Contract report for the

Horticultural Development Council

Cucumber, sweet and chilli pepper, and tomato:
an assessment of current problems and future risks of
Fusarium **diseases in hydroponic crops, and priorities for research**

PC 232

November 2005

Project number:	PC 232
Title:	Cucumber, sweet and chilli pepper, and tomato: an assessment of current problems and future risks of *Fusarium* diseases in hydroponic crops, and priorities for research
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Report:	Annual report, November 2005
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Date project commenced:	1 April 2005
Date project completed:	30 November 2005
Key words:	cucumber, pepper, tomato, hydroponic crops, Fusarium wilt, Fusarium stem rot, *Fusarium oxysporum, Fusarium* species.

AUTHENTICATION

I declare that this work was done under my supervision according to the procedures described herein and that the report represents a true and accurate record of the results obtained.

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1. GROWER SUMMARY

1.1 Headline

This project has critically reviewed fusarium wilts and rots of cucumbers, peppers and tomatoes. Potential control measures are outlined. Priorities for R&D to aid effective and sustainable control of diseases caused by *Fusarium* species are identified.

1.2 Background and expected deliverables

In the HDC 2004 Pesticide Gap Analysis, fusarium wilt of cucumber and fusarium stem and fruit rot of chilli pepper were identified as critical gaps (***) and fusarium wilt of tomato and fusarium stem and fruit rot of sweet pepper as important gaps (**). The UK experience of these diseases at the start of this review is summarised below.

1.2.1 <u>Cucumber</u>

Sporadic severe losses to a fusarium disease have occurred in substrate crops over the last decade, in which plants wilt rapidly and die. Abundant sporulation of fusarium occurs at stem nodes and often there is rapid disease spread throughout a house. The problem appears to be a vascular wilt disease, most probably caused by *F. oxysporum*. Some nurseries appear to have this disease more regularly than others possibly indicating carryover on the nursery and/or a growing environment favourable to the disease. The current fungicide treatment (carbendazim drench) appears to offer limited control while swift removal of affected plants can limit the rate of spread. The sensitivity of fusarium isolates from cucumber to carbendazim is unknown. Carbendazim treatment is not favoured by retailers and spray treatments can cause crop damage and interfere with biological pest control

1.2.2 <u>Sweet pepper and chilli pepper</u>

First described in the UK in 1986, fusarium stem and fruit rot of sweet pepper caused by *F. solani* remains an occasional problem. The disease causes stem rotting at nodes and a fruit end rot. Recently, a different, as yet unidentified fusarium disease of pepper has been noted, causing stem, fruit stalk and internal fruit rot. The problem is reported to be increasing.

1.2.3   <u>Tomato</u>

Most modern tomato cultivars grown in the UK have genetic resistance to races 0 and 1 of fusarium wilt, caused by *F. oxysporum* f. sp. *lycopersici* (*Fol*). Nevertheless, in recent years *F. oxysporum* has been recovered from the roots (commonly) and stem vascular tissue (occasionally) of diseased plants in ADAS and STC laboratories. The role of these isolates in causing disease is unclear. In 2004, one crop in Hertfordshire suffered a severe vascular wilt disease. Samples were sent to the Netherlands and the problem was reported to be due to American race 3 of *F. oxysporum* f. sp. *lycopersici*. There is no published report of this race occurring in the UK[1]. In September 2004, a rockwool crop of cv. Classy developed symptoms suggestive of a vascular wilt disease; severe symptoms were present in around 3% of plants and *F. oxysporum* was consistently recovered from vascular tissue.

Fusarium crown and root rot caused by *F. oxysporum* f. sp. *radicis-lycopersici* occurs most years in the UK, but only in heritage varieties lacking genetic resistance, and here it is sometimes very damaging.

The objectives of this review are:

1. To assess the risk to UK protected cucumber, sweet pepper, chilli pepper and tomato crops from fusarium pathogens
2. To provide current, best-practice guidelines to manage the three most important fusarium diseases present in the UK (fusarium wilt in cucumber and tomato; fusarium stem and fruit rot in sweet pepper)
3. To identify priorities for research in order to improve the management of these diseases.

1.3   Summary of review and main conclusions

1.3.1   <u>How new disease problems occur</u>

The spectrum of diseases that affects a particular crop changes with time, usually in a sporadic and unpredictable manner. Some diseases effectively disappear, for example

as resistant varieties are introduced or the crop production system is changed. Other diseases, previously unreported or new to the crop in the UK occur sporadically. There are three main ways in which a new disease can occur:

A pathogen extends its geographic distribution. This may occur when a pathogen is inadvertently introduced into the country by man (e.g. on seed or transplants); or when it spreads here by natural means (e.g. as spores in the air; or on birds` feet). Fusarium crown and root rot of tomato is considered to have been introduced into several countries on seed. Outbreaks of cucumber downy mildew in England have been attributed both to importation of infected transplants, perhaps in a latent stage and long-distance aerial dispersal of spores from the Continent.

New strains of a pathogen evolve or the host resistance breaks down. A resistant variety can only be said to be resistant to the strains (or races) of the pathogen to which it has been exposed and found to be resistant in a particular (realistic) test procedure. Other strains of a fungus may exist that are pathogenic to the host and are present in the country but have not previously come into contact with the host. This appears to be the explanation for recent outbreaks of Verticillium wilt caused by *Verticillium albo-atrum* in tomato carrying the *Ve*-resistance gene. Alternatively, and especially if resistance is dependant on a single widely-utilised gene, the pathogen may evolve; strains able to overcome the resistance are selected out from the natural, diverse and changing variation present in a population.

A disease totally new to this country and elsewhere is recognised. Fusarium fruit and stem rot of pepper caused by *Fusarium solani* was recorded for the first time anywhere as a disease in the UK. *Phytophthora kernoviae* has recently been recorded for the first time anywhere on rhododendron in Cornwall. The origins of these totally new diseases are individual and subject to debate. The pathogen may have extended its range from a closely-related host (e.g. from potato to tomato); or breeding programmes may have changed the host-resistance status to a minor or previously undescribed pathogen, or the disease may previously have been only a

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[1] The nomenclature of *Fol* races on tomatoes differs between countries. See section 3.1.1. for details.

minor problem and overlooked; or a new pathogen may have evolved by the crossing of two closely-related species.

Current and potential new disease problems are summarised below by crop.

1.3.2   Cucumber

Fusarium wilt (*F. oxysporum* f. sp. *cucumerinum*)

This disease was previously quite common in soil-grown cucumber. The main control methods were by grafting onto resistant rootstocks (e.g. *Cucurbita ficifolia*) and fungicide (e.g. benomyl[2], carbendazim) drenches. A sporadic and sometimes severe fusarium wilt and stem rot that currently affects rockwool-grown crops has been assumed to be caused by the same fungus although there is no evidence to confirm this. The possibility that some reported outbreaks are not fusarium wilt but fusarium root and stem rot caused by *F. oxysporum* f. sp. *radicis-cucumerinum* (*Forc*) (see below) requires investigation.

Three races of *Foc* have been reported. It is not known which of these are present in the UK. A major gene for resistance to *Foc* is closely-linked to a gene (*Ccu*) which confers resistance to scab (*Cladosporium cucumerinum*); most varieties of long and short cucumber contain the *Ccu* gene for scab resistance and are also resistant to fusarium wilt.

Carbendazim drenches offer only limited control. The sensitivity of current strains of *Foc* to carbendazim and other fungicides is unknown. A range of alternative methods have been demonstrated to provide some suppression of fusarium wilts of cucurbits; these include the use of essential oils and plant extracts, chitinolytic bacteria (stimulated by chitosan), compost-tea[3] and a *Pseudomonas putida* seed treatment. Swift removal of affected plants as soon as the disease is detected can limit the rate of spread in both soil-grown and hydroponic crops). However, once the fungus sporulates along the stem then there is a potential risk of air-borne transmission.

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[2] Use of benomyl e.g. Benlate is now revoked in the UK and products containing this active substance are no longer approved for use.
[3] Compost teas are liquids with high microbial populations resulting from the addition of compost to water and infusion with oxygen for a period.

Fusarium root and stem rot (*F. oxysporum* f. sp. *radicis-cucumerinum*)

This newly described disease has caused substantial losses in Greece and Canada. Although not yet confirmed in the UK, it has been described in the Netherlands and France. It is not a major disease in the Netherlands, possibly because most crops are grown in inert (and isolated) growing systems where nursery hygiene conditions tend to be very high.

The temperature optimum for development of cucumber fusarium root and stem rot (17°C) is lower than that of fusarium wilt (29°C). Seed infection is suspected but has not been demonstrated. The disease can be introduced into a glasshouse in infected seedlings, subsequent spread occurs via irrigation water, insects (shore flies and fungus gnats), workers clothing and equipment. Infection that occurs during propagation is particularly important as older plants are less susceptible. Plants wilt at fruiting, especially during hot weather when the plants have a high fruit load.

Grafting onto resistant rootstocks was reported as the only effective means of control in Greece. Varieties differ in susceptibility. Where there is a risk of fusarium root and stem rot, a high standard of nursery hygiene is recommended and crops should be carefully monitored for the disease. Some formulated biocontrol products (Prestop WP, Rootshield Drench, Mycostop) have given control in glasshouse trials though efficacy was affected by seasonal differences in growing conditions. Other cucurbits are susceptible to this disease, but not pepper or tomato.

Fusarium crown and foot rot (*F. solani* f. sp. *cucurbitae*)

This disease is reported in the USA, where it causes a problem on squash and pumpkin. Cucumbers are susceptible but less affected. The fungus survives in the soil and on seed. There have been no confirmed reports of this disease on any cucurbit crop in the UK.

1.3.3 <u>Sweet and chilli pepper</u>

Fusarium wilts (*F. oxysporum* f. sp. *vasinfectum* and a proposed *F. oxysporum* f. sp. *capsici)*

Convincing reports of fusarium wilt in pepper are rare. Two forms of *F. oxysporum* are reported cause wilt, neither of which has been confirmed in the UK. *F. oxysporum* f. sp. *vasinfectum* is described in the Argentina, Italy, Mexico and the USA on sweet and chilli peppers, where it causes a root and stem base decay, stem canker and wilt. A wilt caused by a proposed *F. oxysporum* f. sp. *capsici* (sp. nov.) was reported in the USA (Louisiana) on Tabascum pepper, and subsequently in India on chilli pepper and other *Capsicum* species. This fungal name is not officially recognised. The main symptoms are leaf yellowing wilt and vascular discolouration. Cucumber and tomato were not susceptible.

In soil-grown crops, fusarium wilt of chilli pepper is worse in poorly drained areas. Varieties of chilli pepper resistant to the form known as *F. oxysporum* f. sp. *capsici* are available in India. Seed treatments with carbendazim and *Trichoderma viride* are reported to be effective against this fungus. Compost-tea is reported to be effective against *F. oxysporum* f. sp. *vasinfectum* when applied as a drench to glasshouse soil.

Fusarium solani fruit and stem rot

A fruit and stem rot of sweet pepper caused by *F. solani* was first described in the UK in 1992, more recently in Canada and New Zealand. The main symptoms are node lesions on the stem and fruit decay (generally from the calyx end). Fruit decay is usually a dry rot leading to tissue collapse and the development of white, cream, pink or orange-red spore pustules. In Florida, the disease caused wilting and death of upper portions of the plant but no fruit rot symptoms. Symptom development in Canada was reported to be triggered by a high fruit load, adverse growing conditions (high humidity) and senescence.

Control is based on good hygiene (knives and hands), clean cutting of leaves, shoots and fruit rather than breaking or tearing, preventing damage at the stem base of plants grown in rockwool such as that caused by salt accumulation, controlling the glasshouse climate to avoid extended periods above 95 % RH, and avoiding high temperatures. Removal of all debris and disinfection of the glasshouse and equipment

(chlorine dioxide and QAC[4] found to be effective) are also important to minimise the risk of persistence between crops. No suitable fungicides have been identified. In the UK, the variety Tasty was affected more severely than Mazurka.

Fusarium oxysporum **fruit and stem rot.**

A fruit and stem rot of sweet pepper associated with *F. oxysporum* has recently been noted in the Lea Valley (2005) and in the Netherlands; so far it has been little investigated. The main symptoms are fruit rot often developing from the blossom end; vascular discolouration in the fruit stalk, a lesion on the fruit stalk at its junction with the stem; stem node lesions and spreading stem lesions. *F. oxysporum* was also found sporulating on aborted fruit in the crop. It appears probable that the disease has occurred on at least one nursery for several seasons. Cutting out stem lesions was successful in preventing development of girdling lesions and plant death. A new HDC funded project on this disease commenced in July 2005 (PC 232a).

Fusarium circinatum **fruit rot**

A rot of sweet pepper fruit caused by *F. circinatum* was recently recognised in Canada. It has not been reported elsewhere. Symptoms, which only occurred on mature fruit close to harvest, consisted of discoloured fruit with soft patches and necrotic spots, predominantly at the calyx end. Inoculation of just-opened and fully-opened flowers with spores of *F. circinatum* produced more symptomatic fruit than inoculation of developing fruit. Although *F. circinatum* has a wide host-range (e.g. maize, mango, pine) no disease developed in aubergine, cucumber, lettuce and tomato following artificial inoculation with spore suspensions.

1.3.4   Tomato

Fusarium wilt (*F. oxysporum* f. sp. *lycopersici*)

Fusarium wilt of tomato caused by (*F. oxysporum* f. sp. *lycopersici (Fol)* is currently a relatively rare disease in the UK. At least three different races exist. Their characterisation is notoriously complex and confusing, in part due to the use of two different numbering systems. Most modern cultivars have genetic resistance to races 0 and 1 of the pathogen (American nomenclature races 1 and 2) and this has proved

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[4] QAC – quaternary ammonium compound.

effective for many years. A third race (known as race 2 in the UK; race 3 in American nomenclature) that overcomes the *I-2* resistance gene, has been reported in Australia, Brazil, Florida and Mexico. The resistance of cultivars commonly grown in the UK to different races of fusarium wilt requires clarification.

