination of spores which cause the ear blights. Both subjects could be investigated as short term (up to 3 years) projects.

It is, perhaps, surprising that all five Fusarium species cause similar ear blight symptoms and that there are no interactions between the species and cultivars in the artificial inoculation tests. A detailed study using specific isolates and varying spore loads under a variety of environmental conditions should be done to confirm that these findings reflect what is occurring in the field.

Mycotoxins produced by Fusarium species can cause serious disorders in livestock. Although work to date suggests that they are not normally a hazard in UK-produced grain, further surveys should be done to monitor the position. Also work should be done to establish "acceptable" levels of contamination for each mycotoxin at which no harmful effects on man or livestock would be expected.

Summary of recommendations

Priorities are relevant within the subject of Fusarium diseases.

Inputs: medium = 1-3 man years, low = <1 man year, high = >3 man years.

1. A study of the etiology of Fusarium foot rots, the conditions affecting disease development and the effects of the diseases on the plant, especially on grain yield and quality.

A difficult long-term project, preferably to be carried out mainly at a centre specialising in cereal foot rots.

However, because of the difficult nature of the problem consideration should also be given to some specific short term projects at different centres to encourage new or novel approaches.

High priority, high input

2. Fusarium foot rots, a survey of incidence and causes.

This would be appropriate as an additional project for the ADAS national survey of cereal diseases in England and Wales (with appropriate arrangements for Scotland and Northern Ireland).

A 3-year study in the first place.

Great care should be taken to use appropriate methods, and to liaise with workers who may be working on the etiology of foot rots.

Medium priority, medium input

3. An assessment of the causes of ear blight in field crops and the damage done. In the first place this could be limited to a 3-year survey and would also be appropriate as a special additional part of the ADAS survey in England and Wales (with appropriate arrangements for Scotland and Northern Ireland).

Medium priority, medium input

4. Epidemiology of Fusarium ear blights. A study of the sources of infection, the production and dissemination of spores.
Medium priority, medium input
5. Etiology of ear blight.
Factors affecting infection by five species of Fusarium. This may be considered as a shared project with a plant breeding unit.
Medium priority, medium input
6. Monitoring seed-borne diseases.
This could be carried out periodically or as an on-going project on cereal seed-borne diseases in co-operation with the Official Seed Testing Station, Cambridge.
Low-medium priority, low input
7. Effectiveness of seed treatments.
An independent evaluation of new chemicals and a periodic monitoring of the effectiveness of the chemicals on the market, especially in relation to fungicide resistance.
Projects to be set up as necessary for testing and periodically to monitor for effectiveness. Similar projects have been done by ADAS in the past.
Medium priority, low input
8. Mycotoxins: a comprehensive surveillance of home-grown cereals for all the known Fusarium mycotoxins using the most sensitive methods available. To be done over several years to include all climatic conditions which affect mycotoxin production.
Medium priority, medium input
9. An assessment of the significance of Fusarium mycotoxins in grain to establish "acceptable" levels of contamination for each mycotoxin or combinations of mycotoxins at which no harmful effects on man or livestock would be expected.
Medium priority, medium input

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APPENDIX 1

Fusarium: a list of books and papers.

- BOOTH C. (1971) The genus Fusarium. 237 pp. Commonwealth Mycological Institute, Kew (CAB).
(comprehensive account, classification, descriptions of species, many line drawings and photographs).
- BOOTH C. (1977) Fusarium - a laboratory guide to the identification of the major species. 55 pp. Commonwealth Mycological Institute, Kew (CAB) (brief descriptions, line drawings; keys).
- COLHOUN J. & PARK D. (1964) Fusarium diseases of cereals I. Infection of wheat plants with particular reference to the effects of soil moisture and temperature on seedling infection. Transactions of the British Mycological Society 47, 559-572. (includes a useful simple guide to the identification in culture of Fusarium spp. from cereals).
- C.M.I. Descriptions of Pathogenic Fungi and Bacteria. Commonwealth Mycological Institute, Kew (CAB).
- No. 25 Fusarium avenaceum (1964)
No. 26 Fusarium culmorum (1964)
No. 308 Fusarium poae (1971)
No. 309 Micronectriella nivalis (Fusarium nivale) (1971)
No. 384 Gibberella zeae (Fusarium graminearum) (1973)
- GERLACH W. & NIRENBERG H. (1982) The genus Fusarium - a pictorial atlas. Mitteilungen aus der Biologischen Bundesanstalt für Land- und Forstwirtschaft. Berlin-Dahlem. Heft 209. 406 pp.
(in English; descriptions of over 90 species and varieties with comment and many excellent photographs and line drawings; no keys).
- JOFFE A.Z. (1986) Fusarium Species: Their Biology and Toxicology. 588 pp. Wiley, New York. (expensive but important reference for toxins; not good for taxonomy).

