Part 1 - Summary Details

CRDC Project Number: **DAN 182**

Project Title: Travel: Chris Anderson – Fusarium 2005 Workshop, Kansas USA (25/6/05- 17/7/05)

Project Commencement Date: 25/5/05 **Project Completion Date:** 17/7/05

Research Program: Capacity & Community

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Part 3 – Final Report Guide

Description and purpose of the travel.

I travelled to the USA to attend the Fusarium 2005 workshop at Kansas State University, and to accompany representatives of the Australian cotton industry on a cotton disease study tour of the USA. The purpose of attending the training course was to improve my technical skills as a plant pathologist, with the specific aim of learning new techniques that will enable me to maintain, identify and differentiate species of *Fusarium*. The study tour aimed to view several diseases of cotton that are not yet present in Australia. First hand experience of these diseases will enhance our ability to detect and manage any future incursions. Both the course and study tour provided opportunities to meet other plant pathologists who work not only on cotton, but on a range of other crops and diseases.

Major findings and outcomes

A. I participated in the Fusarium 2005 workshop at Kansas State University. The course taught the basics required to correctly identify numerous *Fusarium* species using morphological techniques as well as molecular sequencing.
   Traditional techniques included the preparation of single spore cultures, vegetative compatibility grouping, and extensive familiarisation with the morphological characteristic of a range of *Fusaria* grown on specialised media. The importance of using specialised media was stressed. When attempting to identify *Fusaria*, it is important to use a minimal media like carnation leaf agar (CLA). CLA induces rapid production of macroconidia in sporodochia enabling differentiation between sporodochia bearing and non-bearing isolates; an important taxonomic characteristic. Rapid production of sporodochia enables easy visualisation of macroconidia, the shape of which is another important taxonomic characteristic. The production of microconidia in false heads or in chains, from mono and/or polyphialides, is also an important taxonomic characteristic and can be observed on either potato dextrose agar (PDA) or CLA. Colony pigmentation and the production of chlamydospores are also very useful for characterising isolates and should be observed on PDA.
   Morphological techniques are often tedious and can take several weeks to complete. Molecular identification is often much quicker. We were supplied with an unknown fungus and were shown how to extract DNA from the isolate. We then amplified the DNA using PCR, sequenced the DNA, and run the DNA sequence through a sequence database. This provided many people with a quick and easy identification of their unknown isolate. The downfall of this process is that a correct identification relies on 1) the DNA sequence being available within the databank, and 2) the correct name being attributed to the correct sequence. In my case, *Fusarium decemcellulare* was not present in the databank and so morphological identification proved to be the only means of identification.
   I now feel that I have the capability to carry out molecular and traditional techniques that will enable me to more thoroughly fulfil the aims of my research.
B. This travel enabled me to become acquainted with many American cotton pathologists and mycologists. I have maintained regular contact with some of the researchers I met at the workshop, and intend to collaborate with Dr Craig Rothrock, Dr Terry Kirkpatrick, Dr Pat Colyer and Dr Mauricio Ulloa. In particular, Dr David Nehl and I intend to test the suitability of Australian soils for reproduction and survival of reniform nematode through collaboration with Dr Terry Kirkpatrick. Dr

Mauricio Ulloa also suggested that a new PhD student, Ms Rebecca Bennet may benefit from visiting my field trials around Moree and Goondiwindi to learn about the Australian strains of Fusarium. The expertise of these researchers will assist me as I undertake research towards a PhD in the near future.

C. I travelled with Mr David Kelly of Cotton Seed Distributors Ltd. (Goondiwindi, Australia) and Mr William Tyrwhitt of Auscott Ltd. (Warren, Australia) to Arkansas, Texas and California, visiting researchers and field sites to learn about American diseases of cotton. We observed many disease including Black Root Rot, Root Knot Nematode, Reniform Nematode, Texas Root Rot, and two different forms of Fusarium wilt. The study tour has provided me with experience required 1) to quickly identify and act upon any incursions of these diseases in Australia, and 2) to develop a risk assessment for each disease relating to its potential to reach Australia and become a problem of economic importance. The risk assessment is detailed in this report.

Other highlights

The usefulness of Fusarium oxysporum as a species concept.