Fusarium wilt generally causes more yellowing of leaves and discolouration of stems than Verticillium wilt. There is usually a pronounced dark brown staining of the vascular tissue, which may be visible externally and extend 1.5 m or more up the stem, distinguishing the disease from both Verticillium wilt (generally a pale brown vascular staining) and fusarium crown and root rot (localised dark brown staining of the vascular tissues around the stem base). The disease is favoured by high temperature (soil temperature >28°C), with little disease progress below 21°C.

Seeds and transplants can be a source of the disease. Spread may occur via spores in the re-circulating irrigation water of 'closed' hydroponic crops and in air. The pathogen does not attack cucumber or pepper. Rootstocks (KVF, KVFN) provide effective control of races 0 and 1, and new rootstocks (Oxyfort, Trifort and Zodiac) are reported to be resistant to race 2 (American nomenclature, race 3) although the type of resistance is unknown. Fungicides provide only partial control. Several micro-organisms give some biological control, as does induced resistance.

Fusarium crown and root rot (*F. oxysporum* f. sp. *radicis-lycopersici*)

Fusarium crown and root rot was a problem in UK crops in the late 1980s but the disease is now seen only rarely, usually on heritage varieties that lack resistance to *F. oxysporum* f. sp. *radicis-lycopersici* (*Forl*). The disease, where it occurs in such varieties, can be a serious problem both in soil-grown and hydroponic crops. Symptoms are usually first seen when the first fruit are near maturity although infection is likely to have occurred many weeks earlier. Infected plants are often stunted, wilt (at first temporarily) and develop a chocolate-brown lesion at the stem base. The roots develop a dry, brown rot, similar to that caused by *Phytophthora*.

Disease spread occurs by conidia dispersed in the air, fungus gnats (*Bradysia* spp.) and by root contact. Long-distance spread may occur on symptomless or mildly

infected plants and in soilless media. In Israel, spread has also been found on the seeds of a weed host.

Resistance to *Forl* is conferred by a single dominant gene. Isolates of *Forl* able to overcome resistance in the variety Trust were reported in 1997 in four greenhouses in Canada. No subsequent reports of new strains of *Forl* in Canada, or elsewhere, were found. Hygiene is important for effective control where a susceptible variety is being grown. The production of disease-free transplants is critical. The fungicide carbendazim and various micro-organisms, alone and in integrated treatments, have been demonstrated to give partial control.

Fusarium stem rot (*F. merismoides*)

A fusarium stem rot caused by *F. merismoides* occurred in Kent in 1984. It was widespread during one season on one nursery, but has not been reported in the last 10 years. Symptoms were reddish brown stem lesions (2-16 x 2 cm) with a dark edge. They were slightly sunken and did not penetrate more than 2 mm into the stem. The lesions did not result in plant death. Pathogenicity of *F. merismoides* to tomato was confirmed when fresh leaf scars were inoculated. This fungus has also been associated with diseases of other crops including potato.

Table 1.1. Causal agents of fusarium diseases of cucumber, sweet and chilli pepper and tomato and their current status (occurrence) in the UK

Crop and disease	Pathogen	Present in the UK
Cucumber		
Wilt	*F. oxysporum* f. sp. *cucumerinum*	Yes
Root and stem	*F. oxysporum* f. sp. *radicis-cucumerinum*	No
Crown and foot rot	*F. solani* f. sp. *cucurbitae*	No
Sweet and chilli pepper		
Wilt	*F. oxysporum* f. sp. *vasinfectum*	No
	F. oxysporum f. sp. *capsici**	No
Fruit and stem rot	~~1.3.4.1.1.1.1.1.1~~*F. solani*	Yes
	~~1.3.4.1.1.1.1.1.2~~*F. oxysporum*	Yes
Fruit rot	*F. circinatum*	No
Tomato		
Wilt**	*F. oxysporum* f. sp. *lycopersici* race 0	Yes
	F. oxysporum f. sp. *lycopersici* race 1	Yes
	F. oxysporum f. sp. *lycopersici* race 2	?
Root rot	~~1.3.4.1.1.1.1.1.3~~*F. oxysporum*	Yes
Crown and root rot	*F. oxysporum* f. sp. *radicis-lycopersici*	Yes
Stem rot	*F. merismoides*	Yes

* Not officially recognised as a valid name.

** The nomenclature of *Fol* races on tomatoes differs between countries. See section 3.1.1. for details.

Table 1.2. A simple risk-assessment of fusarium diseases of cucumber, sweet and chilli pepper and tomato.

Crop and disease	Pathogen	Present in UK	Importance in countries where it occurs	Can it be controlled?	Is this likely to become ineffective	Potential risk for UK crops
Cucumber						
Wilt	*F oxysporum* f. sp. *cucumerinum*	Yes	Moderate	Yes (resistant varieties	Possibly	Moderate
Root and stem rot	*F oxysporum* f. sp. *radicis-cucumerinum*	No	Very (Canada, Greece)	Yes (grafting)	Possibly	Moderate (minor problem in Fr & NL)
Crown and foot rot	*F. solani* f. sp. *cucurbitae*	No	Minor on cucumber; potentially important on outdoor cucurbits	?	?	Low on cucumber. Moderate on squash, pumpkin
Sweet and Chilli pepper						
Wilts	*F. oxysporum* f. sp. *vasinfectum* and f. sp. *capsici*	No	Moderate	Yes (resistant varieties; seed treatment)	Possibly	Low?
Fruit and stem rot	*F. solani*	Yes	Moderate	Yes (hygiene cultural)	No	Low
Fruit & Stem Rot	*F. oxysporum*	Yes	Increasing (Holland)	Partially	-	Moderate
Fruit rot	*F. circinatum*	No	Moderate (Canada)	?	-	Moderate
Tomato						
Wilt	Fol (races 0 &1)	Yes	Low – moderate	Yes (resistant varieties	Possibly	Low
	Fol (race 2)	No?	High	Partially (rootstocks)	Possibly	Moderate
Crown and root rot	*F. oxysporum* f. sp. *radicis-lycopersici*	Yes	Moderate	Yes (resistant varieties	Possibly (1 report)	Moderate
Root rot	*F. oxysporum*	Yes	Low	-	-	Low/moderate
Stem rot	*F. merismoides*	Yes	Low	Partially	?	Low

1.4   Benefits to the industry

- A greater awareness of fusarium disease problems, some of which are difficult to diagnose and may be mistaken for other problems.

- Increased understanding of the diseases.

- A summary of control measures for key diseases

- Ability to plan future research and development on fusarium diseases based on sound knowledge and an assessed risk.

1.5   Best-practice guidelines for disease control

1.5.1   Cucumber wilt

- Ensure that the cause of any wilting is diagnosed correctly and promptly. Abundant pink or orange sporulation at stem nodes serves to distinguish fusarium from verticillium wilt but only occurs when fusarium is well established in a plant. Be aware that occasionally fusarium develops as a secondary infection, and may mask the primary cause of wilting

- Susceptible varieties can be grafted on to the resistant rootstock *Cucurbita ficifolia*, and this may be justified where there is a persistent problem. This method was successful for control of fusarium wilt in soil-grown crops. Resistant varieties are not available.

- If a disease outbreak is spotted at an early stage, all affected plants should be carefully removed, sealed in a bag, taken out of the crop, and disposed of away from the nursery.

- In substrate crops either ensure effective disinfection or replace the growing medium. For soil crops steam sterilisation may be the only effective option available to eliminate the pathogen from the soil. This is only necessary if resistant rootstocks/varieties are not being used.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop.

- Drenches of carbendazim (SOLAs 1479/1995, 1005/2004, 1211/2004 and 1824/2005) should give some suppression of the disease but should not be relied on alone due to the risk of possible resistance development following repeated use.

- In closed growing systems (i.e. situations where hydroponic run-off solution is being collected and re-used), do not recycle without adequate disinfection. Also, even if the solution is not currently being re-used, check that the plants are not rooting out from the drainage slits in the substrate slab into the channel beneath. There are several technologies available for disinfecting hydroponic solution; seek specialist advice.

1.5.2   Pepper fruit and stem rots

Current recommended control measures for the fruit and stem rots caused by *Fusarium solani* and *F. oxysporum* are similar.

- Maintain a high level of hygiene in the glasshouse, with particular attention to regular removal of fallen and aborted fruit. Abundant sporulation of *F. oxysporum* was found on aborted fruit in one crop.

- Ensure fruit, leaves and axillary shoots are removed with a clean sharp knife. Evidence from Canada indicates a greater disease risk of *F. solani* stem rot where torn, ragged wounds are present.

- Where fusarium stem rot has led to irreversible wilting or extensive stem lesions, remove the affected plants from the crop promptly and carefully.

- Where fusarium is a widespread and persistent problem, cut out nodal lesions. Also, during periods of the year when the disease is increasing, consider re-cutting all stem wounds (i.e. including those where there are no visible lesions) in order to remove any incipient lesions.

- Wash and disinfect hands and knives and any other equipment used after handling infected material.

- Cover any containers containing reject fruit or crop debris to minimise the risk of aerial spread or insect transmission from fusarium sporulation on crop waste.

Empty debris containers regularly and clean them as required.

- Position the dripper so that there is no build up of 'salts' around the stem base. Infection is reported to develop in tissues damaged by fertiliser scorch.

- Monitor irrigation carefully so that there is no excessive watering. There are reports that excessive watering may stress plants through oxygen depletion, resulting in *F. solani* shoot lesions.

- Control the glasshouse environment to avoid prolonged periods above 90% RH. This has been shown to reduce ascospore germination of *Nectria haematacocca*, the sexual stage of *F. solani* that is sometimes found on infected plants.

- Seek to grow a balanced crop and avoid environmental extremes. Symptom development is reported to be triggered by various `stress` factors, including a high fruit load and high temperatures.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop. It is essential that all debris is removed from the house and taken away from the nursery or destroyed.

- In conventional (non-organic) crops it is possible that broad-spectrum fungicides applied for the control of other foliar pathogens e.g. azoxystrobin (Amistar) could help prevent infection, though further work would be required to demonstrate this.

1.5.3   Tomato wilt

- Ensure accurate laboratory diagnosis of the cause of any wilting. The symptoms of wilt caused by *F. oxysporum* f. sp. *lycopersici* are not easily distinguished from those of verticillium wilt, fusarium crown and root rot or even *Phytophthora* root rot.

- There are a number of races of *F. oxysporum* f. sp. *lycopersici*. Where possible, choose a variety or rootstock with resistance to races 0 and 1; both of these races are known to be present in the UK. Use of a resistant variety is likely to be the most effective means of disease control.

- If the third race of *F. oxysporum* f. sp. *lycopersici* is confirmed in the UK, consider using grafted plants on a rootstock reported to be resistant to race 2

(American race 3) (e.g. Oxyfort, Trifort and Zodiac).

- If feasible, avoid high temperatures in the glasshouse. Fusarium wilt develops best at high root temperatures (25-28°C).

- Fungicide drench treatment to the roots with carbendazim (SOLAs 1479/1995, 1005/2004, 1211/2004 and 1824/2005) may give some control in conventional (non-organic) crops, but this treatment is likely to be only partially effective. Before application growers are advised to check that the use is permitted by any supermarket being supplied.

- Where irrigation water is abstracted from an uncovered reservoir, where drainage water is collected and recycled, and especially where there is considered to be a high risk of fusarium wilt, ensure that the irrigation water is treated to kill fusarium spores. Experimental work indicates that heat, UV light, ozone and microfiltration, when operating effectively, are effective against the pathogen and can largely prevent dissemination. Slow sand filtration may also be effective though further work is required to determine this.

- Carefully remove infected plants from the crop before any sporulation occurs on the stem base. Seal affected plants in a bag before taking them through the crop. Although sporulation on stems is rare, if it occurs it could lead to extensive disease spread through aerial dissemination of spores.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop. *F. oxysporum* f. sp. *lycopersici* has been isolated from the glasshouse structure, presumed to have arisen from aerially dispersed spores. Resting spores develop in affected tissue and can persist for several years, therefore it is important to remove all crop debris from the site.

- Seed suppliers should routinely monitor seed crops for freedom from fusarium pathogens and only harvest seed from crops found to be free of the disease. Seed-borne infection is known to occur and is a possible source of new infection.

2. SCIENCE SECTION

2.1 Cucumber

2.1.1 Fusarium wilt

2.1.1.1 <u>Introduction</u>

Vascular wilt diseases of cucumber can be caused by three different fungal pathogens, *Fusarium oxysporum* f. sp. *cucumerinum, Verticillium dahliae and V. albo-atrum,* all of which give similar symptoms. Of these, fusarium wilt can spread more quickly and is more damaging than verticillium wilt which rarely affects more than a few plants (ADAS, 1980a). There are also bacterial wilt pathogens, e.g. *Erwinia tracheiphilum*, which could potentially be confused with fungal wilts, though at present they are not known to occur in UK crops. Verticillium and bacterial wilts are not reported further here.

Fusarium wilt occurs in several countries including Australia, China, Germany, Greece, Israel, Japan, the Netherlands, the UK and the USA. There are three races reported, race 1 is predominant in the USA, race 2 is more common in Israel and race 3 occurs in Japan. No information appears to be available on the race(s) occurring in the UK. Except in small, localised areas, it is not of major economic importance in most countries, probably because of the use of resistant cultivars (Zitter *et al.*, 1996).

In the UK, fusarium wilt (*F. oxysporum* f. sp. *cucumerinum*) (*Foc*) was not seen as a problem in cucumber crops following the move into soilless, hydroponic, growing systems in the late 1970's and 80's and the disease was all but forgotten. Over the last decade however, sporadic severe losses have occurred in some UK crops grown on rockwool slabs in which plants rapidly wilted and died. Some nurseries appear to have this disease more regularly than others do. ADAS and STC laboratories have received occasional samples and when investigated we have recovered a *Fusarium* species, fully conforming to *F. oxysporum* from stained vascular tissues of the host. No attempt has been made to type the *Fusarium* or to check pathogenicity to cucurbits through artificial inoculation.

