Grains National Biosecurity Plan
Threat Specific Contingency Plan

Fusarium oxysporum f. sp. *conglutinans*
Fusarium wilt of canola

September 2007

Prepared by Dr Joe Kochman and Plant Health Australia

The scientific and technical content of this document is current to the date published and all efforts were made to obtain relevant and published information on the pest. New information will be included as it becomes available, or when the document is reviewed.

Contents

1. Pest information/status

1.1. Pest Details – *Fusarium oxysporum* f.sp. *conglutinans* (Wollemw.) Snyder & Hansen – Fusarium wilt of Canola

1.1.1 General information

Taxonomic position – Class: Sordariomycetes; Order: Hypocreales; Family: Nectricaceae

The fusarium wilt pathogen is a fungal organism that infects plants through the root system. Often it will invade the plant through wounds in the roots. The vascular system of the plant is adversely affected by the organism as it grows, thus reducing nutrient and water flow through the plant.

Fusarium wilt is a relatively new disease of canola, with initial reports from Russia in 1998 (Anon, 2005a), Canada in 1998 (Lange *et al.*, 2001) and Argentina in 2002 (Gaetán, 2005) but Subramanian (1970) reported fusarium wilt had been recorded in rape crops, the precursor of the current canola crop. The disease is caused by the fungus *Fusarium oxysporum* Schlechtend ex Fr. f.sp. *conglutinans* (Wollenweb.) Snyd. & Hans. (*Foc*) which has a very wide geographical distribution (Subramanian, 1970).

The genus *Fusarium* is very diverse containing many pathogenic species that can cause plant diseases such as wilts (Beckman, 1987), root, crown, tuber and bulb rots, as well as non-pathogenic saprophytes (Nelson *et al*., 1981). The fungus occurs in most areas of the world (Burgess, 1981) with *Fusarium oxysporum* (*Fo*) being one of the most variable species within the genus (Burgess *et al.*, 1994). The pathogenic forms of *Fo* are grouped into *formae speciales* (f.sp.) or special forms, based on their ability to attack particular host plants. More than 120 *formae speciales* and races have been described within *F. oxysporum* (Armstrong and Armstrong, 1981). These *formae speciales* and races can be distinguished from each other or saprophytic forms by pathogenicity testing and some recently developed molecular tests (Bentley *et al*., 2002). *Formae speciales conglutinans* is able to cause a vascular wilt disease of canola and other *Brassica* crops.

F. oxysporum is a cosmopolitan species occurring in most soils of the world (Burgess *et al*., 1994). Distinguishing characteristics of the species include the production of three types of spores, macroconidia (usually 3-4 celled and 35-70µm long), microconidia (usually single celled, 5-15 µm long and formed in false-heads on short monophialides) and chlamydospores (thick walled resting spores which allow the fungus to survive for long periods without a host). It can be confused with *F. solani* which produces microconidia on long monophialides (Burgess *et al*., 1994). Both these species can cause root and crown rots (Jarvis and Shoemaker, 1978) but only *Fo* causes vascular wilts (Nelson *et al*., 1981, Beckman, 1987). *Fo* can infect roots directly just behind the growing point or through wounds at all stages of plant growth, developing in and occluding the vascular system and then causing wilt symptoms.

Symptoms of Fusarium wilt in many crops include wilting, yellowing, stunted plant growth, necrosis of various plant parts and finally plant death. Lange *et al*., (2001) reported chlorosis and necrosis of stems, vascular discoloration, poor seed set, and premature desiccation with Fusarium wilt of canola in Canada while Gaetán (2005) reported that disease symptoms in canola growing in Argentina included 'yellowing, wilting, stunting, and necrosis of leaf tissue and suppressed root development' and the disease 'appeared in irregular-shaped patches following the rows of plants. The first symptom observed was leaf yellowing followed by an irregular, brown necrosis of the leaf margins. Lesions coalesced to form large necrotic areas that led to severe defoliation beginning with the lower leaves. As the disease developed, a pale brown discoloration girdled the stems that progressed from the basal tissues to the apex. Affected plants were stunted and had small pods with no seeds. Diseased plants eventually collapsed and died'. Hence all parts of the canola plant, roots, stems, branches, leaves and pods are affected and the fungus can be isolated from them.

The disease has not been reported in Australian crops of canola to date (August 2007), even though *Fusarium oxysporum* has been recorded on other *Brassica* crops in South Australia and Victoria (Australian Plant Disease Database, DAR37184 a, VPRI 11382 and VPRI11535a) and *Fo conglutinans* has been recorded causing yellowing and stunting in cauliflower and cabbage in Queensland (Simmonds, 1966). In addition, Li *et al*. (2007) reported that 30 percent of the fungi isolated from roots of canola growing in the Wagga Wagga area of New South Wales were *Fusarium* species including: *F. oxysporum*, *F. acuminatum, F. semitectum, F. solani, F. nivalis* and *F. equiseti*. They confirmed the pathogenicity of all the Fusarium isolates but did not report that any of them caused a wilt disease in the canola plants they examined.

There are several possible reasons why fusarium wilt of canola has not been recorded in Australia. The *Fo conglutinans* in vegetable *Brassica* species may be a different strain to that causing wilt in canola, or the causal fungus may not be present or not present in sufficient concentration in soil for symptoms of the disease to appear. Wang *et al*. (1999) reported that concentration of inoculum of *Fusarium oxysporum* f. sp. *vasinfectum* (*Fov*) in the soil was important for development of fusarium wilt in cotton. They found that fusarium wilt symptoms only developed in cotton when susceptible varieties and more resistant varieties were inoculated with conidial concentrations in excess of 10^5 and 10^6 conidia ml^{-1} respectively. In addition, the environmental conditions during the canola growing season in Australia may not be suitable for the development of fusarium wilt. Fusarium wilt of cotton in Australia is largely confined to irrigated crops with rare occurrence in rain-grown crops, even though these crops are grown in close proximity to one another in some wilt affected areas (Kochman pers. obs.).