Dr David M. Geiser from Pennsylvania State University commented that the species *Fusarium oxysporum* is a poor "genetic" species that is probably composed of several groups of genetically distinct fungi. He says, "I would say there are ~45 known identifiable "lineages" (within *Fusarium oxysporum*) that may represent species, or instead, groups of related clones". The implication of this comment is that we need to consider the strains of *Fusarium oxysporum* that we have in Australia as very different to overseas strains of this fungus. For all intents and purposes, we may be dealing with completely different species of fungi compared to those found overseas. Within Australia, Bo Wang and Curt Brubaker's work has identified several distinct "lineages" of *Fusarium oxysporum*. One of these "lineages" includes two groups (VCG 11 and 12) of fungi that are pathogenic on cotton. It is possible that each lineage may be a different species of Fusarium. It is possible that each of the pathogenic groups within lineage A may again be different species altogether. Whilst this is mere supposition on my part, the point to be made is that it is important from a breeding perspective that where we can differentiate strains of *Fusarium oxysporum* on a genetic basis, we should consider these to be distinctly different pathogens that may cause different diseases, otherwise we run the risk of selecting new pathogens to which our current varieties have little or no resistance.

A very useful example of this exists in the USA. Up until recently the American cotton industry believed that it had excellent Fusarium resistance in its Pima varieties. Fusarium wilt (caused by a strain called "race 1") only occurred in conjunction with a nematode infestation that enabled Fusarium to gain access to the plant. Three summers ago, researchers started to notice large welts of dead or missing plants in varieties that were supposedly resistant to Fusarium wilt. *Fusarium oxysporum* was consistently isolated from these dying plants, and no nematodes were found. The problem was that this new *Fusarium oxysporum* (identified as "race 4") was a much stronger pathogen than the *Fusarium oxysporum* upon which Fusarium wilt resistance had been based. More specifically, these were very different diseases. The so called resistance to "race 1" was probably predominantly resistance to the root knot nematode, and not resistance to the fungus.

In Australia, our strains of *Fusarium oxysporum* that attack cotton are more comparable to the American "race 4" in that they do not need nematodes to cause disease. However,

we have a large number of strains that are only mildly pathogenic on cotton. If a nematode was to be introduced from overseas, or a native nematode was to become a pest, it is possible that some of these weaker pathogens may interact synergistically with the nematode to become economically important pathogens. This threat would be easily countered by incorporating nematode resistance into Australian cotton varieties. This would also improve the marketability of Australian varieties currently being sold within the USA.

Fusarium resistance in Australia is selected predominantly on the basis of resistance to VCG 11, which is the stronger and more widespread pathogen. New cotton varieties tend to have higher F-ranks reflecting at least in part improved resistance in the plant against infection by the fungus. The infection process of VCG 11 could be quite different to that of other strains of *Fusarium oxysporum* found in Australian cotton soils. Consequently, by selecting for resistance to VCG 11, we run the risk of selecting for susceptibility to infection by other strains of *Fusarium oxysporum*.

It is important that in our breeding efforts we recognise that Fusarium wilt does not describe a single disease caused by one fungus. Rather, since *Fusarium oxysporum* contains a large number of genetic "lineages", the term Fusarium wilt can describe many different diseases caused by genetically distinct pathogens. This bears direct relevance to our methods of screening for Fusarium resistance. I would suggest that as a preventative measure we should screen varieties against all known pathogenic strains *Fusarium oxysporum* in Australia in an attempt to ensure that as we select for increased resistance to VCG 11, we do not select for increased susceptibility to another strain.

Neozygites fresenii as a natural biocontrol for aphids in American cotton fields.

We visited Dr Don Steinkraus at the University of Arkansas to learn about his work with the entomopathogenic fungus *Neozygites fresenii* (Figure A).

Figure A. ***Neozygites fresenii*** **"sticky" spores on an aphid leg.**

The fungus is an extremely efficacious pathogen of the cotton aphid in the southern states of the USA where relative humidity can be between 90 and 100% within and above the cotton canopy. As a result, epizootics build up from mid June to July within the aphid populations and move throughout the cotton growing areas, killing off the aphids, and therefore negating the need for an aphicide application. An infected aphid will die at around 10pm. At anywhere between 1 and 6am, up to 3000 spores of the fungus are forcibly ejected from the dead aphid. These spores are called ballistospores because of the ballistic nature of their ejection. These spores land and germinate, producing sticky secondary spores which adhere to aphids as they pass by. Throughout the day aphids become infected by the sticky spores which penetrate the aphid and produce initials that eventually become primary ballistospores which are shot out of the aphid when it dies, thus completing the asexual lifecycle of this fungus. This process can kill an aphid in three days, whereas it may take up to 12 days for a parasitic wasp larva to kill an aphid. It would be prudent to investigate the potential of this fungus as a biocontrol for aphids in Australia, particularly late in the season when humidity in the canopy is high.