The relative susceptibility of current commercial cucumber varieties to *Fusarium* wilt (including different races of the pathogen) is not known. There is some concern that an increase in organic production, where crops are soil-grown, may again increase the prevalence of this pathogen in UK crops and a greater awareness of the potential of the pathogen to cause crop loss is required. Similarly, if growers move to all-year - round hydroponic production, this could exacerbate fusarium root and vascular diseases.

2.1.1.2 Symptoms of the disease

Initial infection usually takes place via the roots with subsequent invasion of the stem vascular tissues. The fungus can attack cucumbers at any stage of plant development. Damping-off of seedlings is common, particularly in cool soils (18 - 20°C), and may occur pre-emergence. On mature plants there is rapid disease development, with wilting accentuated by high temperatures, drought stress and by fruit load (Zitter *et al.*, 1996). Symptoms may include:

- Wilting of the lower leaves during the day with some recovery at night. This is usually the first symptom observed on mature plants.
- Wilting of one or more shoots.
- More leaves become affected until eventually the whole plant wilts.
- Plants may die within 3 to 5 days of the first wilt symptoms.
- Brown discolouration of the vascular tissue in stems and roots of wilted plants. This is most obvious if the stem is cut across a node.
- Orange coloration at stem nodes, due to sporulation of the fusarium pathogen.*
- Rapid disease spread throughout a house may be characteristic of the pathogen.*

* Symptoms seen in recent outbreaks of fusarium on rockwool grown cucumber.

2.1.1.3 Disease spread

Soil

Fusarium is primarily soil-borne, though outbreaks are not restricted to crops grown in soil. It survives as chlamydospores in the soil and saprophytically on plant debris and other organic matter (Zitter *et al.*, 1996).

Seed

Foc has been shown to be externally seedborne and capable of surviving on stored seed for at least a year. Dissemination, however, occurs primarily by movement of infested soil and infected plant material (Zitter *et al.*, 1996) and probably also by contaminated water.

2.1.1.4 Risk to other protected vegetable crops

Most isolates specifically infect cucumber though several isolates from the Netherlands also are pathogenic to watermelon and melon (Zitter *et al.*, 1996).

Muskmelon wilt caused by a *Fusarium* species was observed in field crops of *Cucumis melo* L. var. *cantalupensis* in British Columbia, in Canada in 1999. Symptoms were leaf yellowing and wilting. Dark brown lesions on the crown spread into lateral branches and into the fruit so that it frequently rotted. Stem lesion necrosis extended into the cortical and vascular regions, and drops of a dark brown exudate were produced. High humidity incubation of the crown and fruit produced white mycelium. This was found to be *Fusarium oxysporum* f. sp. *melonis,* race 1, rather than *F. oxysporum* f. sp. c*ucumerinum*. Seeds collected from naturally infected mature melon plants in the field were found to be harbouring the muskmelon wilt pathogen (Punja *et al.,* 2001).

2.1.1.5 Control of cucumber fusarium wilt

Resistance
Susceptible cultivars can be grafted onto a resistant rootstock (ADAS, 1980a) though generally this practice is not widely undertaken now in the UK. This is an effective (non-chemical) method for fusarium wilt control. The best resistant rootstocks in Greece are *Cucurbita ficifolia* Bouche, *C. moschata* (Duchesene) Duchesene ex Poir. and *C. maxima* X *C. moschata*, the last being the highest yielding under the climatic conditions in Crete (Vakalounakis, 1999).

Resistance of cucumber to fusarium wilt is controlled by a single dominant gene (*Foc*). The *Foc* gene is linked to the *Ccu* gene, which confers resistance to cucumber scab (caused by *Cladosporium cucumerinum*) (Zitter *et al.,* 1996). Most 'Dutch' long

and short cucumbers should be resistant to fusarium wilt, since they possess the *Ccu* gene (Vakalounakis, 1999).

Fungicides

Fungicide treatments (carbendazim drench on inert substrate crops) appear to offer only limited control, suppressing rather than controlling fungal activity. The sensitivity of fusarium isolates from cucumber to carbendazim and other fungicides is unknown. Carbendazim drench treatments can cause crop damage and spray treatments can interfere with biological pest control. The original recommendation for soil-grown or straw-bed crops was an MBC (benomyl[5]) drench, with a follow-up application after 28 days applied to plants that remain in the house after the removal of infected plants (ADAS, 1980a). Currently, use of carbendazim is permitted as a drench on crops of cucumber grown in inert growing media under SOLAs 1476/2005 (Bavistin DF), 1005/2004 (Delsene 50 Flo), 1211/2004 (Cleancrop Curve) and 1824/2005 (Clayton Chizm FL).

Soil fumigation with methyl bromide was used against fusarium, but is no longer permitted. Commercial formulations of botanical extracts and essential oils are being investigated as possible alternatives for the control of fusarium wilt diseases of other cucurbits. Muskmelon sown in wilt (*F. oxysporum* f. sp. *melonis)* infested soil amended with botanical extracts allowed 80% of crop plants to remain healthy after five weeks, compared with 20% remaining healthy in an untreated control. The infested soil was treated with either 5% or 10% aqueous emulsions of either chilli pepper extract plus essential oil of mustard, or extract of cassia tree, or 70% clove oil. The surface was sealed with plastic for a week before being sown (Bowers & Locke, 2000).

Solarisation of field soil using impermeable plastic sheets has given satisfactory disease control in Crete (Vakalounakis, 1999). Experiments in plastic houses in Greece showed that covering the soil with impermeable plastic sheets (Plastopil Hazorea) to allow solarisation for 30 days almost eliminated *Foc* at a depth of 20 cm and significantly decreased disease incidence in cucumber. This was as effective as

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[5] Approval for benomyl (e.g. Benlate) has been withdrawn in the UK.

solarisation for 15 days and 35 g/m² of methyl bromide. Common polyethylene, rather than impermeable plastic, sheet also reduced disease incidence (Antoniou, 1999). Over many years of study in Greece it has been demonstrated that 4-6 weeks summer soil tarping with transparent polyethylene sheets effectively controls vascular wilts including cucumber wilt, and it is currently used against soilborne pathogens of vegetables in many parts of Greece. Recent research has shown that covering the soil with impermeable plastic after adding the bacterial antagonist *Bacillus* sp. reduces the period of solarisation necessary for control of cucumber wilt to 15 to 20 days (Tjamos *et al.,* 1999). The levels of solar radiation received make this technique impractical in the UK.

Biological control[6]

Two chitinolytic bacterial strains, *Paenibacillus* sp. 300 and *Streptomyces* sp. 385 were found to suppress fusarium wilt of cucumber, with a zeolite based chitosan-amended formulation at a rate of 6 g per kg of soilless potting medium providing the best protection against the disease. The formulation was added to pathogen-infested medium 15 days before planting cucumber seeds. The bacteria produce β-1,3-glucanase and chitinase, and these hydrolytic enzymes may be involved in the suppression of fusarium wilt (Singh *et al.,* 1999). The formulation was still effective after storage for six months at room temperature.

A compost-tea drench onto soil under glasshouse conditions controlled *Foc,* and *in vitro* tests showed that the tea had a mycolytic effect on fusarium microspores and chlamydospores (Diver, 1998).

Wilt of muskmelon, another cucurbit species, caused by *Fusarium oxysporum* f. sp. *melonis* was partially controlled by seed treatment with different strains of *Pseudomonas putida* in a wettable powder formulation. Strain 30 gave 63% efficacy, and strain 180 gave 46% efficacy, in field trials recorded at 90 days after

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[6] Use of bio-control products in the UK (and elsewhere in the EU) is regulated in the same way as conventional pesticides through the Control of Pesticides Regulations 1986 (as amended) (COPR) and the Plant Protection Products Regulations 1995 (as amended) (PPPR). Currently there are no biological control agents approved for use in the UK with specific activity against fusarium diseases of tomato, cucumber or pepper crops.

transplanting. This efficacy was better than that achieved with benomyl (Bora *et al.*, 2004). The effectiveness of this bacteria on cucumber wilt has not been reported.

Research in Canada has shown that the microbial control agents *Trichoderma harzianum* strain T-22 (in Rootshield Drench) and *Gliocladium catenulatum* strain J1446 are effective against root and stem rot of cucumber *F. oxysporum* f. sp. *radicis-cucumerinum*. Application was most effective when applied prior to the onset of infection, or just after the first appearance of symptoms (Rose *et al.,* 2003; Punja, 2004). However, control of fusarium wilt (*Foc*) was not investigated.

Cultural

Swift removal of affected plants as soon as the disease is detected can limit the rate of spread. The affected plants should be placed in a bag directly as they are cut down and carefully disposed of in a covered skip, or by another suitable means (e.g. burning).

In Spain, adding green compost from pruning waste to semi-arid agricultural soil greatly improved the growth of melons in soil infested with the wilt fungus *Fusarium oxysporum* f. sp. *melonis*, although there was no significant decrease in the level of fusarium in the soil. Microbiological and biochemical parameters were altered (Ros *et al.,* 2005). This suggests that the rhizosphere population and plant nutrition are of importance in producing a crop that can tolerate the presence of fusarium.

Liming the soil to raise the pH to 6.5-7.0 and applying nitrogen in the form of NO_3 is also reported to provide some control of the disease.

Hygiene

Special attention should be given to hygiene both during cropping and at the end of the crop. Thorough steam sterilisation is the most effective method of killing the pathogen in soil (ADAS, 1980a). Work with *Fusarium oxysporum* f. sp. *melonis*, an important glasshouse pathogen in Spain, has shown that this fungus was eliminated in waste plant material after two to three days of windrow composting. Laboratory tests showed that a compost temperature of 65°C killed the fungus swiftly, but at a lower temperature of 45°C elimination was only complete after 10 days (Suárez-Estrella *et al.,* 2003).

2.1.2 Fusarium root and stem rot

2.1.2.1 Introduction

Root and stem rot of cucumber caused by *F. oxysporum* f. sp. *radicis-cucumerinum* (*Forc*) is a new and devastating disease of greenhouse crops in Greece and some other countries (Pavlou, 2002). It was first reported in Crete in 1989, with severe crop losses occurring after three years (Rose *et al.,* 2003). It is now the most destructive disease in glasshouse cucumber crops in Crete and on the mainland of Greece (Vakalounakis, 1996: 1999). It was reported in British Columbia, Canada in 1994 and later in Ontario in 2000, causing 10% and 25–35% losses, respectively (Cerkauskas, 2001a). The pathogen has since spread to many glasshouses in British Columbia and continues to be a recurring problem for growers. It has also been reported form Israel, France and the USA (Rose *et al,* 2003) and the Netherlands (Cerkauskas, 2001a; Vakalounakis & Fragkiadakis, 1999). This disease has not been confirmed in the UK to date.

2.1.2.2 Symptoms of the disease

The symptoms described from glasshouse crops or rockwool grown cucumber in Canada (Cerkauskas, 2001a) include:

Wilting
- Wilting of plants at the fruit bearing stage and during hot weather.
- Plants turn brown and die, especially with high fruit loads and hot weather.

Stem lesions
- Yellowish-orange or buff discolouration of the outer tissues of the stem base, but no visible mycelium at this stage.
- Progressive upward colonisation of the stem, with the fungus being isolated beyond visible symptoms.
- Longitudinal cutting through the stem base shows the breakdown of cortical tissues, facilitated by secondary bacterial infection.

- Severely affected plants have a stringy stem with light-salmon or pinkish-orange spore masses and white fungal, cottony-like growth on the outside of the stem.

Roots

The roots of affected plants remain firm (but see comment below).

Rose *et al.* (2003) report that infected roots, crown and stem tissues rot and contain mycelium and spore masses of the pathogen.

The temperature optimum for development of fusarium root and stem rot (17°C) is much lower than that of fusarium wilt (21°C) (Vakalounakis, 1996). Temperatures around 20°C favour infection of young plants, especially if plants are under physiological stress during the first four weeks. Older plants are less susceptible. Disease does not develop at 32°C (Cerkauskas, 2001a).

2.1.2.3   Disease spread

Infection commonly occurs through wound sites arising during transplanting (Cerkauskas, 2001a).

Seed

In Canada seed infection is suspected, but not yet demonstrated (Cerkauskas, 2001a). *F. oxysporum* f. sp. *radicis-lycopersici* (*Forl)* has been reported on tomato seed, and its similarity to *F. oxysporum* f. sp. *radicis-cucumerinum* (*Forc)* increases the probability of *Forc* also being seed-borne.

Soil

The fungus can survive in plant debris and in soil for many years as chlamydospores, or for shorter periods as conidia on glasshouse structures between crops. There are several means by which the disease is known to spread in Canada (Cerkauskas, 2001a).

Water

Spores can spread in re-circulating systems via the irrigation lines. Infection is through root tips. Water dispersal is the most likely means of spread within the

glasshouse, especially in inert substrate crops. The fungus may colonise rockwool blocks (Cerkauskas, 2001a).

Plants

Infected seedlings can introduce the disease into the glasshouse when transplanted. The disease may then spread via root contact within rockwool slabs (Cerkauskas, 2001a).

Air

Aerial spread of spores is unlikely to be an important mode, as although numerous spores are produced on the diseased stem tissue, they are contained within a slimy material from which they are not readily dispersed. A spread of only one to two metres is likely. Spores may be spread from infected plants and growing media by shore flies (*Scatella* spp.) and fungus gnats (*Bradysia* spp.). These can spread spores back into the house if diseased material is taken out and not either covered over or burnt (Cerkauskas, 2001a).

Equipment

Plants can become infected through contaminated pruning instruments. Spores can be spread on workers' clothing (Cerkauskas, 2001a).

2.1.2.4   Control

It is important to apply control measures before seeding and setting plants in the glasshouse since infection probably occurs during the first four weeks of growth (Cerkauskas, 2001a; Punja & Parker, 2000).

Chemical control

Disinfection of the soil by fumigation with methyl bromide has proved effective (Pavlou *et al.*, 2002), but the use of this chemical is no longer permitted.