Given the large area of canola grown in Australia, with a diversity of soil types and climatic conditions, it is likely that fusarium wilt will be found here at some time and a contingency plan to manage this disease is required. The commonality of management options for most crop fusarium wilt diseases will allow the bundling of this plan for *F. oxysporum* f. sp. *ciceris* (fusarium wilt of chickpea) and *Fusarium solani* (Mart.) Appel & Wollenw. emend. Snyder & Hansen f. sp. *glycines* (soybean Sudden Death Syndrome). Neither of these two pathogens is known to occur in Australia (M J Ryley, pers. comm.).

1.1.2 Life cycle

1.1.2.1 Fusarium oxysporum f.sp. conglutinans and f.sp ciceris

Pathogen inoculum may be present, in soil in the field, on infected crop residues from a previous season or it may be introduced to previously uninfected fields at planting within infected seed. Chlamydospores (thick walled resting spores) or the macro and microspores of *Fo* germinate in response to exudates from seeds and roots and infect behind root tips or through wounds in roots. The fungus grows into the vascular system and produces microspores that move up the stem with the flow of sap. The plant tries to prevent progress of the fungus by blocking the vascular tissue, resulting in the brown discolouration in the stem. Seedlings may either wilt and die or survive, but often with stunted growth. Adult plants may wilt and die, especially under conditions of stress. The fungus multiplies and produces large numbers of spores and chlamydospores on plant residue and in soil to start the life cycle once more. Any conditions which stress the plant increase the risk of infection.

Fusarium wilt is also affected by environmental conditions, primarily soil temperatures, moisture and compaction. In general, any factor which contributes to a reduced rate of root growth increases the plant's susceptibility to fusarium wilt. Warm temperatures (>17°C) encourage wilt disease development, and aggressiveness of *F. oxysporum* seems to peak at 25°C. Dry soil favours development of symptoms, although it does not appear to be absolutely necessary for development of the disease.

High plant populations also increase plant stress and favour infection. Improper cultivation, other soil-borne pathogens, and various herbicides are also known to induce injury of young roots and aggravate fusarium wilt damage. The effect of fusarium wilt is most apparent during blossoming and early pod set when the plant and its productivity are more sensitive to stress. The pathogen is disseminated within and among fields by the movement of contaminated soil by wind, irrigation

water, and on equipment. Pathogenic strains of *Fo* are able to survive for long periods in soil and infected crop residues, either as a saprophyte or as chlamydospores.

1.1.2.2 Fusarium solani f.sp. glycines (Fsg)

The life history of *F. solani* is very similar to that of *F. oxysporum.*

1.1.3 Dispersal

Seed-borne inoculum can allow long range dispersal and introduction of the *Fusarium species* (or new races of the pathogen) into new countries, new areas and new fields. Spores of the fungus can be carried in soil attached to farm machinery and on workers' boots and clothing as well as in soil and infected plant debris in irrigation and flood waters. Hence farm hygiene is important in retarding spread of fusarium wilt as well as other diseases and weeds.

1.2 Affected Hosts

1.2.1 Host range

The host range for *Fo conglutinans* includes most of the *Brassicaceae* with race 1 infecting vegetables such as Brussels sprouts, cabbage, cauliflower *etc*. as well as rape (Subramanian, 1970). *Fo ciceris* appears to be confined to chickpea (Brayford, 1992).

While it is widely accepted that the wilt inducing strains of *F. oxysporum* are specific to the particular crops they cause to wilt, there is much evidence that plants of other crops and weeds may be symptomless hosts (Davis *et al.*, 2006). In addition there are reports of *Fusarium oxysporum* f.sp. *vasinfectum* (the causal agent of cotton fusarium wilt) causing wilt symptoms in a range of hosts (Davis *et al*., 2006).

A particular strain of *Fsg* causes the sudden death syndrome in soybeans (Shaner *et al.* 1998).

1.2.2 Geographic distribution

Fusarium oxysporum f.sp. *conglutinans* occurs in; Africa, Asia, Australasia, Europe and North, Central and South America (Subramanian, 1970). Fusarium wilt of canola has not been reported in Australian crops of canola to date (August 2007), even though *Fo conglutinans* has been recorded on other *Brassica* crops in South Australia and Victoria (Australian Plant Disease Database, DAR37184 a, VPRI 11382 and VPRI11535a) and *Fo conglutinans* has been recorded causing yellowing and stunting in cauliflower and cabbage in Queensland (Simmonds, 1966). In addition, Li *et al.* (2007) reported that 30 percent of the fungi isolated from roots of canola growing in the Wagga Wagga area of New South Wales were *Fusarium* species including *F. oxysporum, F. acuminatum, F. semitectum, F. solani, F. nivalis and F. equiseti*. They confirmed the pathogenicity of all the *Fusarium* isolates but did not report that any of them caused a wilt disease in the canola plants they examined.

1.2.3 Symptoms

1.2.3.1 Fusarium wilt of canola and chickpea (Fusarium oxysporum f. sp. conglutinans and f.sp ciceris)

External symptoms include stunted growth and leaves appear dull and wilted, before yellowing, and necrosis of leaf tissue. Root, pod and seed development may also be reduced (Lange *et al.*, 2001, Gaetán, 2005). Similar symptoms occur in chickpeas (Haware, 1990).