*Macrophomina phaseolina cotton boll rot in Texas***.**

Dr Gary N. Odvody of Texas A&M University recently observed severe charcoal boll rot in cotton fields in south Texas. Lint was badly contaminated by small black sclerotia (see Fig. B) belonging to the fungus *Macrophomina phaseolina* which is usually associated with charcoal rot of cotton, a disease that affects the stem and root system. Dr Odvody commented that the outbreak was localised and probably caused by a heavy rainfall event

that splashed spores of the fungus from the soil up onto the bolls. He feels that this disease is unlikely to ever become common.

Figure B. Lint contaminated with sclerotia of *M. phaseolina*.

Itinerary

NAME	POSITION	LOCATION	DAYS	PURPOSE
Dr Craig Rothrock	Professor, Plant Pathology	University of Arkansas, Fayetteville	5	To visit Dr Rothrock's lab and field experiments in Arkansas. We observed root knot nematode, black root rot, and an interaction between these two pathogens. We also observed reniform nematode. Dr Rothrock explained to us a very useful pre-emptive fungicide matrix that can be used by growers to predict seedling disease pressure and therefore make important management decisions regarding the application of fungicides.
Dr James Stewart	Professor, Altheimer Chair for Cotton Research and Development	University of Arkansas, Fayetteville	1	To discuss the progress of work begun by Dr Stewart on the engineering of a frog gene into cotton that has potential to increase resistance to a number of diseases.
Dr Don Steinkraus	Professor, Biological Control	University of Arkansas, Fayetteville	1	To discuss the use of *Neozygites fresenii* for the biocontrol of aphids un US cotton production systems.
Dr Tim Kring	Professor, Entomology	University of Arkansas, Fayetteville	1	To hear about Dr Kring's work with insecticides for controlling aphids.

Dr Terry Kirkpatrick	Professor, Plant Pathology	University of Arkansas, Southern Arkansas	2	Dr Kirkpatrick accompanied us on our visits to Dr Rothrock's field trial sites.
Dr Pat Colyer	Professor, Plant Pathology	Louisiana State University, Southern Arkansas	2	Dr Colyer accompanied us on our visits to Dr Rothrock's field trial sites.
Dr Arin Robinson	Zoologist	United States Department of Agriculture, College Station, Texas	1	To learn about the work being carried out by USDA on the reniform nematode in cotton.
Dr Charlie Howell	Research Plant Pathologist	United States Department of Agriculture, College Station, Texas	1	To hear about Dr Howell's work on seed vigor.
Dr Jinggao Liu	Research Molecular Biologist	United States Department of Agriculture, College Station, Texas	1	To hear about Dr Liu's attempts to clone the genes responsible for the production of the two enantiomers of gossypol in cotton.
Dr Alois Bell	Research Plant Pathologist	United States Department of Agriculture, College Station, Texas	1	Dr Bell was involved in discussions about the work of Drs Robinson, Liu and Howell.
Dr Tom Isakeit	Associate Professor Plant Pathology	Texas A&M University, College Station, Texas	1	To visit Dr Isakeit's lab and field sites and observe Texas root rot.
Mr Phillip Denton	Laboratory Manager	JG Boswells, Corcoran, California	1	To see how the Boswell Corporation runs its own laboratory testing facility to monitor pathogen levels on its farms.
Dr Stephen Oakley	Vice President R&D	California Planting Cotton Seed Distributors, Shafter, California	1	To learn about the seed production system at CPCSD.
Dr Mauricio Ulloa	Research Geneticist	United States Department of Agriculture, Shafter, California	2	To visit Dr Ulloa's field trial sites where race 4 Fusarium is present in Pima varieties.
Dr Bob Hutmacher	Associate Specialist	United States Department of Agriculture, Shafter, California	2	Dr Hutmacher accompanied us with Dr Ulloa.

Are there any potential areas worth following up as a result of the travel?

1) As mentioned above, it would be pertinent to screen new cotton varieties against all known pathogenic strains of *Fusarium oxysporum* present in Australian cotton production areas so as to detect increases in virulence among lesser known strains that could present as major threats to the industry if left unchecked.