Infected seed treated with thiram (50 WP; 4.5 oz per 110 lb of seed) reduced plant mortality in trials where the fungicide treated seed was either immersed in a spore suspension for five to ten minutes before planting, or planted into artificially inoculated sphagnum moss compost (Rose *et al.,* 2003).

Hygiene

Seedlings should be checked for early death and be discarded together with surrounding healthy plants in the same tray. Prior to transplanting, the plants should be examined for symptoms such as wilting and stem infection and only apparently healthy material should be retained for transplanting. Apparently infected plants should be sent to a plant clinic for diagnosis (Cerkauskas, 2001a).

Monitoring for symptoms should continue in the glasshouse, with workers being made aware of the need to notify and mark any infected plants, and ensure that they do not spread infection from them. It was reported that pruning equipment should be dipped into a disinfectant (not specified) after each contact with the affected plant (Cerkauskas, 2001a). Trolleys and trays from the infected area should not be used in the rest of the house, disinfectant footbaths utilised at the entrance, disposable gloves and overshoes used in the area, and the areas should be worked in last.

When infected plants are removed, care should be taken not to allow contact of affected portions of plants with adjacent plants. One or two plants either side of these should also be removed carefully at the same time. The plants should be placed in a plastic bag directly as they are cut down. They should then not be left on an open waste heap or else spores may be carried back into the house (Cerkauskas, 2001a).

Infested slabs, bags, cubes or other media should be discarded or steam sterilised. Ensure all crop debris and strings are removed from the infected area. Drip lines and drip pegs of affected plants should either be replaced or cleaned with acid followed by

disinfectant. Irrigation lines should be flushed several times over a 24-hour period at the end of the crop season to kill spores. The house should be disinfected thoroughly (Cerkauskas, 2001a); efficacy of different types of disinfectant was not reported.

In Greece, glasshouses are disinfected with formaldehyde solution between crops (Pavlou, 2002).

Cultural control

Solarisation with impermeable plastic sheets over glasshouse soil in Greece was ineffective in soils with high root and stem root inoculum potential (although effective against cucumber fusarium wilt) (Vakalounakis, 1999).

Resistance

Grafting commercial Dutch-type cucumber hybrids onto various resistant *Cucurbita* rootstocks has been found to give effective control of root and stem rot. Of the six *Cucurbita* spp. found to be resistant to *Forl,* namely A27, *Cucurbita ficifolia,* Patron F(1), Peto 42.91 F(1), TS-1358 F(1), and TA-148 F(1), the last three showed superior horticultural performance in a glasshouse trial in Crete (Pavlou *et al.* (2002). In Greece, grafting on resistant rootstocks (*C. ficifolia, C. moschata* and *C. maxima* X *C. moschata*) was the only effective disease control method (Vakalounakis, 1999).

Seed dealers in Canada are reported to hold listings of susceptible and resistant cultivars (Cerkauskas, 2001a). Resistance genes to fusarium root and stem rot of cucumber have not been identified (Rose *et al.*, 2003).

Biological control

This should be targeted at the propagation stage of the cucumber crop (within 30 days after seeding) as this is when primary infection is likely to occur. Experiments with biological control agents added to either peat compost or the seedling cavity in rockwool blocks, followed by pathogen inoculation, have shown some products to be effective (Rose *et al.,* 2003). The addition of compost made from tomato plant and fruit waste together with sawdust significantly reduced seedling mortality, with suppression partially eliminated by compost sterilisation. *Gliocladium catenulatum*

(formulated as Prestop WP and Prestop Mix) also significantly reduced seedling mortality when applied 24 hours prior to inoculation. Crab / shrimp shell chitin (4% v/v) in peat compost reduced the population of *Forc,* but increased disease severity.

Neither *Trichoderma harzianum* (RootShield Drench), *Streptomyces griseoviridis* (Mycostop), nor *Trichoderma (Gliocladium) virens* (SoilGard) reduced disease incidence under experimental conditions, but Prestop WP, RootShield Drench and Mycostop were effective in two out of three glasshouse trials. Their efficacy was affected by seasonal differences in glasshouse growing conditions, which affected the incidence and severity of *Forc* root and stem rot. Seed treatment with *Pseudomonas chlororaphis* strain 63-28 reduced plant mortality (Rose *et al.,* 2003). It should be noted that many of the products mentioned above are not available in the UK; and none are approved for use to control fusarium diseases in cucumber in the UK.

2.1.2.5   Risks to other glasshouse crops

Pepper and tomato do not show disease symptoms. Pumpkin and squash show only mild symptoms. Watermelon is susceptible. Muskmelon is as highly susceptible as cucumber (Cerkauskas, 2001a).

2.1.3   Fusarium crown and foot rot

2.1.3.1   Introduction

Fusarium crown and foot rot, *Fusarium solani* f. sp. *cucurbitae,* infects all cucurbits. It is reported as infecting cucumbers in California, but only causing a problem on squash and pumpkin. The disease occurs most often in the Central Coast of California (Davis *et al.,* 2004). There are two races of the disease, race 1 attacking any part of the plant, while race 2 attacks only the fruit (Davis *et al.,* 2004).

2.1.3.2   Symptoms of the disease

Davis *et al*. (2004) recorded the symptoms on squash and pumpkin:
- Water-soaked lesions on the stem at the soil line.

- Infected plants wilt and die.

- On the fruit, a lesion usually begins on the area that is resting on the ground.

- Fruit lesions are firm and dry, with the decayed area exhibiting a concentric ring pattern.

2.1.3.3 Disease spread

The fungus survives in the soil and on seed (Davis *et al.,* 2004).

2.1.3.4 Cultural Control

Rotation out of cucurbits for two to three years and the use of clean seed is recommended (Davis *et al.,* 2004).

2.2 Sweet and chilli pepper

2.2.1 Fusarium wilts

2.2.1.1 Introduction

Two forms of fusarium are reported to cause wilt in pepper, *Fusarium oxysporum* f. sp. *vasinfectum* (syn. *F. annuum*) and a newly proposed *Fusarium oxysporum* f. sp. *capsici*. The latter name is not officially recognised. Neither disease has been confirmed in the UK. We have occasionally recovered *Fusarium* (species not determined) from roots of UK-grown hydroponic pepper plants showing basal decay, though we have never been able to determine if these are primary pathogens, secondary opportunists or even beneficial antagonists.

Although there are numerous reports of fusarium wilt of Capsicum species in the literature, there are few convincing cases of a fusarium vascular wilt disease (Pernezny *et al*, 2003). The most convincing case is in wilted Tabasco pepper (*C. frutescens*) plants in Louisiana (Rivelli, 1989). Rivelli proposed the name *F. oxysporum* f. sp. *capsici* sp. nov. This name has subsequently been used in India (Anon, 2000).

F. oxysporum f. sp. *vasinfectum* has been reported on sweet peppers in the USA in Ohio (Miller *et al*., 1996) and historically on Chile peppers in New Mexico (Sanogo, 2003) and Mexico (Keita *et al.*, 2002).

2.2.1.2 Symptoms of the diseases

The main symptoms of fusarium wilt in soil-grown pepper caused by *F. oxysporum* f. sp. *vasinfectum* (Miller *et al*., 1996) are in order of appearance:

- Decay of the roots and stem base
- Lower leaf wilting soon leading to wilting of the entire plant.
- Dark brown, sunken, and eventually girdling cankers occurring at the base of the plant

The main symptoms of wilt caused by the proposed *F. oxysporum* f. sp. *capsici* (Sanogo, 2003) are:

- Leaf chlorosis
- Vascular discolouration
- Plant wilting

Little research has been done on the disease cycle or epidemiology of this disease. Infection was confined to Tabasco pepper, Chile pepper and other *Capsicum* species and the fungus was not pathogenic to aubergine, cucumber and tomato. High temperature and high moisture are conducive to symptom development.

The main symptoms of *F. oxysporum* f. sp. *capsici* on chilli pepper, as described by the Indian Agricultural Advisory Company (Anon., 2000), are:

- Upward and inward rolling of the leaves
- Leaves turn yellow and die
- Discolouration of the vascular system, particularly in the lower stem and roots.

2.2.1.3   Spread of fusarium wilts

Soil

Both fusarium wilts are soil-borne, persisting several years (Miller *et al*., 1996; Anon., 2000). In field-grown chilli, localised, often poorly-drained areas become affected, wilt and die (Anon., 2000).

Seed

Seed transmission has not been reported. However, seed treatment has been found to give effective control (Anon., 2000).

No information was found on occurrence of air, water, insects, young plants or glasshouse equipment acting as sources of these pathogens.

2.2.1.4   Risks to other protected vegetable crops

Fusarium wilts also occur on tomato and eggplant, but they are infected by different types of *Fusarium oxysporum* than the type infecting peppers (Miller *et al*., 1996).

2.2.1.5   Control

Variety

In India, chilli varieties resistant to *F. oxysporum* f. sp. *capsici* are available (Anon., 2000).

Chemical control

Drenching with 1% Bordeaux mixture or Blue copper against F. oxysporum f. sp. capsici has been suggested in India. Seed treatment with 2 g carbendazim per kg of seed has also been found to be effective (Anon., 2000).

Biological control

Seed treatment with 4 g *Trichoderma viride* has been found effective against *F. oxysporum* f. sp. *capsici* on field grown chilli in India. 2 kg of *T. viride* is mixed with 50 kg farmyard manure, sprinkled with water and covered with thin polythene sheet. When mycelial growth is visible on the heap after 15 days, the mixture is applied to each acre of chilli pepper (Anon., 2000).

Fusarium wilt of pepper caused by *F. oxysporum* f. sp. *vasinfectum* has been shown to be controlled in glasshouse soil by the application of compost tea as a drench. Conidia and chlamydospores were destroyed when the activity of the tea was tested *in vitro* (Diver, 1998).

2.2.2   Fusarium solani fruit and stem rot

2.2.2.1   Introduction

There are two species of fusarium identified as causing fruit and stem rot of pepper in the UK, and a third species in Canada. Fruit and stem rot caused by *F. solani* was first recognised in the UK in 1992 (Fletcher, 1994), with more recent reports from New Zealand (Tyson, 2001) and Canada (Cerkauskas, 2001b). A fusarium internal fruit rot of sweet peppers caused by *Fusarium subglutinans* (subsequently described as *F.*

circinatum) has recently been reported as a new disease in British Columbia and Alberta, Canada (Utkhede & Mathur, 2003; Mathur & Utkhede, 2004), but it has not been reported on pepper crops in the UK. In 2005, a fusarium rot of sweet pepper on a UK nursery, which caused both internal and external fruit rot, together with fruit stalk and main stem lesions, was identified as *F. oxysporum* (O'Neill, unpublished). No wilt symptoms were observed. This disease therefore appears to be distinct from the pepper wilt *F. oxysporum* pathogens reported from the USA and Mexico.

2.2.2.2 Symptoms

Both stem and external fruit rotting symptoms on sweet pepper have been shown to be caused by *Fusarium solani* (Mart.) Sacc. (teleomorph: *Nectria haematococca, Berk & Br*) (Fletcher, 1994). The main symptoms recorded in the UK are as follows:

- Dark brown, slightly sunken lesions on the stem.
- Lesions may develop initially at the junction of the rockwool block and the stem, spreading to girdle the stem.
- Later in the season, nodes higher up the stem, and fruit, develop lesions.
- Dark-brown or black nodal lesions develop following the colonisation of senescent fruit bearing laterals.
- Fruit decay generally starts at the calyx end. The rotting spreads to the shoulder of the fruit.
- A dry rot of the fruit occurs, eventually affecting the whole fruit, with the tissue often collapsing in a concentric line pattern.
- Pink sporodochia appear on the older stem lesions and also on affected fruits.
- A large proportion of the plants can become affected, randomly throughout the house, with high plant losses in the final month of cropping.
- Disease severity between cultivars can differ, with shorter fruiting laterals probably increasing cultivar susceptibility.

A first report of this disease from New Zealand (Tyson, 2001) also mentioned:

- The leaves of infected plants exhibited features resembling magnesium deficiency
- The fruit surface became covered in whitish aerial mycelium, cream / yellow spore masses, and masses of distinctive red perithecia.

In Florida, the first reported outbreak in 1999 caused severe yield losses (Lamb *et al.*, 2001) with symptoms of:

- Wilting and death of the upper portions of the plant, but no fruit rot symptoms.

In Canada, the two stages of the growth cycle of the fungus, the perfect stage *(N. haematococca)* and the imperfect (*F. solani*) are known to occur concurrently on pepper stem tissues (Cerkauskas, 2001b). Infection without symptom development may occur on the shoots, with symptom development evident as much as two to three months later or at the end of the season. Symptom development is triggered by plant stress arising from a heavy fruit load, adverse environmental conditions, or senescence (Cerkauskas, 2001b).

2.2.2.3   Spread of fruit and stem rot

Conidia can be carried in water or on tools and hands. Plant debris and glasshouse fittings can carry the disease between crops. In Canada it has been found that forcible ascospore ejection from perithecia is the primary means of natural disease dissemination. This occurs at night when higher humidity (greater than 95%) is present to favour disease development (Cerkauskas, 2001b).

2.2.2.4   Control

Symptoms are associated with leaf and fruit picking wounds and leaf drop (Tyson, 2001). Knives require disinfection and hands should be washed after handling diseased material. In the UK there has been time-consuming cutting out of nodal lesions, associated with the picking scars and senesced fruit, to stop the spread of the rot, but the area is then exposed to further spores. In Canada, clean-cutting of leaves, axillary shoots and fruit with a sharp knife was shown to be important, rather than breaking and tearing (Jarvis *et al.*, 1994).

Infection develops at sites of tissue damage caused by evaporated 'salt' accumulation at the stem base (Jarvis *et al.*, 1994). This damage can be reduced by not allowing rockwool blocks to dry out. The fertiliser dripper should be positioned away from the stem base (Cerkauskas, 2001b). Correct monitoring of the rockwool blocks is also

important so that there is no excessive wetness. Over-watering can stress plants through oxygen depletion, resulting in a high incidence of shoot lesions (Cerkauskas, 2001b).