Internal symptoms can be revealed by lengthwise cutting of the stem of affected plants to show continuous brown discolouration of the vascular system and sometimes the entire stem. External symptoms do not always reflect the extent of discolouration in the stem. Sometimes the discolouration is visible in one side of the stem. Again similar symptoms occur in chickpeas (Haware, 1990)

1.2.3.2 Soybean Sudden Death Syndrome (Fusarium solani f.sp. glycines) (Fsg)

The foliar symptoms of sudden death syndrome (SDS) may appear any time from flowering through to pod fill (Shaner *et al.*, 1998). The first visible symptom is the appearance of small, yellowish, interveinal chlorotic blotches in leaves, generally in the middle of the canopy. These quickly increase in size and number and the tissue within the blotches becomes brown and dies. The leaf veins, petioles, and stems remain green for some time after most of the interveinal leaf tissue has died. Entire plants may become affected and leaf blades drop, leaving erect, somewhat green petioles attached to the stems. The foliar symptoms of SDS may be confused with some other diseases, such as brown stem rot or stem canker. The root and lower stem tissues of plants exhibiting these symptoms must be closely examined to confirm a diagnosis of SDS (Shaner *et al.*, 1998).

1.3 Entry, establishment and spread

Entry potential: **MEDIUM**
There is considerable evidence from other crop fusarium wilt studies that pathogenic strains of *Fo* have been introduced into countries in seed and infected plant debris (Davis *et al.* 2006). These are possible means of long range introduction to Australia of *Fo conglutinans*, *Fo ciceris* and *Fs glycines* strains that cause wilt and sudden death in canola, chickpea and soybean respectively.

There is further evidence that in Australia *Fo vasinfectum* strains developed from indigenous populations of *Fo* (Davis *et al.*, 1996, Bentley *et al.*, 2000, Kochman *et al.*, 2002) following the cultivation of cultivars which were subsequently found to be very susceptible to the pathogen. This could happen with canola and chickpea wilt pathogens and the sudden death syndrome in soybeans in Australia.

Establishment potential: **HIGH**
Fusarium wilt of canola can persist in field for very long periods, and is considered to be a permanent introduction in affected fields overseas.

Fusarium oxysporum and *Fusarium solani* occur widely and are common in Australian soils. Hence, canola and chickpea fusarium wilt inducing races and soybean sudden death inducing strains could become established in Australia if introduced from overseas in infected seed, infected soil or plant debris attached to imported second-hand farm machinery.

Spread potential: **HIGH**
The potential for dispersal on machinery and clothing, in overland flood flows and the possibility of seed-borne dispersal would facilitate rapid dispersal and establishment of the diseases across production regions.

***Economic impact*: HIGH**
Once introduced, pathogenic strains of *Fo conglutinans, Fo ciceris* and *Fs.glycines* are almost impossible to eradicate and failure to control the build-up of the diseases could result in fields becoming unsuitable for canola, chickpea or soybean production respectively.

***Environmental impact*: NEGLIGIBLE**
The two races of *F oxysporum* and one *Fusarium solani* are not known to attack native vegetation and both species are widespread in Australian soils (Burgess *et al.*, 1994).

***Overall risk:* MEDIUM**
The introduction and spread of new races of *Fo conglutinans* would have a very significant impact on the Australian canola industry and its participants, particularly as canola is grown in large areas of NSW, Victoria, South Australia and Western Australia. Chickpea and soybeans crops occupy smaller areas but the introduction *Fo ciceris* and *Fs glycines* would have significant impact on each crop respectively.

The most effective means of managing fusarium diseases in crops is to grow disease resistant cultivars so any introduction of these pathogens would require significant investment in assessment of germplasm for reaction to these new races or strains and subsequent plant breeding efforts to develop resistant varieties to these new races or strains.

1.4 Diagnostic information

1.4.1 Diagnostic protocol

1.4.1.1 Fusarium oxysporum f.sp. conglutinans and f.sp ciceris

Primary identification is on the basis of symptoms that include stunting, wilting, chlorosis, brown discolouration within the stem and plant death. The fungus *Fo* is readily isolated from infected plants. Distinguishing characteristics of the species include the production of three types of spores, macroconidia (usually 3-4 celled and 35-70µm long), microconidia (usually single celled, 5-15 µm long and formed in false-heads on short monophialides) and chlamydospores (thick walled resting spores which allow the fungus to survive for long periods without a host) (Burgess *et al.*, 1994). As saprophytic and pathogenic forms of *Fo* are indistinguishable morphologically, pathogenicity tests will need to be conducted to ensure the isolate is able to re-infect and cause wilt symptoms in canola (for *Fo conglutinans*) and chickpeas (for *Fo ciceris*) respectively.

There are a number of races reported for each form species with up to four in *Fo conglutinans* and race 1 infecting the *Brassica* vegetables and rape (now canola) (Subramanian, 1970). Jimenez-Gasco *et al.*, 2004) reported *Fo ciceris* consists of two pathotypes, one causing yellowing and wilting symptoms and eight races (races 0, 1B/C, 1A and 2-6) of diverse geographical distribution. Molecular diagnostic tools have been widely used to identify pathogenic races within *Fo* form species.

1.4.1.2 Fusarium solani f.sp. glycines

Primary identification is on the basis of symptoms which result in the sudden death of soybeans with *F. solani* consistently isolated from infected plants. Like *F. oxysporum, F. solani* produces three types of spores, micro, macro and chlamydospores but one of the main distinguishing features between the two species is that the microconidia are borne in false-heads on long monophialides in *F. solani* and short monophialides in *F. oxysporum* (Burgess *et al.*, 1994).

Shaner *et al.* (1998) reported that two races of *Fsg* infect soybean and cause root rot but only one strain, sometimes called "form A" or the "blue strain", produces toxins that are rapidly translocated from the roots to the foliage to the causes the sudden death syndrome. The other is called "form B" and causes some root rot, but not sudden death.