2) It would also be pertinent to explore the possibility of breeding nematode resistance into Australian cotton varieties. This would be a pre-emptive measure to provide a

management tool for possible incursions of reniform nematode. At present our varieties do not show resistance toward reniform nematode and as a result, the industry has no means of dealing with potential incursions of this economically important pathogen of American cotton production systems.
3) Moreover, the industry would be wise to invest in research to assess the impact of native nematodes on Fusarium wilt and Verticillium wilt, especially on the less virulent strains of these fungi. There is potential for a synergistic interaction similar to that between "race 1" Fusarium and Root Knot Nematode in the USA to develop between our weaker strains of Fusarium and plant parasitic nematodes.
4) Australian varieties of cotton and Pima should be screened for resistance to race 4 Fusarium.
5) The fungus *Neozygites fresenii* could be investigated as a potential biocontrol for aphids in Australian cotton production systems.

Relevance or possible impact on the Australian Cotton Industry?

Risk Assessment: What is the potential for American cotton diseases to spread to Australia and becomes problems of economic significance?

Root knot nematode
Pathogen(s): *Meloidogyne incognita*. (nematode), may interact with the fungi *Theilaviopsis basicola* and *Fusarium oxysporum*. May also interact with various seedling pathogens including *Rhizoctonia spp*. and *Pythium spp*.
Distribution: Throughout most of the US cotton belt.
Ideal soil conditions: Sandy
Ideal climatic conditions: warm and moist.
Disease symptoms: Stunted plants. Galling and deformation of tap and lateral roots at site of nematode infestation (see Fig. C). **With *T. basicola*:** as for root knot nematode with typical root blackening caused by colonisation of the root cortex (see Fig. D). **With *F. oxysporum*:** as for root knot nematode, with vascular discoloration (see Fig. E), wilting, chlorosis and necrosis of leaves, leading to plant death. Often associated with heavy stand loss. **With seedling pathogens:** increased stand loss and post emergent damping off.
Disease aetiology: The nematode is present in the soil and survives well on alternate hosts including corn, susceptible soybean and weeds. Eggs attached to cotton root or tissues of other susceptible hosts are the primary means of over-wintering. Symptoms are usually most severe following a susceptible rotation crop like corn or soybean, because nematode numbers in the soil are high. Nematodes infest the roots of seedlings causing galling, deformation and stunting. This may be associated with disruption of vascular tissue. **With *T. basicola*:** The black root rot fungus and root knot nematode are present in soil. The fungus can survive between crops by producing resting spores. When cotton reaches seedling stage, the black root rot pathogen infects the cortical tissue of the plant in much the same way that it does in Australian cotton production systems. Usually, the plant would grow out of this infection by sloughing off the outer cortical layers. However, disease pressure from nematode infestation prevents the cotton from growing out of the black root rot infection thus stunting the crop for a much longer period than would otherwise occur in the absence of the nematode. Moreover, nematodes can facilitate infection of the vascular tissue of cotton plants by *T. basicola*. This may result in some seedling death. **With *F. oxysporum*:** the fungus survives well in soil as a saprophyte on organic matter. However, this strain of *F. oxysporum*, known as "race 1" is only a weak

pathogen and cannot infect cotton in the absence of the nematode. When root knot nematodes are present in the soil, even at low numbers, and not necessarily infesting the roots, the fungus is able to penetrate roots and move into the vascular tissue causing wilting, foliar symptoms and eventual death of the host plant. **With seedling pathogens:** the nematode may facilitate infection of seedlings by these fungi.

Control Measures: Control of the nematode is the primary means of controlling both interactions. Current trials run by Dr Craig Rothrock indicate the chemical 1-3 dichloropropene, marketed as Telone II provides excellent suppression of symptoms (see Figure F). The fumigant is applied to the soil 1-2 weeks before cotton is sown. The nematode can also be controlled by rotating with Rice as flooding helps to reduce nematode populations, and Rice is a non-host. Other effective rotations include peanuts, resistant soybeans and sorghum.