Ascospore germination can be reduced by controlling the glasshouse climate to avoid extended periods above 95% humidity at the leaf surface. This occurs when the relative humidity in the glasshouse is 90% (Cerkauskas, 2001b).

Controlled temperature studies have shown that the rate of disease development is greater at higher temperatures, and so keeping the house cooler could be considered (Lamb *et al,* 2001), but temperatures were not specified.

Effective disinfection between crops to eliminate inoculum is necessary to stop initial infection of the crop. Fletcher (1994) found macro-conidia were killed by 4 to 8% solutions of domestic bleach (4% available chlorine), 2.5 to 10% Purogene (chlorine dioxide) and 2 to 4 % Tego (quaternary ammonium compound), but not by phenolics. However, *Fusarium solani* also produces chlamydospores in dead or dying plant tissue (Cerkauskas, 2001b), and it is not known how effectively they may be controlled. It is essential to remove all debris from the house and destroy it, with material from infected rows being removed from the house within the life of the crop.

In Canada, biological control with either Mycostop (*Streptomyces griseoviridis),* or Prestop Mix (*Gliocladium catenulatum)* added to rockwool blocks has been found to be effective against *Fusarium oxysporum* on cucumbers (Rose *et al.*, 2003), and so may be effective against *F. solani.* In Canada, sexual fruiting bodies of *N. haematococca* have been seen growing on the rockwool slabs (Cerkauskas, 2001b).

Although Fletcher (1994) found that cv. Tasty was more severely affected than cv. Mazurka, no difference in susceptibility was found in Florida between the cultivars Cubico, Kelvin, Triple 4 and Grizzly (Lamb *et a*l, 2001).

There are no fungicides with a label recommendation for the control of fusarium stem rot of pepper in the glasshouse in Florida (Lamb *et al.*, 2001). Jarvis *et al.* (1994)

found no suitable fungicides after *in vitro* screening, and there are no products registered in Canada for stem rot control on peppers.

In the UK, Amistar (azoxystrobin) has a SOLA (1295/02) for use on peppers as a spray against powdery mildew. This fungicide warrants investigation to determine if it provides some control of fusarium stem rot.

2.2.3   Fusarium oxysporum fruit and stem rot

A fruit and stem rot of pepper has occurred on at least one UK nursery for several years in succession, with the problem increasing; in 2005 the cause was identified as *F. oxysporum* (O'Neill unpublished). A fruit and stem rot caused by *F. oxysporum* is reported to occur also in the Netherlands (Aad Vijverberg, pers. comm.).

2.2.3.1   <u>Symptoms of fruit and stem rot</u>

- Aborted fruit, often with white sporulation.
- Externally visible fruit rot usually originating at the blossom end.
- A brown lesion visible externally at the junction of the fruit stalk with the main stem.
- A brown lesion at stem nodes.
- Pale brown stem lesion with a dark brown border, extending around 5 cm above and below an infected node.
- Dark brown stem lesion extending 1-2 m along the stem
- Internal brown rot in the pith and vascular tissue of the fruit stalk; visible at the cut end.
- Brown longitudinal streaks may be visible externally on the fruit stalk if the internal rot penetrates tissue close to the surface.
- White fusarium sporulation on the internal seed-bearing region of the fruit.
- No wilting reported to date, nor stunting of plant growth.
- No stem base rot observed.
- No root death.

In the UK, in 2005, the rot was found in one glasshouse crop in early March and had increased to cause substantial losses, to approximately 20% of the day's pick, by mid-April (O'Neill, unpublished).

2.2.3.2 Spread of fruit and stem rot

Seed

Information from the Netherlands suggests that *F. oxysporum* may occur on the outside of sweet pepper seed, but not internally (Aad Vijverberg, pers comm.). Seed transmission of *F. oxysporum* has been reported in several crop species where fusarium wilt or crown and root rot diseases occur (Maude, 1996). However, if seed is the source of the pathogen, the disease symptoms in pepper are unusual in that no wilting, or root and stem base rot has been observed.

2.2.3.3 Control of fruit and stem rot

Interim measures in the UK include cutting out the rot from the main stem to prevent further spread into the stem.

2.2.4 Fusarium circinatum fruit rot

This disease has not been reported from the UK. Fusarium fruit rot of sweet peppers caused by *Fusarium subglutinans* (Wollenweber & Reinking), subsequently described as *F. circinatum,* was recently recognised in Canada (Utkede & Mathur, 2003). In addition to sweet pepper, *F. circinatum* infects other major food crops such as maize and mango and severely affects pineapple in Brazil. Many species of pine are susceptible to pitch canker caused by *F. circinatum* (editor's note to Agnet page for Utkhede & Mathur, 2003).

2.2.4.1 Symptoms of external and internal fruit rot

Infection in commercial glasshouses of sweet peppers in British Columbia and Alberta, Canada has been described by Utkhede & Mathur (2003), in orange peppers cv. Sympathy MZ:
- Symptoms appear on mature fruit at harvest.
- Discoloured soft patches or necrotic spots develop predominantly at the calyx end.

- Occasionally soft patches are seen anywhere on the mature fruit at harvest time.

- Seeds and the surrounding area inside the fruit are covered with fungal growth and pink spores.

- No evidence of root infection.

At one commercial holding in British Columbia in 2001, approximately 40% of fruits were severely affected, with 10% affected the following year (Utkhede & Mathur, 2003).

2.2.4.2 Spread of external and internal fruit rot

Water

Artificial inoculation of just-opened and fully opened flowers with 20 µl drops of conidial suspensions of *F. circinatum* (without wounding) produced a high proportion of diseased fruit, more so than following inoculation of developing fruit (Utkhede & Mathur, 2003; 2004). Approximately 80% of inoculated fruits and flowers developed symptoms identical to those seen from natural infection. This suggests spore splash or contact (e.g. by insects) could be important in natural disease spread.

Seed

Utkhede & Mathur (2004) found that none of the seeds from infected pepper fruits germinated. This would mean that infected plants could not be produced from them. It is unlikely that visibly rotting fruit would be harvested for seed, but a slower or lower level of infection could still allow seed to be harvested. There is no record of infected pepper seedlings.

2.2.4.3 Risks to tomato and other glasshouse crops

No disease was produced on lettuce (Utkhede & Mathur, 2003), tomato, cucumber or eggplant using artificial inoculation with spore suspensions (Utkhede & Mathur, 2004).

2.2.4.4 Control of external and internal fruit rot

Variety

Fruits of *C. annuum* cvs Bison and Mazurka were less susceptible than those of cvs Sympathy and 444 (Utkhede & Mathur, 2004).

Rotation

Artificial inoculation tests suggest that lettuce, tomato, cucumber and eggplant could be planted after an infected pepper crop without producing a rot and thus give an interval in which spores could die.

2.3 Tomato

Since the move into hydroponic production systems many of the previously persistent soil-borne disease problems have disappeared. In the last few years there has been an increase in reports of wilting in hydroponic tomatoes and this has corresponded with an increase in diversification of the crop to cherry, plum, vine, beefsteak and other types to satisfy the increasing market. More recently, there has been renewed interest in organic production and a number of growers have consequently moved back into soil-grown systems. The new and diverse cultivars being grown do not necessarily have the full complement of resistance genes against the key pathogens such as *Fusarium oxysporum* f. sp. *lycopersici* and/or *F. oxysporum* f. sp *radicis-lycopersici*.

ADAS and STC have received an increasing number of tomato samples in the last few years with foot-rot, wilt, vascular staining and plant collapse. In some cases, especially where the vascular discoloration has been marked, we have recovered a *Fusarium* sp. associated with wilted plants; in others, where the vascular staining has been lighter in colour, *Verticillium* has been recovered. We have also occasionally consistently recovered a *Fusarium* sp. from the roots of inert substrate (hydroponic) and/or NFT plants showing overall poor vigour.

No strain typing has been conducted on the isolates recovered and no pathogenicity testing has been undertaken to determine the virulence of particular isolates recovered.

2.3.1 Fusarium wilt of tomato

2.3.1.1 <u>Introduction</u>

A fusarium wilt of tomato was first recorded in England in 1895 (Jones *et al*., 1991). Previously described as *F. lycopersici* and *F. bulbigenum* f. sp. *lycopersici* (Brooks, 1953), the pathogen is now accepted as *F. oxysporum* f. sp. *lycopersici*. The fungus infects only members of the genus *Lycopersicon*. By 1965 the disease was common in glasshouse crops in north-western Europe and in parts of the USA. Two forms of fusarium, *Fusarium redolens* and *Fusarium oxysporum* f. sp. *lycopersici,* were

recognised in 1971 as causing tomato wilt in England (Schofield, 1971). *F. redolens* is considered by many authors simply to be a form of *F. oxysporum* that has a characteristic odour in culture (of lilac).

2.3.1.2   Race nomenclature

The nomenclature of fungal races is notoriously complex. With *F. oxysporum* f. sp. *lycopersici* this is particularly so because of the existence of two different numbering systems. An outline is given below.

Populations of *Fusarium oxysporum* f. sp. *lycopersici* are differentiated into races based on differences in pathogenicity to different cultivars. The resistance of the plant is controlled by dominant genes, designated I-1 and I-2 (Gabe, 1975). A cultivar may have neither resistance gene, I-1 only, I-2 only or both I-1 and I-2. The numbering of the races of *F. oxysporum* f. sp. *lycopersici* is determined be relating their virulence to the host's resistance genes. Race 0 is virulent only on those cultivars that carry no resistance gene. Conversely, all cultivars that carry I-1 or I-2 are protected against attack by race 0. The I-1 resistance gene protects against race 0, but it is overcome by race 1. The I-2 resistance gene protects against races 0 and 1, but is overcome by race 2. With two resistance genes a maximum of four (2^2) potential fusarium races can be identified (Table 2.1).

Table 2.1. The relationship between potential races of *Fusarium oxysporum* f. sp. *lycopersici* and genes for resistance in tomato using the 0,1… system of nomenclature.

Resistance genes in tomato	Races of Fol			
	0	1	2	1.2
Nil	S	S	S	S
I-1	R	S	R	S
I-2	R	R	S	S
I-1, I-2	R	R	R	S

R = resistant   S = susceptible

More recently, four resistance genes (I, I-1, I-2 and I-3) to *F. oxysporum* f. sp. *lycopersici* have been described (Bonnema, 1997), implying a potential of 16 races (2^4).

American authors have generally numbered races of *F. oxysporum* f. sp. *lycopersici* starting from 1, rather than 0, i.e. race 1 is virulent only on those varieties lacking resistance genes and race 3 is virulent on varieties lacking I-2 (Table 2.2).

Table 2.2. The relationship between potential races of *Fusarium oxysporum* f. sp. *lycopersici* and genes for resistance in tomato using the 1,2…. system of race nomenclature.

Resistance genes in tomato	Races of Fol			
	1	2	3	2.3
Nil	S	S	S	S
I-1	R	S	R	S
I-2	R	R	S	S
I-1, I-2	R	R	R	S

R = resistant   S = susceptible

The disadvantages of this system are that:

a) it results in an irrational numbering (e.g. resistance gene I-2 is overcome by race 3, whereas is the 0, 1, 2.. system, resistance gene I-2 is overcome by race 2);

b) it is not consistent with the internationally accepted numbering of races for other tomato pathogens (e.g. *Fulvia fulva*, ToMV). The traditional 0, 1, 2… system is therefore used in this review. Where required for clarification, the 0,1,2.. system is referred to as UK nomenclature and the 1,2,3… system as USA nomenclature.

2.3.1.3   Occurrence of races

There are three known races of *Fusarium oxysporum* f. sp. *lycopersici* (*Fol*), races 0, 1 and 2 (races 1, 2 and 3 in American nomenclature). Race 0 was prevalent at the beginning of the twentieth century and is now the most widely distributed throughout the world. The I-1 gene completely controlled fusarium wilt for 20 years before race 1 became a problem (Kroon & Elgersma, 1971). Race 1 became widespread in tomato growing areas in the USA after 1961. Race 1 is now known from the UK, the Netherlands, Israel, Morocco, Australia and Brazil (Jones *et al.*, 1991). Following initial reports from Brazil in 1966, a race 2 (American race 3) (overcoming the resistance gene *I-2*) is now present in various states of America, Mexico and Australia (Gale *et al.*, 2003). Routine monitoring did not reveal it in Israel (Katan *et al.*, 1997). In the USA, resurgent interest in the planting of heritage varieties has increased the

incidence of fusarium wilts (Miller *et al.,* 1996), as has the introduction of cherry tomato cultivars in Israel that lack genes for resistance (Katan *et al.,* 1997).

In 2004, one crop in Hertfordshire suffered a severe vascular wilt disease. Samples were sent to the Netherlands and the problem was reported to be due to *Fol* race 2 (American race 3) (Nigel Moore, pers. comm.). There is no published report of this race occurring in the UK. In 2004, a rockwool crop of cv. Classy developed symptoms suggestive of a vascular wilt disease, with severe symptoms in about 3% of plants and *F. oxysporum* was consistently recovered from vascular tissue (O'Neill, unpublished); race-typing was not undertaken.

2.3.1.4   Saprophytic or weakly pathogenic strains of *F. oxysporum*

In the USA, light-brown discolouration of tomato roots has been attributed to saprophytic-type isolates *F. oxysporum* that do not cause wilt symptoms in tomato or other vegetable crops (Shishkoff & Campbell, 1990).

In the 1990's *F. oxysporum* was occasionally recovered from the roots and stem vascular tissue in UK laboratories. When seedlings in an agar plate test were inoculated with an isolate of *F. oxysporum* obtained from the roots of cv. Blizzard, only the root tips became soft and discoloured and there was no development of either fusarium wilt or crown and root rot symptoms (O'Neill, unpublished). This root rotting fusarium may thus be a weakly-pathogenic isolate distinct from those causing wilt or crown and root rot.

2.3.1.5   Symptoms of the disease

Symptoms of fusarium wilt caused by *Fol* are not easily distinguished from those of verticillium wilt, especially in the early stages of infection, although there is a tendency for more yellowing of the plants (leaves and stems) with fusarium wilt, often with one side of the plant more severely affected (ADAS, 1980b; Fletcher, 1984). In the case of fusarium infection the vascular discolouration is a very deep brown, often referred to as chocolate brown, whereas with Verticillium it tends to be a much lighter-brown in colour.