1.4.2 Reference documents

- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

1.5 Response checklist

1.5.1 Checklist

Guidelines for Response Checklists are still to be developed. The following checklist provides a summary of generic requirements to be identified and implemented within a Response Plan:

- ☐ Destruction methods for plant material, soil and disposable items
- ☐ Disposal procedures
- ☐ Quarantine restrictions and movement controls
- ☐ Decontamination and farm cleanup procedures
- ☐ Diagnostic protocols and laboratories
- ☐ Trace back and trace forward procedures
- ☐ Protocols for delimiting, intensive and ongoing surveillance
- ☐ Zoning
- ☐ Reporting and communication strategy

1.5.2 Reference documents

- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

1.6 Delimiting survey and epidemiology study

Apart from a few industries, there appears to be little routine crop disease survey activity in Australia. Hence, it is most likely that a grower or private consultant will be the first to report some suspicious new symptoms in a crop which may indicate the occurrence of a new disease. Unlike diseases caused by airborne pathogens that have the ability to spread rapidly (*e.g.* the rusts, ergots), soil-borne diseases such as those caused by *Fusarium* tend to spread more slowly.

Delimiting surveys should comprise local surveys around the area of initial detection concentrating on areas of poor growth.

There are a number of survey procedures which have been developed to identify diseases in field crops that are applicable to canola, chickpeas and soybeans. Some, such as the Operational Procedure – Plant Biosecurity Plant Health Certification Services Manual for 'Inspection of seed crops to meet overseas import conditions for seed for sowing' (Anon 2005b), have

been developed by government agencies such as the Queensland Government, Department of Primary Industries and Fisheries. The Australian Cotton Cooperative Research Centre (now the Cotton Catchment Communities CRC) has developed an 'Integrated Disease Management' (IDM) package which includes information on principles of disease management, disease identification, assessing diseases on the farm and how to reduce the risk of spreading diseases both within farms and regionally. The IDM package has a comprehensive section on fusarium wilt of cotton.

1.6.1 Sampling method

Field inspections should include a transect or track through a field that allows representative sampling of the entire field with, on average, one inspection site of 10 m^2 of plants per hectare. Plants should be assessed for yellowing, stunting, wilt and plant death. Stems should be broken or sliced to reveal vascular discolouration in case plants are not showing external symptoms.

Aerial inspections would also be useful as fusarium wilts often occur in patches or along rows in fields and these patches are usually visible from the air (Kochman, pers. obs.). Remote sensing and infra-red technologies have also been used to identify bare areas in cotton fields. Field inspections of these areas are needed to confirm that they are caused (in fact) by a *Fusarium* pathogen.

1.6.2 Epidemiological study

There are many factors that affect the development of fusarium wilt diseases in fields. These include: the presence of virulent strains in the soil, susceptibility of the crop varieties, soil type, soil fertility, climatic conditions, irrigated or non-irrigated crops and interactions with other soil borne microorganisms. Inoculum densities in the soil are also important as disease symptoms may not be apparent when there are low levels of the pathogenic strains in the soil (Davis *et. al*., 2006).

Symptoms may occur in a small number of plants or in large areas in any part of the field. Lange & McLaren (2002) found that, in a targeted survey of 12 canola fields in Manitoba, Canada, the disease was not seen in the random 100-plant samples collected from three of the 12 fields even though it was present in other areas of these fields highlighting the irregular nature of occurrence of the disease. Hence, as well as random transect surveys, aerial surveys would also be useful in identifying bare, dead, stunted or wilted areas in fields which would then need to be checked to confirm or deny the presence of the pathogen.

The disease can occur at any stage of plant growth, particularly when crops are stressed. Factors such as, high plant populations, improper cultivation, other soil-borne pathogens, and various herbicides are all known to induce injury of young roots, causing plant stress. These can aggravate fusarium wilt damage.Early infections can kill seedlings soon after emergence, leaving bare areas in the crop. Later infection can cause wilting, yellowing and death of adult plants.

The effect of fusarium wilt is most apparent during blossoming and early pod set when the plant and its productivity are more sensitive to stress. .

1.6.3 Models of spread potential

The pathogen is disseminated within and among fields by the movement of contaminated soil by wind, irrigation water, overland flood flows and on equipment and workers' clothing. Fusarium wilt in cotton was likely to have been spread from the Downs to the St George area of Queensland (a distance of some 400 km) in flood flows because the first fields affected in the St George area were irrigated from flood flows. Pathogenic strains of *Fo* are able to survive for long periods in soil and infected crop residues, either as a saprophyte or as chlamydospores. Hence the importance of minimising overland flood flows over any areas identified as affected.

Seed production must not occur in any affected areas to minimise the possibility of seed transmission of the disease to new areas.

1.6.4 Pest Free Area (PFA) guideline

- Statistical field survey for symptoms on host plants.
- Plant and soil sampling using appropriate diagnostic tests.
- Survey around irrigation systems or waterways that may have transported chlamydospores.
- Aerial inspection or remote sensing should also be used where possible, with suspect patches inspected and sampled to confirm or deny the presence of the pathogen.

1.6.5 Reference documents

- *PLANTPLAN, Appendix 3: Sampling procedures and protocols for transport, diagnosis and confirmation of EPPs – Plant Health Australia (2006)*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

1.7 Availability of control methods

Once introduced and established, pathogenic strains of *F. oxysporum* and *F solani* can survive in soil for long periods, even in the absence of crop hosts, effectively preventing eradication. Hence containment procedures to retard the spread of the disease are required and suitable procedures have been developed by the Australian cotton industry following the discovery of fusarium wilt in cotton in 1993 (Kochman, 1995). Procedures developed to retard the spread of cotton fusarium wilt can be directly applied to retard the spread of fusarium wilt in canola, chickpea and soybean sudden death syndrome in soybeans. They are described in the cotton IDM guidelines (Allen et al., updated 2003) and other publications available on the Cotton Catchment Communities website (www.cotton.crc.org.au) under the disease and microbiology section.

1.7.1 General procedures for control

Keep traffic out of affected areas and minimize it in adjacent areas. Stop irrigating affected (irrigated crops) areas and use bunding to divert overland flood flows around them (both irrigated and dryland crops).

Adopt best-practice farm hygiene procedures to retard the spread of the pathogen between fields and adjacent farms.