Risk of spread to Australia: Root knot nematode (*M. incognita*) is already present in Australia. However, this nematode is not recorded from cotton. This could be because the heavy clay soils in which Australian cotton is grown are not suitable for survival of the nematode, or it may be because the strains of *M. incognita* that are present in Australian soils lack virulence towards cotton. Strains of this nematode that are known to be virulent towards cotton could spread from America to Australia, probably in soil on dirty machinery. **With *T. basicola*:** this fungal pathogen is present in Australia. **With *F. oxysporum*:** "race 1" *F. oxysporum* is not present in Australian cotton soils and could easily be imported in soil and trash on dirty farm machinery. **With seedling pathogens:** these seedling pathogens are already present in Australia.

Potential to damage Australian cotton production: As the nematode is favoured by sandy soils, and Australian cotton is grown on relatively heavy clay soils, Root Knot Nematode is unlikely to establish in Australian soils. Thus, interactions with *T. basicola*, "race 1" *F. oxysporum* and seedling pathogens are unlikely to establish. Therefore this pathogen should not be considered a threat to Australian cotton production.

Action required: None. It is possible that some Australian native nematodes may interact with *T. basicola*, *F. oxysporum*, and seedling pathogens in Australian soils and so some research on this topic would be beneficial.

Figure C. Galling on lateral roots caused by root knot nematode.

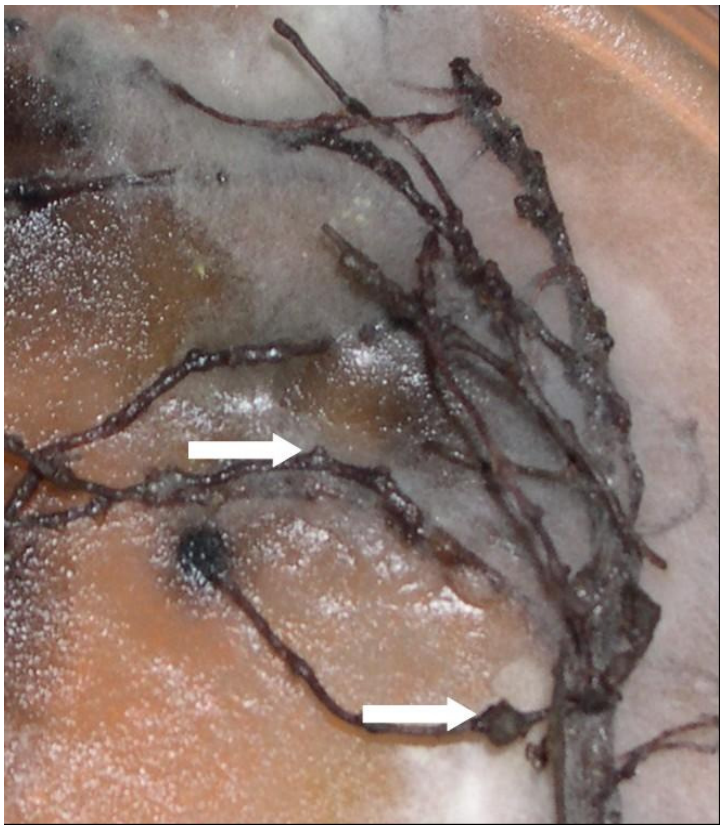

Figure D. Cotton roots with blackening attributable to infection by *Thielaviopsis basicola* and severe galling (arrows) attributable to root knot nematode.

Figure E. Root galling and vascular discoloration indicating an interaction between root knot nematode and "race 1" *F. oxysporum*.

Figure F. A: cotton plants displaying stunting attributable to the black root rot and root knot nematode interaction. B: neighbouring plants treated with Telone, without stunting.

Reniform nematode

Pathogen(s): *Rotylenchulus reniformis* (see Fig. G)

Distribution: Predominantly in the eastern regions of the cotton belt; North Carolina to Texas. Not recorded in California.

Ideal soil conditions: Not favoured by sandy soils to the extent that root knot nematode is. Occurs across a broad range of soil types.

Ideal climatic conditions: warm and moist.

Disease symptoms: Stunting. *R. reniformis* tends to be uniformly distributed throughout a field thus making it difficult to differentiate between stunted plants and healthy plants. Yield loss is a good indicator that there may be a nematode problem. Detection of the nematode must be done by microscopic examination of soil samples; although in sandy soils, female nematodes may be seen protruding from the root with the naked eye (see Fig. H).

Disease aetiology: Female nematodes move into seedlings shortly after seed germination to feed. Soil populations may build up during the season to extremely high numbers. As numbers increase diseases severity increases. This nematode may also increase the severity of Fusarium wilt. The nematode has been found in soil under cotton at depths of up to 7-8 feet. It is easily distributed with the flow of irrigation and flood water. It has been suggested that when reniform and root knot nematode occur in the same field, root

knot nematode numbers decline possibly because cotton is a more favourable host for reniform nematode.