Wilt symptoms on English tomato crops have been described by Schofield (1971) and Fletcher (1984), and match those recorded in Israel by Katan *et al.* (1997) and also in the USA by Jones *et al.* (1991) and Miller *et al.* (1996), although only Schofield (1971) mentions sporulation on the stem. Plants may be attacked at any growth stage and symptoms may include:

Growth

- Infected seedlings are stunted.

- On young plants there is yellowing of cotyledons, and lower leaf vein clearing, yellowing and collapse.

- The entire plant may be killed, often before it reaches maturity.

Wilting

- Petioles droop.

- On older plants, leaves yellow, wilt and die from the base of the plant upwards.

- Symptoms on older plants generally become apparent during the interval from blossoming to fruit maturation.

- Wilting may affect only one side of the plant, or one side shoot.

Vascular staining

- Pronounced brown discolouration of the vascular tissue, which may be visible externally.

- On splitting open the stem, dark, chocolate-brown streaks run lengthways where the vascular tissue is stained.

- The discolouration often extends upward for some distance. To 1.5 m or more in cherry tomatoes.

- Browning is especially evident where the petiole joins the stem, and can be seen in petiole scars.

- A chrome yellow stripe may be seen on outside of stems with internal vascular symptoms.

Stem lesions

- Areas of the stem surface on the older parts of affected stems may become sunken to form lesions.

- Fusarium spores may occur on the sunken stem lesions. In Israel on cherry tomatoes, sporulation has been reported without the presence of external lesions.
- The pith remains healthy.

Roots

- Roots are frequently rotten, although the root system may look healthy.
- Adventitious roots may develop up the stem.

Fruit

- Fruit infection occasionally occurs, with vascular tissue discolouration within the fruit.

The production of vascular discolouration well up the plant is a symptom that particularly distinguishes fusarium wilt disease from that of crown and root rot caused by *F. oxysporum* f. sp. *radicis-lycopersici* (*Forl).*

Sporulation on the lower stem is a common feature of *Forl*, but seems to feature less in descriptions of *Fol* wilt. It is possible that practices such as drip irrigation, which increases humidity at the stem base, encourage sporulation. In Israel a pink or yellowish layer of principally macro-conidia, or less frequently a whiter layer dominated by micro-conidia, has been observed on the lower 10 to 15 cm (occasionally 25 cm) of the stems of fusarium wilt infected cherry tomatoes. These plants were slowly desiccating, in an advanced stage of disease development. Spores were trapped from the air (Katan *et al.*, 1997).

A vegetative compatibility (heterokaryon) test, using *nit* mutants, has been used to distinguish between *F. oxysporum* f. sp. *radicis-lycopersici* and *F. oxysporum* f. sp. *lycopersici*. In Israel, races 0 and 1 of fusarium wilt belong to the vegetative compatibility group (VCG) 0030, whereas fusarium crown and root rot belong to five other VCGs (Katan *et al.,* 1997).

The disease is encouraged by high temperature. The optimum soil temperature is 28ºC, with inhibition of the disease below 21ºC and above 33ºC. The first outbreaks

tend to occur in warmer areas of the glasshouse and in warm propagation areas (Schofield, 1971).

Disease development is favoured by low soil moisture, short day length, low light intensity, nutrients low in nitrogen and phosphate and high in potassium, and a low pH (Schofield, 1971; Jones *et al.*, 1991).

2.3.1.6 Pathogen infection mechanisms and host defence

From a study of inoculated hydroponic culture medium it was seen that the hyphae rapidly colonise the tomato root surface and penetrate the epidermis within 24 hours. The fungus then grows towards the stele, overcoming plant defence reactions in the hypodermis, so that within 7 days the stele is densely colonised (Olivain & Alabouvette, 1999).

2.3.1.7 Disease spread

Soil

Fusarium persists in the soil for several years. Infection takes place through the roots (Schofield, 1971). The fungus is present in root debris but can persist in the soil as chlamydospores (Fletcher, 1984). Chlamydospores form abundantly (Jones *et al.,* 1991).

Seed

Seed-borne infection is known to occur (Schofield, 1971) and is a possible source of the disease (Fletcher, 1984).

Plants

Transplants are a source of long-distance spread of the disease (Jones *et al.*, 1991).

Drip pegs and other glasshouse equipment

Soil/substrate contaminated material may carry spores into the next crop, which is particularly important if longer-lived chlamydospores have developed.

Water

Conidia may be washed from the stem bases into re-circulating irrigation water, and so reach the roots of healthy plants. The potential of *Fol* to be disseminated in a closed hydroponic production (rockwool) following artificial inoculation, and for its control with disinfection equipment, was investigated at STC (McPherson *et al*., 1995). An isolate of the pathogen provided by Dr JT Fletcher, ADAS Wye, was demonstrated to be pathogenic to tomato seedlings cv. Ailsa Craig prior to point-source introduction in a crop of the same cultivar.

Dissemination was monitored by tracking the development of wilt symptoms in the crop and by specific isolation from Ailsa Craig 'bait' plants placed in the centre of rockwool slabs where the roots were 'washed' in the recycled irrigation solution. Classic symptoms of *Fusarium* wilt developed, including a foxy-red staining of the vascular tissues, and the pathogen was observed to spread (28% plants visibly infected) in the re-used irrigation solution in the absence of any disinfection system. The pathogen also spread to infect the uninoculated control plants and this was considered to be a result of aerial or mechanical dissemination following active sporulation from stem lesions high up the plants. Where the hydroponic solution was disinfected using heat (Pasteurisation), UV (Priva 'Vialux'), Ozone (Ozotech ozoniser) or microfiltration (Memcor Ltd), dissemination of the pathogen was largely prevented. Whilst there is a limited amount of evidence to suggest that *Fusarium* spp. are removed or partially removed from recirculated hydroponic solution with slow sand filters (Runia *et al*, 1997; Wohanka, 1995) further work is required in long-season crops like tomato or pepper to demonstrate freedom from disease using this technique of solution disinfection.

It should also be noted that there is potential for spread via root contact in crops where the solution is collected in gutters (but not recirculated) or even in conventional hydroponic crops where the roots are allowed to develop from the drainage slits in the substrate wrapper.

Air

It is known that fusarium wilt is spread short distances in the air (Schofield, 1971). Katan *et al.* (1997) trapped air-borne spores at a height of 15 cm, and isolates of *Fol*

from plant strings and glasshouse walls were presumed to have arisen from air contamination.

Cultural control

The disease development is slowed if soil pH is raised to 6.5 to 7.0. The disease progresses faster in plants growing with low nitrogen, and so using nitrate nitrogen rather than ammoniacal nitrogen slows wilt development (Jones *et al.,* 1991).

2.3.1.8 Risk to other protected vegetable crops

Other solanaceous crops, such as pepper, are infected by *Fusarium oxysporum* wilts, but each crop is infected by a different type or host-specific strain of the pathogen (Miller *et al.*, 1996).

2.3.1.9 Control

Varietal resistance

Most modern round tomato cultivars grown in the UK, Europe, Israel and the USA have genetic resistance to races 0 and 1 of fusarium wilt, caused by *Fol* (ADAS, 1980b; Katan *et al.*, 1997; Miller *et al.*, 1996). Before the development of resistant cultivars, tomato production in Florida was nearly destroyed by fusarium wilt (Jones *et al.,* 1991). The rootstocks KVF and KVFN are also resistant to fusarium wilt (Schofield, 1971). Monogenic resistance to race 2 (American race 3) has been identified (Jones *et al*., 1991). The rootstocks Oxyfort from De Ruiter and Zodiac from Yates were recently reported by the seed companies to be resistant to race 2 (American race 3) of fusarium wilt (A. Lee pers. comm.). Trifort is also reported to be resistant (J Overvoorde, pers. comm.). It is unclear whether the resistance of these rootstocks is unrefined or monogenic. Resistance to a third race in *Lycopersici chilense* is polygenic (Bonnema, 1997).

Chemical control

Drench treatments of the growing crop with benzimidazole fungicides gave only partial control (Fletcher, 1984). There are no currently approved pesticides (including Specific Off-Label Approvals) with a specific recommendation for use against fusarium on glasshouse tomatoes in the UK (Whitehead, 2005). However, as the

target pathogen is not a statutory condition of approval, some fungicides approved for use on glasshouse tomato for control of other diseases (e.g. carbendazim) may provide some suppression of fusarium infection.

Treatment of infested soil is most satisfactorily carried out with steam, provided that it is done to a sufficient depth, below the level of root penetration and cultivations (Fletcher, 1984).

Biological control

In the USA, treatment of tomato seedlings with *Gliocladium virens, Trichoderma hamatum, Pseudomonas fluorescens* and *Burkholderia cepacia* before planting into fusarium wilt infested soil gave 30 to 65% reduction of fusarium wilt. Commercially available *G. virens* (SoilGard) and *T. harzianum* (RootShield) granules incorporated into potting compost (0.2% w/v) reduced the disease by 62 to 68% (Larkin & Fravel, 1998). *Penicillium oxalicum* conidia applied as a seedbed drench before sowing tomato seedlings gave a 50% reduction in fusarium wilt in a glasshouse trial (Larena *et al.*, 2003). It should be noted that the products mentioned above are not approved for use to control fusarium on tomato in the UK.

Systemic Acquired Resistance (SAR) is another mode of potential biological control. Under controlled environment conditions, *Phytophthora cryptogea* zoospores were sprayed onto above ground tomato tissue and followed by inoculation with *Fol*. Although both fungi were found in the stems, there were no wilt symptoms over the next 50 days (Attitalla *et al.,* 2001).

Induced Systemic Resistance (ISR) may also be involved in biological control. In tests with isolates of non-pathogenic *Fusarium oxysporum* and *Fusarium solani* collected from fusarium-wilt suppressive soil, these gave 50 to 80% reduction of disease incidence (Larkin & Fravel, 1998). Further tests showed that a specific isolate, of *F. oxysporum*, was most effective at the higher temperature of 27°C optimum for disease development (Larkin & Fravel, 2002). *F. oxysporum* f. sp. *dianthi* was also shown to induce resistance to Fol in tomato and significantly reduced wilt symptoms (Kroon *et al*., 1991). Inoculation experiments with another non-pathogenic *F. oxysporum* strain

protected tomatoes in either hydroponics or compost against fusarium wilt. Wilt resistance was thought to be induced by the non-pathogenic strain, as there was an increase in chitinase, β-1,3-glucanase and β-1,4-glucosidase activity by the plants (Fuchs *et al.,* 1997). The non-pathogenic strain may also compete with the pathogen for the colonisation of the root surface and cause defence reactions by the host in the cortex such as wall appositions, intracellular plugging and deposits which mean that these are in place before pathogen attack (Olivain & Alabouvette, 1997).

Vermicompost (i.e. compost generated by worms) added to various container media inhibits the infection of tomato plants by *Fol.* The vermicompost had high numbers of bacteria and fungi, and when the compost was sterilised it lost its effectiveness (Szczech, 1999).

2.3.2   Fusarium crown and root rot

2.3.2.1   Introduction

Fusarium crown and root rot of tomato is caused by *Fusarium oxysporum* f. sp. *radicis-lycopersici* (*Forl*). It was first reported causing serious rotting in glasshouse tomatoes in Japan, Canada and the USA in 1974, although it was initially recorded as another race of fusarium wilt (Yamamoto *et al.,*1974; Jarvis & Thorpe, 1976). It now affects tomato production in Japan, Canada, Mexico and the USA, both in the glasshouse and in the field (Jones *et al.,* 1991) and is also known to occur in many countries in Europe (including the UK) and the Mediterranean region (Katan & Katan, 1999; Can *et al.,* 2004). It causes significant yield losses in glasshouse tomatoes of between 20 – 60% (Jarvis *et al.*, 1983) and in field-grown tomatoes in Florida (Roberts *et al.*, 2001). It is one of the most prevalent soil-borne diseases of tomato. In the UK, it was first noted in 1988 and caused serious problems for several years. Now it is seen occasionally on heritage varieties lacking resistance to *Forl*. It is a serious problem in closed systems, with rockwool as a growing medium and where re-circulation of nutrient solution is practiced (Hartman & Fletcher, 1991).

The pathogen has different vegetative compatibility groups (VCGs) and molecular techniques can map their geographical distribution. It appears possible, from these

results, that an isolate from Palm Beach in Florida spread to Europe (Roberts *at al*, 2001). VCG 0094 (divided into three sub-groups based on hyphal interactions) was found to be dominant in 1988, 1990 and 1991 isolates from the UK, with this group also present in Belgium, France and in the Netherlands, but not in Israel (Katan & Katan, 1999). The population structure of *Forl* in Italy is similar to that reported in Israel (Di Primo *et al.,* 2001).

2.3.2.2   Symptoms of the disease

Seedling infection has been reported by Roberts *et al.* (2001) in Florida. Seedlings showed stunting, yellowing and premature loss of cotyledons and lower leaves.

Seed inoculation with macro-conidia by Jarvis & Thorpe (1976) produced chocolate-brown lesions in the root-shoot transition zone.

Jarvis & Thorpe (1976), Jones *et al.* (1991) and Roberts *et al.* (2001) also describe later infections. The first clear symptoms are usually seen when the first fruit is at or near maturity. Clusters of symptomatic plants often occur within a row. Symptoms may include:

Growth
- Infected plants may be stunted.
- Yellowing along the margins of the oldest (lower) leaves.
- Yellowing is soon followed by necrosis, and collapse of the leaf petiole.
- Symptoms develop slowly upwards on successively younger leaves.

Wilting
- Wilting first occurs during the warmest part of the day, and plants appear to recover overnight.
- Some plants wilt slowly so that they are still alive at the end of harvest, although they produce reduced numbers of inferior fruit.
- Other plants wilt quickly and wither.

Root rot

- Root infection first occurs on the adventitious roots, with fungal penetration primarily through the cortex, but the xylem is frequently discoloured several centimetres in advance of the mycelium.