Ensure that planting seed production does not take place on affected farms and do not use seed from these farms to plant the next crop as *Fusarium* species can be seed borne.

1.7.2 Control if small areas are affected

Pull out the affected plants, as well as healthy plants 1-2 metres into the area surrounding the patch and burn them in the patch. Particular care must be taken to minimize the transfer of infected soil from the area. Raking and burning the whole field at this stage is NOT an option as this procedure is likely to spread the pathogen over the field.

1.7.3 Control if large areas are affected

Kill any surviving plants in the area, preferably with herbicides and leave them to die in place. Once the dead plants have broken down, sow an alternative crop such as a cereal or grass pasture to prevent erosion. All equipment used on the site should be thoroughly cleaned down, with products such as a farm degreaser or a 1 % bleach solution and washed down with a pressure cleaner on the affected farm. The clean down procedure should be carried out on hard standing or preferably a designated wash-down area to avoid mud being recollected from the affected site onto the machine. A wash-down pad design and operational procedures are described in the cotton IDM package.

1.7.4 Cultural control

Fusarium wilt of canola can be minimized by all good disease prevention measures, such as rotation with non-host species, *Brassica* weed control in cereal crops, and control of annual weeds in crop borders and headlands. However, as the causal fungus can survive in soils for long periods of time, there still can be some residual disease inoculum even with good management practices (Lange and McLaren, 2002).

1.7.5 Host plant resistance

Resistant cultivars offer the best hope for control of fusarium wilt. Genetic resistance already exists in *Brassica napus*, since a number of varieties such as 46A76, SW RideR, Quantum and Nexera 710 appear to be resistant or moderately resistant to fusarium wilt.

1.7.6 Chemical control

Disease control methods with a poorer chance of success than genetic resistance include seed applied and foliar fungicides, crop rotation, and sanitation. Obtaining useful levels of control with seed-applied fungicides will probably be difficult because the disease appears to be most aggressive late in the growing season, long after seed-applied fungicides can reasonably be considered effective. However, seed- treatments may reduce losses by improving plant stands. Foliar fungicides tend to be expensive, and must be applied as protective sprays well before symptoms become apparent (Lange and McLaren, 2002).

Fumigants provide control of Fusarium wilt (but not eradication), and are not cost effective for routine use in canola.

1.7.7 Reference documents

- *PLANTPLAN – Plant Health Australia (2006)*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

2 Course of Action – Eradication Methods

2.1 Destruction strategy

2.1.1 Destruction protocols

- Disposable equipment, infected plant material or soil should be disposed of by autoclaving, high temperature incineration or deep burial.
- Fumigation with methyl bromide is unlikely to control fusarium of canola as this method has been unsuccessful for eradication of fusarium wilt of cotton.

2.1.2 Decontamination protocols

- Machinery, equipment, vehicles in contact with infected plant material or soil should be washed using high pressure air or water in a designated wash down area. Any waste water, soil or plant residues should be contained.
- Disposable overalls and rubber boots should be worn when handling infected soil or plant material. Boots, clothes and shoes in contact with infected soil or plant should be disinfected at the site or double bagged to remove for cleaning.
- Skin and hair in contact with plant material or soil should be thoroughly washed.

2.1.3 Priorities

- Confirm the presence of the pathogen.
- Prevent movement of vehicles and equipment through affected areas.
- Inform all groups in the Industry.
- Attempt to control overland flood flows.

2.1.4 Plants, by-products and waste processing

- Seed harvested from infected plants and infected plant material should be destroyed by (enclosed) high temperature incineration, autoclaving or deep burial (in a non-cropping area).

2.1.5 Disposal issues

- Once introduced and established, pathogenic strains of *F. oxysporum* and *F solani* can survive in soil for long periods, even in the absence of crop hosts, effectively preventing eradication.
- Particular care must be taken to minimize the transfer of infected soil from the area. Raking and burning the whole field at this stage is NOT an option as this procedure is likely to spread the pathogen over the field.

2.1.6 Reference documents

- *PLANTPLAN, Appendix 18: Disinfection and decontamination – Plant Health Australia (2006).*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

2.2 Quarantine and movement controls

2.2.1 Quarantine priorities

- Seed harvested from infected plants.
- Infected plant material and soil.
- Machinery, equipment, vehicles and disposable equipment in contact with infected plant material or soil.
- People (including boots and clothing) in contact with plant material or soil.

2.2.2 Movement control for people, plant material and machinery

As with any crop disease the causal agents of any new symptoms need to be identified and proven to be the cause of those symptoms (Koch's postulates completed). Once symptoms of a fusarium wilt disease are observed the pathogen is usually well established in the soil so the possibility of eradication is most unlikely. Hence any zoning, quarantine or movement controls will pertain to containment and management of the pathogens.

The industry affected will need to be informed of the location and extent of the disease occurrence. People, vehicle and machinery movements, from and to affected farms, will need to be controlled to ensure that infected soil or plant debris is not moved off-farm on clothing, footwear, vehicles or machinery. Clothing and footwear worn at the infected site should not leave the farm or they are thoroughly disinfected, washed and cleaned before wearing off-farm.

Vehicles and machinery used on the site should be thoroughly cleaned down, with products such as a farm degreaser or a 1% bleach solution and washed down with a pressure cleaner prior to leaving the affected farm. The clean down procedure should be carried out on hard standing or preferably a designated wash-down area to avoid mud being recollected from the affected site onto the machine. Any crop seed from the affected site should not be used for planting seed.

After the initial identification of an incursion, or new pathogen strain development, the containment procedures will, in the main, need to be controlled by the industries affected because the largest impact of the diseases is likely to be on production. The industry will need to develop information packages for its growers, detailing; how to identify the diseases, procedures to reduce the risk of spreading the disease within affected farms and measures that growers can take to minimize the risk of introducing the disease.