Control measures: Apart from the application of nematicides before (Telone II) and at planting, crop rotation is the best means of controlling this nematode. Non-susceptible crops include corn, sorghum, rice, peanut and resistant soybeans (T. Kirkpatrick).

Risk of spread to Australia: This nematode is easily spread in soil and thus could be imported on dirty machinery.

Potential to damage Australian cotton production: As this nematode survives well in numerous soil types, *R. reniformis* could pose a serious threat to the Australian cotton production if imported. *R. parvus* has been found in cotton fields in southern Queensland, but is not considered to be a serious pest.

Action required: This nematode has potential to cause significant yield losses. Thus, we should insist on strict quarantine requirements for imported used farm machinery, and should monitor our own soils for the presence of this nematode to ensure early detection of any future incursion.

Figure G. *Rotylenchulus reniformis* adults.

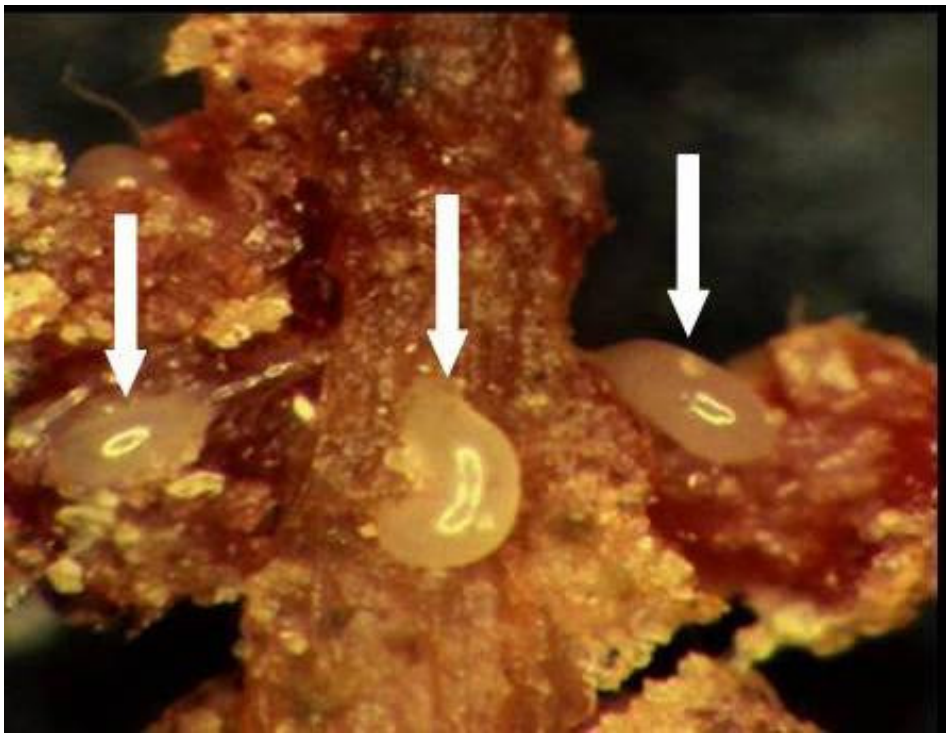

Figure H. Female reniform nematodes protruding from a cotton root. These would appear as sandy blobs when the plant is first removed from the soil, but become apparent after rinsing in water. (Photo by Charles Overstreet)

Texas (or Cotton) root rot

Pathogen(s): *Phymatotricopsis omnivorum*

Distribution: Native to the southern coast of USA.

Ideal soil conditions: High pH vertisols, with an optimal pH of between 7.2 and 8.0 (Percy, 1983), calcareous (Calcium carbonate >1%), montmorillonites (a geological term for a type of clay that is prone to cracking and swelling and which rapidly looses its structure when submerged in water). The fungus is favoured by low sodicity and will not survive in soils with > 2-3% meq/100g soil (Percy, 1983). High rainfall and soil waterlogging favours the pathogen as it produces Carbon dioxide, increasing levels of which induce the formation of sclerotia (survival spores) which are the pathogens primary means of reproduction (Lyda and Kenerley, 1993).