- Round, brown lesions are visible at the origins of rotted lateral roots.

- The fungus progresses slowly into the tap root and lateral roots, causing a dry, brown rot of the root cortex and xylem across the entire root system.

- The tap root becomes rotted off.

Stem lesion

- A pronounced chocolate brown lesion girdles the hypocotyl (root / shoot junction).

- A broad necrotic lesion on the stem surface, usually develops from the soil line to 10 to 30 cm above it.

- Discolouration of the stem vascular tissue, seen as brown streaks, can occur but extend no further than 20 to 30 cm above the soil line. Internal symptoms often extend no more than 10 cm above the girdling lesion.

- An abnormal proliferation of adventitious roots may occur above the infected region.

- Abundant pink (also described as yellow to orange) sporulation is visible on above ground necrotic lesions and dead plants. Sporulation in outdoor crops may be greater after rain or fog.

Fusarium crown and root rot vascular discolouration, which may be seen when the stem is sectioned lengthways, extends for a shorter distance up the stem than that of fusarium wilt (Roberts *et al.,* 2001). Fusarium is readily isolated from the vascular tissue following crown and root rot. Such isolation procedures may be necessary to distinguish it from the chocolate brown lesions of either *Phytophthora* basal rot, of *Rhizoctonia* root rot or vascular wilts caused by bacterial pathogens e.g. brown rot (*Ralstonia solanacearum*), which similarly occur at or above soil level on wilting mature plants.

The disease is favoured by cool temperature, 10°C to 20°C, which is in contrast to the hot temperatures favoured by fusarium wilt (Roberts *et al.,* 2001).

2.3.2.3   Pathogen infection mechanisms and host defence

The fungus was believed principally to invade susceptible plants through wounds and natural openings created by newly emerging roots (Roberts e*t al*., 2001). However, the infection process of tomato roots *in vivo* has now been visualised using scanning microscopy and green fluorescent protein (GFP) to mark the fungus (Lagopodi *et al.,* 2002). The first contact between the fungus and the host takes place at the root hair zone by mingling and by the attachment of hyphae to the root hairs. The grooves along the junctions of the root epidermal cells are preferentially colonised. In the roots observed, there was an absence of specific infection sites, such as sites of emergence of secondary roots, root tips, or wounded tissue. Specific infection structures, such as appressoria, were also absent. The absence of specific entry sites does not exclude the possibility of hyphae taking paths of less physical resistance into the roots.

2.3.2.4   Disease spread

The epidemiology of the disease has been reviewed by Jarvis (1988) and Ozbay & Newman (2004).

Air

Micro-conidia are commonly found in the air of infected glasshouses (Jones *et al.*, 1991). They are formed in great abundance in necrotic tissue and will spread by air currents to readily re-infest sterilised soil (Rowe *et al.,* 1977; Roberts *et al*, 2001). If micro-conidia are capable of infecting other plants and causing disease in the same growing season then this would enhance the dissemination capacity of infected plants.

Macro-conidia have also been found to naturally infect tomatoes growing in the field (grown in containers isolated from the soil) by direct infection of the foliage, with 82% of the plants developing typical crown and root rot symptoms. Artificial foliage inoculation of glasshouse tomatoes infected 75 to 100% of plants. Downward movement of the pathogen from the foliage to the crown and roots was observed. Wounding enhanced pathogen invasion and establishment in the foliage (Rekah & Shtienberg, 2000; Rekah *et al.,* 2001).

Soil

Chlamydospores occur in large concentration in the soil and will germinate to infect the tomato roots (Jones *et al.,* 1991). They have thicker walls than micro-conidia, and so can survive for more than one cropping season (Roberts e*t al*, 2001). There is negligible movement of the pathogen within soil (Rekah *et al.,* 1999). Movement of the fungus in the soil in the absence of roots was shown by monitoring experiments to be less than 2.5 cm (Roberts *et al.*, 2001). In Israel, it was found that primary infection in tomatoes was from over-summering inoculum in the soil, and that the onset of symptoms was related mainly to the rate of pathogen proliferation in the soil around the roots rather than the initial inoculum density. The range of disease onset timings in the plants was attributed to differences in the level of soil suppressiveness (affecting pathogen proliferation) across the field. This monocyclic phase of the disease (i.e. occurring once during crop production) overlaps with the separate polycyclic phase of secondary infection (i.e. occurring several times during crop production) as a result of root-to-root contact. Disease onset in plants infected via root contact with their diseased neighbours was unrelated to either initial soil inoculum density or pathogen root zone proliferation (Rekah *et al.,* 2001).

Spread in the field in Israel to tomatoes on the seeds of a weed, *Tamarisk nilotica*, after colonisation of its roots by the pathogen has also been found (Rekah *et al.,* 2001).

Water

The fungus is reported to require root contact to spread in the crop (Roberts *et al.*, 2001). Uninfected plants growing in the same rockwool production slabs as infected plants were found not to develop substantial disease symptoms. Rockwool systems may be less vulnerable to rapid spread of the disease than would seem likely (Mihuta-Grimm *et al.*, 1990). Airborne spore dispersal and movement on plants and equipment may be more important.

Plants

The pathogen may be carried on symptomless or mildly infected transplants (Hartman & Fletcher, 1991) and in soilless media (Brammall & McKeown, 1989).

The pathogen spreads laterally from plant to plant, primarily by root contact, to produce plant disease clusters in the crop (Roberts *et al.*, 2001). In field soil in Israel, the disease spread up to four plants (2 m) either side of an inoculated focus plant, with root intermingling between plants facilitating the spread.

Insects

Plants can also become infected when conidia from sporulating stems are carried by adult fungus gnats (*Bradysia* spp.) to the roots and hypocotyls of healthy plants. The fungus also enters the body of larvae feeding on the roots, but they do not appear to enhance plant infection by the pathogen (Gillespie & Menzies, 1993).

Seed

Seed-borne dispersal is a possibility, and whether or not it occurs is important for long-range dispersal (Jarvis, 1988). It has been found to spread on the seeds of a weed host (Rekah *et al.*, 2001).

Glasshouse equipment

Chlamydospores attached to soil particles can be spread about the nursery by people and equipment (Roberts *et al*., 2001).

2.3.2.5   Risk to other protected vegetable crops

Some isolates of the crown and root rot organism are slightly pathogenic to pepper and eggplant, and many legumes are moderately susceptible (Rowe, 1980). Cucumbers and peppers were affected by seedling inoculation, but not all varieties. None of the three lettuce varieties tested with *Forl* developed root lesions (Menzies *et al.,* 1990). The fungus has also been isolated from roots of a number of weeds that may occur in glasshouses, such as shepherd's purse (*Capsella bursa-pastoris*) and chickweed (*Stellaria media)* (Roberts *et al.*, 2001). The reference does not indicate if the weeds showed disease symptoms.

2.3.2.6 <u>Control</u>

Once plants become infected they cannot be cured, losses can only be minimised by avoiding stress. Preventative measures are essential. Management procedures have been reviewed by Jarvis (1988) and Ozbay & Newman (2004), and these should be integrated to reduce the impact of the disease.

Varietal resistance

When *F. oxysporum* f. sp. *radicis-lycopersici* was first recognised, it was found that varieties resistant to races 1 and, or 2 of fusarium wilt showed no resistance to crown and root rot. The disease was originally designated as a new race, race 3 of *F. oxysporum* f. sp. *lycopersici,* although it did not produce the typical wilt symptoms (Rowe, 1980). Inoculation trials by Jarvis and Thorpe in 1976 found some resistant *Lycopersicon* species and hybrids, and a positive correlation between seedling and mature plant susceptibility.

Resistance to *Forl* was recently identified (Scott & Jones, 2000) and is now incorporated into most commercial cultivars, both for greenhouse and field crops (Roberts *et al.*, 2001). A single dominant gene confers resistance. It is tightly linked to the Tm-2 resistance gene to tobacco mosaic virus (Vakalounakis *et al*., 1997). Resistant varieties have tended to produce lower yields than standard varieties (Ozbay & Newman, 2004), although at least one resistant field variety has equivalent yield to susceptible cultivars (McGovern, 1994). Isolates able to overcome *Forl* resistance were reported in Canada (Elmhirst, 1997) affecting crops of the variety Trust on four nurseries.

Grafting susceptible varieties onto resistant rootstocks is widely used in many Mediterranean countries and North America, particularly as tomato grafting promotes growth, increases yield and improves fruit quality (Ozbay & Newman, 2004).

Hygiene

Spores are found on decaying tomato waste heaps (Jones *et al.,* 1991) and could be carried back into the house by flies. Any waste should be covered over and disposed

of quickly off-site. The glasshouse must be cleaned thoroughly and equipment disinfected before reuse, particularly if it will come into direct contact with the growing media. Any diseased plants need to be completely removed (Ozbay & Newman, 2004). Surface disinfection of trays with a quaternary ammonium salt was found to be ineffective, but control was successful after Styrofoam trays were steamed at 71°C for 45 minutes (McGovern, 1994).

Control of fungus gnats may reduce or slow the spread of fusarium in an infected crop (Gillespie & Menzies, 1993).

Chemical

Soil steaming followed by fungicide drenching has proved effective for soil-grown crops (Rowe & Farley, 1978; Jones *et al.,* 1991), but captafol cannot now be used. There are no currently approved pesticides (including Specific Off-Label Approvals (SOLAs) with a label recommendation for use against fusarium on glasshouse tomatoes (Whitehead, 2005). However, it is likely that carbendazim applied for control of verticillium wilt will give some control. Treatment of protected tomato grown in either inert substrates, soil or peat bags, or recirculating hydroponic solution is permitted under SOLAs 1010/2004 (Delsene 50 Flo) and 1207/2004 (Cleancrop Curve). Carbendazim reduced disease severity by over 50% when used on inoculated tomato seedlings at >50 ppm, but had little effect at lower concentrations (Omar *et al.,* 2005).

Soil steaming (80 to 85°C for 4 to 6 hours) on its own has led to problems with recontamination of the soil with airborne fusarium crown and root rot micro-conidia. Micro and macro-conidia germinate and grow rapidly on the nutrients released during the steaming process. When these nutrients are exhausted the mycelium converts to chlamydospores that only germinate in the presence of host root exudates or other suitable nutrients. It is necessary to ensure that there is good sanitation in and around the glasshouse to remove airborne infection sources (Rowe *et al.,* 1977).

Methyl bromide + chloropicrin formulations have commonly been used to fumigate soil for the control of fusarium crown and root rot, but is known not to give complete

eradication. The production and use of methyl bromide is being phased out, though alternatives such as 1,3-dichloropropene, chloropicrin, dazomet and fosthiazate have been shown to provide some crown and root rot control (Ozbay & Newman, 2004). Metam sodium injected into Florida fields failed to reduce disease, and application to the surface produced variable results. However, rotovation into the beds produced reductions in incidence equivalent to those achieved by methyl bromide + chloropicrin (McGovern *et al.,* 1998). Metam sodium, particularly when combined with solarisation, and an iodine based compound, Plantpro 45, have both given as good control of crown and root rot as methyl bromide + chloropicrin (Ozbay & Newman, 2004). Equivalent control was also given in a Greek glasshouse using commercial preparations of sulphur + *Thiobacillus* spp. at 100 g/m² and 150 g/m² of chitin in combination with soil solarisation (Bourbos & Venetis, 1999).

In Turkish tests, mixtures of various plant extracts, marketed as Akse-Bio-1 to 4, have been added to tomato drip-watering systems and irrigation water to control fungi, bacteria, nematodes and insects in field and glasshouse crops. (Tuzun & Yegen,1999). The preparations show direct activity by inhibition of fungal growth and sporulation of *Fusarium* spp. and other soil pathogens. The extracts indirectly increase the beneficial microbial population, and activate the plant's own defence mechanisms (Yegen & Tuzun, 1999).

Cultural

The production of disease-free transplants is of the utmost importance in control of crown and root rot. Introduction of the pathogen to healthy transplants in a rockwool system appears to be much less significant, as delayed initiation of the disease can mean that normal yields are still possible (Mihuta-Grimm *et al.,* 1990).

Transplanting should be done without tissue damage, as these wounds are likely to be sites for infection, and when soil or media is above 20°C, as the fungus grows best from this temperature down to 10°C. Direct seeding produces a higher frequency of infection than transplanting (Ozbay & Newman, 2004).

Growing conditions should be modified, if possible, to avoid low soil pH, use of ammoniacal nitrogen, and to prevent waterlogging as these exacerbate the disease (Roberts *et al.,* 2001). Crown rot is more likely to occur where the soil contains high levels of chloride salts (Ozbay & Newman, 2004). Partial management of fusarium crown and root rot could be developed based on host nutrition. Disease severity was less in tomatoes after two weeks in nutrient solution including nitrate nitrogen, and significantly increased in solution including ammonium-nitrogen. The presence or absence of other macro- and micro-elements also affects disease severity (Duffy & Defago, 1999).

The use of *Chamaecyparis obtusa* bark fibre slabs in place of rockwool are reported to greatly reduce the incidence of fusarium crown and root rot, mainly because of fungal inhibition from volatile oils and non-volatile substances in the bark (Yu & Komada, 1999).

Infected plants produce prolific adventitious roots above the basal stem lesion (with more produced the greater the degree of stem girdling), and mounding of soil or compost around the base of affected soil-grown plants to encourage new root production should delay the death of the plant sufficiently to produce at least some crop (Jarvis & Thorpe, 1976).

In some situations rotation out of tomatoes to a non-host such as lettuce may be possible, avoiding alternative hosts such as aubergine (eggplant) and pepper. However, the fungus can survive in soil for many years (Ozbay & Newman, 2004). Planting a lettuce crop into steam sterilised soil and incorporating its residues into the soil before planting tomatoes over two years gave control of crown and root rot. The pathogen was still present in the soil, but the lettuce seemed to decrease infection in tomato. This might have been either through the release of biologically active materials from the lettuce, or by enhancing microflora activity (Jarvis & Thorpe, 1981).