2.2.3 Reference documents

- *PLANTPLAN – Plant Health Australia (2006)*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia*.

2.3 Zoning

2.3.1

The size of each quarantine area will be determined by a number of factors, including the location of the incursion, climatic conditions and the proximity of the infected property to other infected properties.

2.3.2 Destruction zone

All host plants are to be destroyed within 100 m of where infected plants are identified. Particular care needs to be taken to ensure that soil and plant material are not moved into surrounding areas not showing symptoms of the disease. Where possible, destruction should take place in dry conditions to limit mud being spread within the field on boots and protective clothing.

Alternatively or in addition to, the Destruction Zone may be defined as contiguous areas associated with the same management practices as the infected area i.e. the entire trial, paddock or farm if the initial infection was not identified until after management had been undertaken.

2.3.3 Quarantine zone

The Quarantine Zone is defined as the area where voluntary or compulsory restraints are in place for the affected property(ies). These restraints may include restrictions or movement control for removal of plants, people, soil or contaminated equipment from an infected property.

2.3.4 Buffer zone

A Buffer Zone may or may not be required depending on the incident. It is defined as the area in which the pest does not occur but where movement controls or restrictions for removal of plants, people, soil or equipment from this area are still deemed necessary. The Buffer Zone may enclose an infested area (and is therefore part of the Control Area) or may be adjacent to an infested area.

2.3.5 Restricted Area

The Restricted Area is defined as the zone immediately around the infected premises and suspected infected premises. The Restricted Area is established following initial surveys that confirm the presence of the pest. The Restricted Area will be subject to intense surveillance and movement control with movement out of the Restricted Area to be prohibited and movement into the Restricted Area to occur by permit only. Multiple Restricted Areas may be required within a Control Area.

2.3.6 Control Area

The Control Area is defined as all areas affected within the incursion. The Control Area comprises the Restricted Area, all infected premises and all suspected infected premises and will be defined as the minimum area necessary to prevent spread of the pest from the Quarantine Zone. The Control Area will also be used to regulate movement of all susceptible plant species to allow trace back, trace forward and epidemiological studies to be completed.

2.3.7 Reference documents

- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*
- *PLANTPLAN*
- *International Standards for Phytosanitary Measures No 1: Phytosanitary principles for the protection of plants and the application of phytosanitary measures in international trade (2006).*

2.4 Decontamination and farm clean up

2.4.1 Decontamination procedures

2.4.1.1 General guidelines for decontamination and clean up

- Keep traffic out of affected area and minimize it in adjacent areas. Stop irrigating (irrigated farms) affected areas and use bunding to divert overland flood flows around them (both on irrigated and dryland farms).
- Adopt best-practice farm hygiene procedures to retard the spread of the pathogen between fields and adjacent farms.
- Ensure that planting seed production does not take place on affected farms and do not use seed from these farms to plant the next crop as *Fusarium* species can be seed borne.
- Once the disease has been confirmed in an area, growers should only grow resistant varieties. The industry should identify any varieties susceptible to the pathogen and replace them with resistant varieties. (Dosdall *et al*., 2006) reported that most canola varieties in Canada were resistant to *Fo conglutinans* and there was no incidence of fusarium wilt in those varieties during the 2005 season. It should be noted that overseas varieties may not be resistant in Australia if the disease has developed from indigenous *F. oxysporum* populations and this would require varietal assessment for resistance and possibly enhanced breeding programs in Australia.
- Procedures developed to retard the spread of cotton fusarium wilt can be directly applied to retard the spread of fusarium wilt in canola, chickpea and soybean sudden death syndrome in soybeans. They are described in the cotton IDM guidelines (Allen *et al*., updated 2003) and other publications available on the Cotton Catchment Communities website (www.cotton.crc.org.au) under the disease and microbiology section.

2.4.1.2 Decontamination if disease is identified in a small area

- Raking and burning the whole field at this stage is **NOT** an option as this procedure is likely to spread the pathogen.
- Pull out the affected plants, as well as healthy plants 1-2 metres into the area surrounding the patch and burn them in the patch. Particular care must be taken to minimize the transfer of infected soil from the area.

2.4.1.3 Decontamination if disease is identified in large areas

Kill any surviving plants in the area, preferably with herbicides and leave them to die in place. Once the dead plants have broken down, sow a non-host crop such as a cereal or grass pasture to prevent erosion. All equipment used on the site should be thoroughly cleaned down, with products such as a farm degreaser or a 1 % bleach solution and washed down with a pressure cleaner on the affected farm. The clean down procedure should be carried out on hard standing or preferably a designated wash-down area to avoid mud being recollected from the affected site onto the machine. A wash-down pad design and operational procedures are described in the cotton IDM package (Allen *et al*., updated 2003).

2.4.2 General safety precautions

For any chemicals used in the decontamination, follow all safety procedures listed within each MSDS.

2.4.3 Reference documents

- *PLANTPLAN, Appendix 18: Disinfection and decontamination – Plant Health Australia (2006).*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

2.5 Surveillance and tracing

2.5.1 Surveillance

Detection and delimiting surveys are required to delimit the extent of the outbreak, ensuring areas free of the pest retain market access requirements and appropriate quarantine zones are established.

Initial surveillance priorities include the following:

- surveying all host growing properties in the pest quarantine area;
- surveying properties identified in trace forward analysis as being at risk;
- surveying host growing properties that are reliant on trade with interstate or international markets which are sensitive to Fusarium wilt of canola presence;
- surveying commercial nurseries selling at risk host plants;
- surveying other host growing properties and backyards.

2.5.2 Survey regions

Establish survey regions around the surveillance priorities identified above. These regions will be generated based on the zoning requirements (section 2.3) for Fusarium wilt of canola, and prioritised based on their potential likelihood to currently have or receive an incursion of this pest. Surveillance activities within these regions will either: allow for the area to be declared pest free and maintain market access requirements; or establish the impact and spread of the incursion to allow for effective control and containment measures to be carried out.