Ideal climatic conditions: warm temperatures and high rainfall. During the growing season, daytime temperatures of above 35C are favourable. The fungus will survive well where the air temperature does not fall below -23C and where the annual average temperature is 15.6C or higher (Lyda and Kenerley, 1993). Soil temperatures of between 15 and 35C will favour the production of sclerotia, with greatest production occurring at 28C (Lyda and Kenerley, 1993).

Disease symptoms: Disease onset is sudden. Plant death usually emanates from a single focus of inoculum in the field and moves out from that point as the fungus grows through the soil by extending hyphal strands between plants. Plants wilt in a very similar manner to Fusarium wilt and then die, turning black and crisp (see Fig. I). Wilting plants may be removed from the ground to find white and yellow hyphal strands on the roots and basal

areas of the stem (see Figure J). In order to diagnose this disease, these strands should be examined microscopically for the presence of the distinctive acicular cruciform hyphae (see Fig. K). Stem girdling (see Fig. L) is also indicative of infection by this fungus. Splitting the stem of an infected plant will often reveal red discolouration from within the roots to about half way up the stem. This could easily be mistaken for sudden wilt.
Disease aetiology: The fungus over-winters by producing copious sclerotia in pockets at various depths in the soil. When temperatures rise, the sclerotia germinate, producing hyphae that infect plants as they move through the soil. When a hypha reaches a root, it is generally thought to colonise the root system, causing the root to rot and die, thus killing the plant. *P. omnivorum* may also invade the vascular tissue causing the plant to wilt and die, thus explaining the vascular discolouration that is often associated with this disease. The fungus moves through a field by elongating hyphal strands through the soil from plant to plant. Occasionally, after rain or periods of prolonged humidity and low radiation, hyphal mats form on the soil surface. These mats produce large quantities of unviable asexual spores. The purpose of this phenomenon is not understood.
Control measures: There is no effective control for this fungus. Dr Tom Isakeit of Texas A&M University is currently trialling a variety of fungicides applied to plants as a stem-directed spray. Results are not yet available.
Risk of spread to Australia: This fungus could be transported to Australia on dirty earth moving or agricultural machinery.
Potential to damage Australian cotton production: The average annual temperature across the cotton growing regions of Australia is > 15.6C. Cotton is usually grown in heavy cracking, mid-high pH vertisols, that can be calcareous. However, the majority of irrigated soils are sodic with an exchangeable cation potential (ESP) of > 6%. This equilibrates to a sodicity of approximately 2.7 meq/100g soil, making most Australian soils unsuitable for this pathogen (Percy, 1983).
Action required: Texas root rot is unlikely to become a widespread problem in Australian cotton production as our sodic soils are not conducive to survival of the pathogen. In those instances where sodicity is low, there is potential for this fungus to establish and cause yield loss. Therefore, pathologists should remain observant and examine plants for cruciform hyphae where other symptoms may indicate an outbreak of Texas root rot.
References: Lyda, SD and Kenerley CM (1993) Biology and Ecology of *Phymatotrichum omnivorum* in *Biology of Sclerotial-forming fungi*, The Texas Agricultural Experiment Station, College Station, Texas.
Percy, RG (1983) Potential range of *Phymatotrichum omnivorum* as determined by edaphic factors. *Plant Disease* **67 (9)**: 981-983.

Figure I. Dead plants (foreground) killed by Texas root rot.

Figure J. Typical white mycelia and yellow strands (A) at the base of a cotton stem, and stem girdling (B).