Solarisation, when the soil is covered with clear plastic or a mulch during a 2 to 8 week period with plentiful solar radiation, results in both physical and biological

processes to control pathogens and other soil pests. It has been shown to reduce populations of *Forl* down to a depth of 5 cm. Solarisation and biofumigation with bovine manure in a closed glasshouse has reduced *Forl* chlamydospore viability (Ozbay & Newman, 2004).

Biological

Pseudomonas fluorescens WCS365 is an excellent competitive coloniser of tomato root tips after bacterisation of seed or seedlings, giving some control of *Forl.* The bacterial cells show a chemotactic response (i.e. respond to a gradient in concentration of a chemical) towards fusaric acid, a secondary metabolite secreted by *Fusarium* strains, with a positive correlation between chemo-attractant activity and fusaric acid level. Fusaric acid, probably together with other metabolites, thus plays a role in the bacterial colonisation of the hyphae (De Weert *et al.,* 2004). Tomatoes transplanted into rockwool cubes and given nutrient solution inoculated with both *Forl* and Pseudomonads had a significantly reduced extent of crown and root after 21 days (Sharifi-Tehrani *et al.,* 1999).

In the USA, *Trichoderma harzianum* (as Plantshield) was applied to glasshouse tomatoes either by soaking the rockwool cubes at sowing, or the rockwool blocks into which seedlings were transplanted at 5 weeks old. The blocks were inoculated with *Forl* two weeks after transplanting. *T. harzianum* applied at transplanting was the more effective application. There was a reduction of at least 73% in disease incidence and a 48% reduction in disease severity (Ozbay *et al.,* 2001; Ozbay *et al.,* 2004).

Ozbay and Newman (2004) have reported research on a number of biological control agents. *Trichoderma harzianum, Aspergillus ochraceus, Penicillium funiculosum, Glomus intraradices, Streptomyces griseovirdis* (as Mycostop)*, Bacillus subtilis, Bacillus pumilus* and *Paenibacillus macerans* have all reduced fusarium crown and root rot. *T. harzianum* and *P. macerans* mixed into compost prior to seed sowing enhanced transplant growth in the glasshouse and suppressed crown and root rot (Datnoff & Pernezny, 2002). Application of non-pathogenic *Fusarium oxysporum* and *F. solani* has also produced control, although Louter & Edgington (1990) still saw cortical discolouration in glasshouse tomatoes with cross-protection by *Fusarium*

oxysporum, although plants did produce larger, heavier fruit. Isolates of *Fusarium culmorum*, *Penicillium brevicompactum* and *P. crustosum* tested in soilless culture increased tomato yield and reduced crown and root rot incidence and severity in an inoculated, but not a naturally infected trial (Menzies & Ehret, 1997). Hypovirulent binucleate *Rhizoctonia* have also given partial control (Muslim *et al.,* 2003). As noted previously, none of the products mentioned above are approved for use to control fusarium diseases of tomato in the UK.

The bioactive oligosaccharide chitosan, derived from crab-shell chitin, reduced fusarium crown and root rot when applied either as a seed coating or a substrate amendment. Foliar application of oligandrin (isolated from *Pythium oligandrum*) to tomato plants restricted fungal growth to the outer root tissues. Wall appositions were enlarged at infection sites. There was induced systemic resistance with sensitisation of the plants so that they reacted more efficiently to fungal attack by the accumulation of fungitoxic compounds at the sites of attempted pathogen penetration (Benhamou *et al.,* 2001). Chitosan in combination with *Bacillus pumilus* is even more effective than using either bio-control agent alone, as pathogen growth becomes mainly restricted to the root epidermis and higher amounts of callose (a cell wall deposit) accumulate in the root tissues. The plants also reacted by producing an opaque matrix along the root wall junctions in most intercellular spaces (Benhamou, 1999).

Peat moss amended with pulp and compost from paper mill residues compost substantially reduced crown and root rot associated symptoms. Physical barriers to fungal penetration are formed in the plant, including callose-enriched wall appositions and osmiophilic deposits around the sites of pathogen ingress. However, amending this compost with *Pythium oligandrum* resulted in a considerable reduction in disease incidence as the extent and magnitude of the cellular changes induced by the compost was substantially increased (Pharand *et al.,* 2002).

Integrated control
Isolates of the bacteria *Bacillus megaterium* and *Burkholderia cepacia* reduced severity of fusarium crown and root rot in seedling bioassays. An integrated treatment of *B. megaterium* C96 combined with carbendazim at 10 ppm suppressed disease to

below 20% of that obtained when carbendazim was used alone at 100 ppm (Omar *et al.,* 2005).

2.3.3   Fusarium stem rot

A fusarium stem rot caused by *F. merismoides* occurred in Kent in 1984 (Fletcher & Lord, 1985). It was widespread during one season on one nursery with occasional reports from other nurseries. However, it has not been reported in the last 10 years. Symptoms were reddish brown stem lesions (2-16 x 2 cm) with a dark edge. They were slightly sunken and did not penetrate more than 2 mm into the stem. The lesions did not result in plant death. Pathogenicity of *F. merismoides* to tomato was confirmed when fresh leaf scars were inoculated. This fungus has also been associated with diseases of other crops including potatoes (Jamalainen, 1955).

3. GUIDELINES FOR DISEASE MANAGEMENT

3.1 Cucumber wilt

- Ensure that the cause of any wilting is diagnosed correctly and promptly. Abundant pink or orange sporulation at or between stem nodes serves to distinguish fusarium from verticillium wilt, but only occurs when fusarium is well established in a plant.

- Susceptible varieties can be grafted on to the resistant rootstock *Cucurbita ficifolia*, and this may be justified where there is a persistent problem. This method was successful for control of fusarium wilt in soil-grown crops. Resistant varieties are not available.

- If a disease outbreak is spotted at an early stage, all affected plants should be carefully removed, sealed in a bag, taken out of the crop, and disposed of away from the nursery.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop.

- In substrate crops either ensure effective disinfection or, preferably, replace the growing medium. For soil crops, unless there is evidence of natural suppression of the disease steam sterilisation may be the only effective option available to eliminate the pathogen from the soil. This is only necessary if resistant rootstocks/varieties are not being used.

- For conventional (non-organic) crops drenches of carbendazim (SOLAs 1479/1995, 1005/2004, 1211/2004 and 1824/2005) should provide additional protection and give some suppression of the disease but should not be relied on alone due to the risk of possible resistance development from repeated use.

3.2 Pepper fruit and stem rots

Control measures for the fruit and stem rots caused by *Fusarium solani* and *F. oxysporum* are similar.

- Maintain a high level of hygiene in the glasshouse, with particular attention to regular removal of fallen and aborted fruit. Abundant sporulation of *F. oxysporum* was found on aborted fruit in one crop.

- Ensure fruit, leaves and axillary shoots are removed with a clean sharp knife. Evidence from Canada indicates a greater disease risk of *F. solani* stem rot where torn, ragged wounds are present.

- Where fusarium stem rot has led to irreversible wilting or extensive stem lesions, remove the affected plants from the crop promptly and carefully.

- Where fusarium is a widespread and persistent problem, cut out nodal lesions. Also, during periods of the year when the disease is increasing, consider re-cutting all stem wounds (i.e. including those where there are no visible lesions) in order to remove any incipient lesions.

- Wash and disinfect hands, knives and any other equipment used after handling infected material.

- Cover any containers containing reject fruit or crop debris to minimise the risk of aerial spread or insect transmission from fusarium sporulation on crop waste. Empty debris containers regularly and clean them as required.

- Position the dripper so that there is no build up of 'salts' around the stem base. Infection is reported to develop in tissues damaged by fertiliser scorch.

- Monitor irrigation carefully so that there is no excessive watering. There are reports that excessive watering may stress plants through oxygen depletion, resulting in *F. solani* shoot lesions.

- Control the glasshouse environment to avoid prolonged periods above 90% RH. This has been shown to reduce ascospore germination of *Nectria hae*matacocca, the sexual stage of *F. solani* that is sometimes found on infected plants.

- Seek to grow a balanced crop and avoid environmental extremes. Symptom development is reported to be triggered by various `stress` factors, including a high fruit load and high temperatures.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop. It is essential that all debris is removed from the house and taken away from the nursery or destroyed.

3.3 Tomato wilt

- Ensure accurate laboratory diagnosis of the cause of any wilting. The symptoms of wilt caused by *F. oxysporum* f. sp. *lycopersici* are not easily distinguished from those of verticillium wilt, fusarium crown and root rot, or even *Phytophthora* root rot.

- There are a number of races of *F. oxysporum* f. sp. *lycopersici.* Where possible choose a variety or rootstock with resistance to races 1 and 2; both of these races are known to be present in the UK. Use of a resistant variety is likely to be the most effective means of disease control.

- If the third race of *F. oxysporum* f. sp. *lycopersici* is confirmed in the UK, consider using grafted plants on a rootstock reported to be resistant to race 3 (e.g. Oxyfort, Trifort or Zodiac).

- If feasible, avoid high temperatures in the glasshouse. Fusarium wilt develops best at high root temperatures (25-28°C).

- Fungicide drench treatment to the roots with carbendazim (SOLAs 1479/1995, 1005/2004, 1211/2004, and 1824/2005) may give some control, but this treatment is likely to be only partially effective.

- Where irrigation water is abstracted from an uncovered reservoir, where drainage water is collected and recycled, and especially where there is considered to be a high risk of fusarium wilt, ensure that the irrigation water is treated to kill fusarium spores. Experimental work indicates that heat, UV light, ozone, microfiltration and possibly slow sand filtration, when operating effectively, are effective against the pathogen and can largely prevent dissemination.

- Carefully remove infected plants from the crop before any sporulation occurs on the stem base. Seal affected plants in a bag before taking them through the crop. Although sporulation on stems is rare, if it occurs it could lead to extensive disease spread through aerial dissemination of spores.

- After an outbreak of fusarium wilt, thoroughly clean the glasshouse and equipment prior to introduction of the new crop. *F. oxysporum* f. sp. *lycopersici* has been isolated from the glasshouse structure, presumed to have arisen from aerially dispersed spores. Resting spores develop in affected tissue and can persist for several years; therefore it is important to remove all crop debris from the site.

- Seed suppliers should routinely monitor seed crops for *Fusarium* pathogens and only harvest seed from those crops found to be free of the disease. Seed-borne infection is known to occur and is a possible source of infection.
- The use of nitrate nitrogen rather that ammoniacal nitrogen is reported to slow the development of fusarium wilt.

4. PRIORITIES FOR RESEARCH AND TECHNOLOGY TRANSFER

4.1 Cucumber

2.1. Collect representative isolates of *F. oxysporum* from UK crops and determine by pathogenicity tests with appropriate hosts and differential cultivars of cucumber the occurrence of races 1, 2 and 3 of *F. oxysporum* f. sp. *cucumerinum* in the UK and whether or not *F. oxysporum* f. sp. *radicis-cucumerinum* is present in the UK.

3.2. Alert UK growers to the symptoms and risk of fusarium root and stem rot (*F. oxysporum* f. sp. *radicis-cucumerinum*) through a presentation at the CGA Conference or by other means.

4.3. Investigate the susceptibility of cucumber varieties commonly grown in the UK to the races of *F. oxysporum* f. sp. *cucumerinum* present in the UK, by collation of information from seed suppliers and pathogenicity tests where required.

5.4. Investigate the potential occurrence of pathogenic *Fusarium* species on cucurbit seed in the UK.

6.5. Generate baseline sensitivity data using both type culture and current isolates of *F. oxysporum* f. sp. *cucumerinum* to carbendazim and monitor isolates for resistance to the fungicide.

7.6. Evaluate the efficacy of alternative fungicides (e.g. strobilurins, triazoles) against *F. oxysporum* f. sp. *cucumerinum*.

8.7. Investigate the efficacy of carbendazim applied as a stem spray for control of *F. oxysporum* f. sp. *cucumerinum*.

4.2 Pepper

1. Confirm the pathogenicity of *F. oxysporum* isolated from pepper fruit to pepper (project now agreed: PC 232a), investigate disease epidemiology and devise control measures.

2. Investigate the occurrence and pathogenicity to pepper of *Fusarium* species on pepper seed.

3. Generate baseline sensitivity data for isolates of *Fusarium* from pepper to carbendazim.

4. Determine the efficacy of strobilurin (e.g. azoxystrobin) and other fungicides against fusarium stem rot and fruit rots.

5. Secure isolates of *Fusarium* from roots and stem base of pepper and determine their pathogenicity to some current UK varieties.

4.3 Tomato

1. Collect isolates of *F. oxysporum* (and other species if found) from UK crops with wilt and root rot symptoms and determine by pathogenicity tests on different cultivars of tomato which races of *F. oxysporum* f. sp. *lycopersici* are currently present.

2. If fusarium crown and root rot occurs on allegedly resistant varieties, test isolates to determine if there is a resistance-breaking strain in the UK.

4.4 General

1.

1. Seek to secure photographs of all the main diseases described in this report. Update the industry of fusarium diseases affecting cucumber, sweet and chilli pepper and tomato.

2. Seek to develop effective succession planning of experienced personnel with basic diagnostic and host-pathogen investigative skills.

1.3. Determine the efficacy of promising, synthetic and non-synthetic pesticide treatments against fusarium wilt in soil and hydroponic crops, including essential oils, compost tea and soil amendments.

2.4. Investigate the options for bio-control to avoid the need for fungicide application (and residue risk) and evaluate their efficacy, including the use of non-pathogenic *Fusarium* spp.

3.5. Determine the efficacy of slow sand filtration or similar techniques for the effective disinfection of water and recycled nutrient solution and as an alternative to pesticide application.

4.6. Develop improved diagnostics for rapid identification of *Fusarium* spp. affecting protected crops.

5.7. Develop robust protocols for pathogenicity screening of isolates of *Fusarium* recovered from protected crops.

5. ACKNOWLEDGEMENTS

6. REFERENCES

The following is a list of fusarium references that were consulted during this review. Although they are not all quoted in the text, they are listed here as a reference source for those who wish to do further reading on the subject.

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