Steps outlined below form a basis for a survey plan. Although categorised in stages, some stages may be undertaken concurrently based on available skill sets and resources.

Phase 1:

- Identify properties that fall within the buffer zone around the infested premise.
- Complete preliminary surveillance to determine ownership, property details, production dynamics and tracings information (this may be an ongoing action).

Phase 2:

- Preliminary survey of host crops in properties in buffer zone establishing points of pest detection.

Phase 3:

- Surveillance of an intensive nature to support control and containment activities around points of pest detection.

Phase 4:

- Surveillance of contact premises. A contact premise is a property containing susceptible host plants, which are known to have been in direct or indirect contact with an infested premises or infected plants. Contact premises may be determined through tracking movement of materials from the property that may provide a viable pathway for spread of the disease. Pathways to be considered are:
    - Items of equipment and machinery which have been shared between properties including bins, containers, irrigation lines, vehicles and equipment;
    - The producer and retailer of infected material if this is suspected to be the source of the outbreak;
    - Labour and other personnel that have moved from infected, contact and suspect premises to unaffected properties (other growers, tradesmen, visitors, salesmen, crop scouts, harvesters and possibly beekeepers);
    - Movement of plant material and nursery stock from controlled and restricted areas; and

- Storm and rain events and the direction of prevailing winds that result in wind-driven spread of the insect during these weather events.

Phase 5:

- Surveillance of nurseries, backyards and native and weed hosts of Fusarium wilt of canola.

Phase 6:

- Agreed area freedom maintenance, following control and containment.

2.5.3 Post-eradication surveillance

Specific methods to confirm eradication of Fusarium may include:

- Monitoring of sentinel plants
  - Sentinel plants are to be grown in pots using soil removed from the affected site. Plants are to be grown under quarantine containment glasshouse conditions and monitored for symptoms of infection.
- Surveys comprising soil or plant sampling for Fusarium to be undertaken for a minimum of 12 months after eradication has been achieved.

2.5.4 Reference documents

- *PLANTPLAN, Appendix 18: Disinfection and decontamination – Plant Health Australia (2006).*
- *Technical guidelines for the development of pest specific response plans – Dr Peter Merriman and Dr Simon McKirdy (2005), Plant Health Australia.*

3 References

Allen, S.J., Nehl D.B. and Moore, N.Y. 2003. Integrated Disease Management Guidelines for Australian cotton. (www.cotton.crc.org.au), and using the following sections: Industry, Publications, Disease and Microbiology.

Anonymous. 2005a. Canola Industry Biosecurity Plan. Perpetual draft, December 2005. Western Australia Department of Agriculture p 44.

Anonymous. 2005b. Inspection of seed crops to meet overseas import conditions for seed for sowing. Operational Procedures- Plant Biosecurity Plant Health Certification Services Manual. Revision 1 September 2005. Queensland Government, Department of Primary Industries and Fisheries.

Armstrong, G.M. and Armstrong J.K.1981. Formae speciales and races of *Fusarium oxysporum* causing wilt diseases. pp. 391-400 in P.E.Nelson, T.A. Tousson, and R.J. Cook, eds. *Fusarium*: Diseases, Biology and Taxonomy. Pennsylvania State University Press, University Park.
AQIS Phytosanitary Database. (www.aqis.gov.au), Exporting from Australia, PHYTO.

Beckman, C.H. 1987. The nature of wilt diseases of plants. American Phytopathology Society Press.

Bentley, S., Kochman, J.K., Moore, N.Y, Pattermore, J.A., Gulino, L. and. O'Neill W.T. 2000. DNA diagnostics for fusarium wilt of cotton. In: Proceedings of the 10th Australian Cotton Conference, Brisbane, Queensland. Australian Cotton Growers Association, Wee Waa, Australia. 455-461.

Brayford, D. 1992. *FUSARIUM OXYSPORUM* f.sp. *CICERIS*. IMI Descriptions of fungi and bacteria, No. 1113. Mycopathalogia 118: 43-44.

Burgess, L.W. 1981. General Ecology. Fusarium: Diseases, Biology and Taxonomy. (eds. P.E. Nelson, T.A. Toussoun and R.J. Cook) pp. 225-235. The Pennsylvania State University Press, University Park.

Burgess, L.W., Summerell B.A., Bullock S., Gott K.P. and Backhouse, D. 1994. Laboratory Manual for *Fusarium* Research 3rd Edition. University of Sydney, NSW, Australia.

Davis, R.D., Moore, N.Y. and Kochman, J.K. 1996. Characterisation of a population of *Fusarium oxysporum* f.sp. *vasinfectum* causing wilt of cotton in Australia. Australian Journal of Agricultural Research 47: 1143-1156.

Davis, R.M., Colyer, P.D., Rothrock, C.S. and Kochman, J.K. (2006). Fusarium Wilt of Cotton: Population Diversity and Implications for Management. Plant Disease: 90 692- 703.

Dosdall, L., Turkington, K., Kutcher, R., Strelkov, S., Klein-Gebbinck, H., Lange, R., Clayton, G., Hartman, M. and Brandt, S. 2006. Managing insects and diseases in canola. Farmtech 2006 Proceedings, Canada pp 52-62.

Gaetán, S.A. 2005. Occurrence of Fusarium Wilt on Canola Caused by *Fusarium oxysporum* f. sp. *conglutinans* in Argentina. Plant Disease 89:432.

Haware, M.P. 1990. Fusarium wilt and other important diseases of chickpea in the Mediterranean area. Options Méditerranéennes-Série Séminaires - No 9 - 1990: 61-64.

Jarvis, W.R. and Shoemaker, R.A. 1978. Taxonomic status of *Fusarium oxysporum* causing foot and root rot of tomatoes. Phytopathology 68:1679-1680.