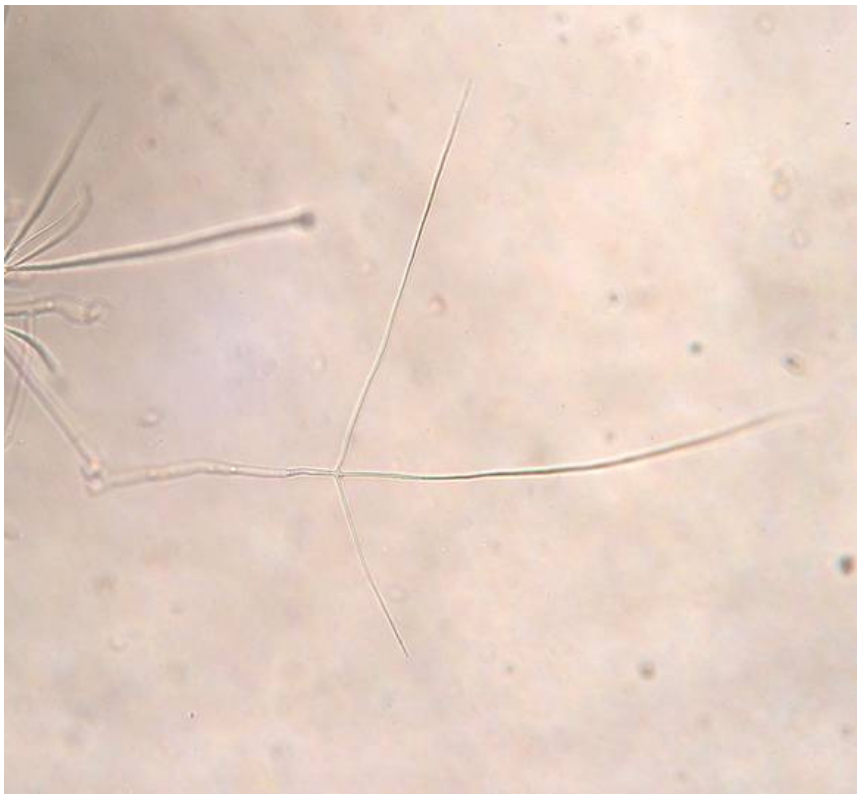

Figure K. An acicular cruciform hypha. This is a diagnostic feature of *Phymatotricopsis omnivorum*.

Figure L. Girdling of the stem at soil level and below caused by infection by *Phymatotricopsis omnivorum*.

Fusarium wilt (race 4)

Pathogen(s): *Fusarium oxysporum* (Race 4)
Distribution: San Joaquin Valley, California, USA
Ideal soil conditions: Various soil types including silty clays.
Ideal climatic conditions: cool wet conditions.
Disease symptoms: similar to Fusarium wilt in Australia. Infected plants are often severely stunted. The vascular tissue becomes develops a brown discolouration. Discolouration will extend from the roots to the growing tip as the fungus moves up the plant in the vascular tissue. Leaves may turn chlorotic and necrotic and the plant wilts and dies from the top down. This particular strain of *F. oxysporum* is especially virulent towards Pima varieties and does not require a nematode interaction to infect plants. In severe fields, heavy stand loss may occur when susceptible varieties of Pima are planted (see Fig. M). This strain of *F. oxysporum* also infects upland cotton, but the disease is generally less severe than that seen in Pima. For instance, vascular discolouration in *G. hirsutum* does not extend the entire length of the stem as it does in *G. barbadense*.
Disease aetiology: the fungus persists on organic matter in the soil. The fungus can infect plants at all growth stages. As in Australian Fusarium wilt, initial infection occurs in the roots, and progresses up the plant through the vascular tissue causing the plant to block up its own vascular system, wilt and die.
Control measures: This disease appears to be worse when cotton is sown during cool wet conditions. Therefore, avoiding these conditions by sowing later is management option. It is thought that this pathogen has been spread from farm to farm in mud on tomato harvesters, thus farm hygiene is an important tool in preventing the further spread of this pathogen. The most effective tool in the management of this disease is the growing of resistant Pima varieties. The most resistant variety is Phytogen 800, which can be seen below alongside susceptible varieties (Fig. M).
Risk of spread to Australia: This fungus could be easily spread on dirty farm and earth moving machinery.
Potential to damage Australian cotton production: This strain of Fusarium is probably not as virulent towards *G. hirsutum* as the strains already present in Australia. Therefore, with the adoption of resistant *G. hirsutum* varieties such as Sicot F1 and Sicala 45, and the next generation of these varieties, it is unlikely that this fungus will pose a major threat to the Australian cotton industry. However, this remains to be thoroughly tested. This fungus could threaten the sustainability of Pima production in Australia and should be regarded as an important quarantine risk for this reason.
Action required: American researchers should test the virulence of this strain of Fusarium towards Australia Pima and upland cotton varieties.

Figure M. Phytogen 800 (A) and susceptible (B) lines of Pima in a field with race 4 *F. oxysporum*.

Sharing the knowledge

Several interviews with researchers were recorded by Mr David Kelly. These will be available on the CSD website for researches, cotton growers and consultants to download and view. This report to CRDC and NSW DPI will be the main means of communication of results of this travel. I would be more than happy to present the results of this travel, in particular the risk assessment, as a presentation to interested parties if asked to do so.