- MARASAS W.F.O., NELSON P.E. & TOUSSOUN T.A. (1984) Toxigenic Fusarium Species, Identity and Mycotoxicology. 328 pp. Pennsylvania State University Press, University Park and London. (a companion volume to Nelson, Toussoun & Marasas (1983); comprehensive account of toxigenic Fusarium species in relation to incidence and distribution, diseases of humans and animals, toxicity to experimental animals and mycotoxins produced).
- NELSON P.E., TOUSSOUN T.A. & COOK R.J. (1981) Fusarium: Diseases, Biology and Taxonomy. 457 pp. The Pennsylvania State University Press, University Park and London. (review articles on most aspects of Fusarium).
- NELSON P.E., TOUSSOUN T.A. & MARASAS W.F.O. (1983) Fusarium Species: An illustrated manual of identification. The Pennsylvania State University Press, University Park and London. 193 pp. (excellent descriptions with photographs, keys).
- NIRENBERG H.I. (1981) A simplified method for identifying Fusarium spp. occurring on wheat. Canadian Journal of Botany **59**, 1559-1609. (keys, brief descriptions and relevant photographs, a method designed to allow the development of stable features so that each species or variety can be distinguished).

Fusarium species isolated from barley grain at harvest in England and Wales in 1974.

Species	No. of isolates	No. of isolates producing zearalenone
<u>F. culmorum</u>	431	270
<u>F. graminearum</u>	44	3
<u>F. avenaceum</u>	63	1
<u>F. moniliforme</u>	8	2
<u>F. tricinctum</u>	32	-
<u>F. poae</u>	41	-
<u>F. sporotrichioides</u>	20	-
<u>F. semitectum</u>	5	-
<u>F. nivale</u>	21	1
<u>F. sambucinum</u>	3	1
<u>F. fusarioides</u>	76	-
<u>F. tabacini</u>	6	-
Unidentified	37	-

Incidence of Fusarium mycotoxins detected in home-grown wheat and barley.

		Origin	No. of samples analysed	DAS	No. of samples positive							
					DON	FNX	HT-2	NEO	NIV	T-2	ZEN	
<hr/>												
1980												
	Feed Barley	England	43	-	22	-	-	-	-	-	-)(1)
	Malt Barley	England	42	-	13	-	-	-	-	-	-)
1980												
	Wheat	UK	34	0	1	0	0	0	0	0	0)
1981												
	Wheat	UK	95	0	4	0	0	0	0	0	0)
1982												
	Wheat	UK	70	0	27	0	0	0	0	0	0)(2)
	Wheat	England/ Wales	55	0	6	3	-	-	0	0	-)
	Wheat	England	10	-	-	-	-	-	-	-	0)(3)
1984												
	Wheat	England	31	-	20	-	-	-	17	-	4)
	Wheat	Scotland	3	-	1	-	-	-	1	-	3)(4)
	Barley	Scotland	8	-	4	-	-	-	2	-	8)

DAS = Diacetoxyscirpenol

(1) Gilbert et al, 1983

DON = Deoxynivalenol

(2) Osborne & Willis, 1984

FNX = Fusarenon X

(3) MAFF, 1987

HT-2 = HT-2 toxin

(4) Tanaka et al, 1986

NEO = Neosolaniol

NIV = Nivalenol

T-2 = T-2 toxin

ZEN = Zearalenone

Occurrence of nivalenol, deoxynivalenol and zearalenone in 1984
harvest of home-grown wheat.

Sample origin	Nivalenol (ug/kg)	Deoxynivalenol (ug/kg)	Zearalenone (ug/kg)
England	60	4	ND
"	ND	ND	ND
"	4	11	ND
"	ND	58	ND
"	ND	9	ND
"	670	ND	ND
"	18	ND	ND
"	45	ND	2
"	94	9	ND
"	330	8	ND
"	ND	31	ND
"	ND	ND	ND
"	19	32	1
"	ND	312	ND
"	31	26	ND
"	8	18	ND
"	ND	11	ND
"	ND	31	ND
"	200	4	ND
"	ND	ND	ND
"	32	4	ND
"	ND	ND	ND
"	4	9	ND
"	22	4	ND
"	ND	10	ND
"	ND	ND	3
"	27	6	ND
"	20	ND	1
"	ND	29	ND
"	148	ND	ND
"	ND	ND	ND
Scotland	ND	26	10
"	ND	ND	3
Percent positive	<u>52</u>	<u>64</u>	<u>18</u>
Mean in positives ug/kg	101	31	3

ND - Not detected