Jiménez-Gasco, M.M., Navas-Cortés, J. A. and Jiménez-Díaz, R.M. 2004. The *Fusarium oxysporum* f. sp. *ciceris*/*Cicer arietinum* pathosystem: a case study of the evolution of plant-pathogenic fungi into races and pathotypes. International Microbiology 7:95–104.

Kochman, J.K. 1995. Fusarium wilt in cotton – a new record in Australia. Australasian Plant Pathology 24: 74.

Kochman, J., Swan, L., Moore, N., Bentley, S., O'Neill, W., Mitchell, A., Obst, N., Lehane, J., Gulino, L.L. and Salmond, G. 2002. The Fusarium threat – are we making the progress? Proceedings of the 11th Australian Cotton Conference, Brisbane, 13-15 August 2002, pp 643-652.

Lange, R.M., Harrison, L.M., Hall, M., MacDonald, K. and Kharbanda, P.D. 2001. The first report of fusarium wilt of canola in Alberta, 1999. Abstracts, Alberta Regional Meeting, The Canadian Phytopathological Society 1999 in Canadian Journal of Plant Pathology 23: 185

Lange, R and McLaren, D 2002. Fusarium Wilt – A New Disease of Canola. University of Manitoba website. Viewed 6 March 2007, http://www.umanitoba.ca/afs/agronomists_conf/2002/pdf/lange.pdf

Li, M., Murray, G.M. and Ash, G.J. 2007. New root diseases of canola in Australia. Australasian Plant Disease Notes 2: 93–94

Nelson, P.E., Toussoun, T.A. and Cook, R.J. 1981. Fusarium: Diseases, Biology and Taxonomy. The Pennsylvania State University Press, University Park.

Shaner G.E., Scott, D.H. and Abney, T.S. 1998. Sudden death syndrome in soybeans. Soybean Diseases, Department of Botany and Plant Pathology, Purdue University Cooperative Extension Service. BP-58, 4 pages.

Simmonds, J.H. 1966. Host Index of Plant Diseases in Queensland. Published by the Queensland Department of Primary Industries, Brisbane. pp 14, 15.

Subramanian, C.V. 1970. *Fusarium oxysporum* f.sp. *conglutinans*, C.M.I. Descriptions of Pathogenic Fungi and Bacteria No 213.

Wang, B., Dale, M.L. and Kochman, J.K. (1999) Studies on a pathogenicity assay for screening cotton germplasms for resistance to *Fusarium oxysporum* f.sp. *vasinfectum* in the glasshouse. Australian Journal of Experimental Agriculture 39**:** 967-974.

4 Appendices

Appendix 1. Standard diagnostic protocols

For a range of specifically designed procedures for the emergency response to a pest incursion refer to Plant Health Australia's PLANTPLAN, Appendices 2 and 3.

Appendix 2 Experts, resources and facilities

The following table lists the experts who can be contacted for professional diagnostics and advisory services in the case of an incursion.

Expert	State	Details
Dr Joe Kochman	Qld	Kochman Crop Disease Management Consulting 24 Cawdor Road HIGHFIELDS Qld 4352
Dr Edward Liew	NSW	Plant Pathologist Botanic Gardens Trust Mrs Macquarie's Rd SYDNEY NSW 2000 Ph: (02) 9231 8189 Edward.liew@rbgsyd.nsw.gov.au
Dr David Nehl	NSW	NSW Department of Primary Industries Elizabeth Macarthur Agricultural Institute PMB 8 CAMBDEN NSW 2151 Ph: (02) 4640 6430 David.nehl@dpi.nsw.gov.au
Dr Linda Smith	Qld	Senior Plant Pathologist Qld Department of Primary Industries and Fisheries 80 Meiers Rd INDOOROOPILLY Qld 4068 Ph: (07) 3896 9538 Linda.smith@dpi.qld.gov.au

The following table lists the facilities available for diagnostic services relevant to Fusarium wilt of canola in Australia.

Facility	State	Details
DPI Victoria Knoxfield Centre	Vic	621 Burwood Highway Knoxfield VIC 3684 Ph: (03) 9210 9222 Fax: (03) 9800 3521
DPI Victoria Horsham Centre	Vic	Natimuk Rd Horsham VIC 3400 Ph: (03) 5362 2111 Fax: (03) 5362 2187
DPI New South Wales Elizabeth Macarthur Agricultural Institute	NSW	Woodbridge Road Menangle NSW 2568 PMB 8 Camden NSW 2570 Telephone: (02) 4640 6327 Fax: (02) 4640 6428
DPI New South Wales Tamworth Agricultural Institute	NSW	4 Marsden Park Road Calala NSW 2340 Ph: (02) 6763 1100 Fax: (02) 6763 1222

DPI New South Wales Wagga Wagga Agricultural Institute	NSW	PMB Wagga Wagga NSW 2650 Ph: (02) 6938 1999 Fax: (02) 6938 1809
SARDI - Plant Research Centre, Waite Research Precinct	SA	Hartley Grove Urrbrae 5064 South Australia Ph: (08) 8303 9400 Fax: (08) 8303 9403
Grow Help Australia	QLD	Entomology Building 80 Meiers Road Indooroopilly QLD 4068 Ph: (07) 3896 9668 Fax: (07) 3896 9446
Department of Agriculture and Food, Western Australia (AGWEST) Plant Laboratories	WA	3 Baron-Hay Court South Perth WA 6151 Ph: (08) 9368 3721 Fax: (08) 9474 2658

Appendix 3. Communications strategy

See PLANTPLAN

Appendix 4. Market access impacts

There are no records in the AQIS PHYTO data base that identify any export or phytosanitary requirements or restrictions to any country for the three pathogens causing fusarium wilt of canola, fusarium wilt of chickpea or sudden death syndrome of soybean. Hence, currently there are no apparent reasons why the discovery of any of these pathogens in Australia should cause market access issues for the commodities involved.