

FINAL REPORT

Determining the cause, extent, impact and potential control measures for an unidentified disorder in sunflower crops in Central Qld

DAQ00097

Project Details

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- **Supervisor:** Richard Routley
- **Organisation:** Department of Agriculture, Fisheries & Forestry
PO Box 102 Toowoomba QLD 4350
- **Contact Name:** Richard Routley
Phone: 07 4688 1121
Email: richard.routley@daff.qld.gov.au

Summary

An unknown plant disorder has devastated sunflower crops across Central Queensland (CQ) since 2004 and has resulted in major reductions in sunflower plantings. This project identified the causal agent of the disorder as tobacco streak virus (TSV), a member of the *Ilavirus* genus. The virus is transmitted by thrips that carry infected pollen grains from infected weed and crop hosts. *Partheniumhysterophorus*, a major and widespread weed of pastures in CQ, is the major alternative host of the virus. Several other common weeds and field crops are also known to be hosts. TSV caused significant yield losses in CQ mungbean crops in 2006-07. Preliminary recommendations to minimise the impact of TSV have been developed.

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Conclusions

Key conclusions emerging during this project are:

1. The 'Central Queensland sunflower disorder' is caused by TSV, a member of the *Ilavirus* genus.
2. The virus is spread when thrips transmit virus-infected pollen from crop or non-crop host plants to a susceptible crop.
3. *Parthenium hysterophorus*, a common weed in CQ, is a symptomless host of the virus and is considered to be the major source of infection.
4. Many other common weed species are known to be hosts. However, the presence of TSV in other weeds has not been demonstrated in CQ.
5. The following hosts were susceptible to TSV in glasshouse transmission tests and produced severe necrotic symptoms: chickpeas, faba beans, peanuts, mungbeans, azuki beans, French beans, soybeans and sunflowers. Cotton was also infected, but without necrosis.
6. TSV caused major yield losses in many mungbean crops in the Emerald region in 2006-07. TSV has been observed in commercial chickpea crops in a small number of cases but did not cause economic damage.
7. Insecticidal seed treatments were not shown to have an effect on TSV incidence or impact.
8. The severity of infection (% plants infected) of TSV in susceptible crops varies widely (0 to >50%) and appears to be related to vector populations, supply of infected pollen and proximity to sources of infection.
9. The impact of the disorder on individual infected plants ranges between complete plant death and minor visual symptoms and is related to the timing of infection.
10. There is some evidence of differential varietal tolerance of TSV among commercial sunflower cultivars.
11. Control measures will focus on varietal tolerance and control or avoidance of vectors and alternative hosts.

Recommendations

Preliminary recommendations to minimise the impact of TSV in susceptible crops have been developed based on current knowledge and are included in extension brochures. In summary, these recommendations are based on:

1. control of alternative hosts, in particular, parthenium weed
2. avoidance of planting susceptible crops close to areas infested with parthenium weed
3. providing a buffer zone between susceptible crops and parthenium infested areas
4. monitoring thrip populations and consideration of insecticidal control if numbers are high (no thresholds established)
5. use of planting seed from non-infested crops (to avoid any possibility of seed transmission).

Recommendations regarding future research, development and extension (RD&E) needs are provided in the following section of this report.

Outcomes

This project successfully identified the cause of the disorder which has devastated the CQ sunflower industry since 2004. The cause of the disorder was previously unknown. The causal agent (TSV) has also resulted in significant yield losses in mungbean crops in the Emerald district in 2006-07.

Both sunflower and mungbean are minor but significant crops in CQ grain farming systems. They both have a niche and contribute to cropping options available to grain producers. The annual area of sunflowers produced in CQ typically fluctuated between 20,000 and 50,000 hectares (ha) prior to the occurrence of the disorder, while the mungbean area ranged between 5000 and 20,000 ha per annum depending on seasonal conditions. There are significant areas of irrigated mungbeans produced in the Emerald and surrounding irrigation areas.

TSV has caused yield losses of up to 30% in both sunflower and mungbean crops, but more significantly, the occurrence of TSV has resulted in graingrowers losing confidence in growing them. This resulted in a reduction in sunflower areas to between 2000 and 4000ha in each of the past two summer seasons. If similar trends occur with mungbean plantings, the direct economic impact of TSV could approach \$15 million per annum in lost production in CQ.

There is also the potential for TSV to have an impact in other production areas where suitable vectors and hosts are present.

The findings of this project are an important first step in developing management and genetic solutions to TSV in the grain cropping industries, and restoring confidence in sunflower and mungbean production in CQ.

Achievement/Benefit

Background

In recent years, a large proportion of CQ sunflower crops have been affected by an unknown plant disorder that has significantly reduced yields and grower returns. As a result, many sunflower producers were reluctant to continue sunflower production until the cause of the disorder and control measures were identified. This uncertainty has had a major impact on the area planted and production of sunflowers in CQ. In the late 1990s and early 2000s, CQ was a major sunflower production area with annual plantings of around 30,000 to 50,000ha. In contrast, plantings in 2005-06 and 2006-07 were approx. 2000 to 4000ha each year. Although seasonal conditions and relatively low sunflower seed prices played a part, concern regarding the disorder was a major factor in this reduction in production.

Preliminary attempts to identify the cause of the disorder had been inconclusive. However, herbicide residuals or drift, nutrient disorders (possibly boron deficiency), an unknown pathogen, adverse environmental conditions or any combination of these factors had been suggested.

This project aimed to identify the causal agent or agents of the disorder and to develop preliminary recommendations for control or avoidance. The project was approved in late 2005 with a commencement date of 1 January 2006. A full-time, experienced senior technical officer (Mr John Ladewig) was appointed to the project and his activities were overseen by a project management group consisting of scientists from a range of relevant disciplines, together with agribusiness and industry representatives. The project was supervised by an experienced agronomist (Mr Richard Routley), in recognition of the multidisciplinary approach taken.

Initial methodology

The initial project methodology included glasshouse and field experiments designed to confirm (or otherwise) the general view that the disorder was related to herbicide effects, as well as intensive monitoring of any incidence of the disorder in commercial crops. Three glasshouse experiments and one field experiment were conducted in early 2006 to test the hypothesis that the disorder was due to herbicides (either residual effects of soil applied herbicides or drift from herbicides applied in adjacent paddocks). Herbicide treatments included in these experiments were glyphosate[#], 2,4-D[#], metsulfuron-methyl[#], atrazine[#], imazethapyr[#] and fluroxypyr[#]. No herbicide treatments included in these trials were successful in reproducing the symptoms of the disorder and as a result, the herbicide hypothesis was effectively ruled out.

Details of all sunflower producers and sunflower crops sown in the 2005-06 and 2006-07 seasons were collated by project staff, based on seed sales and local knowledge. All known crops were inspected during the growing season to detect the presence of the disorder. The number and area of crops sown were low in both seasons (< 10 producers and approx. 3000 to 4000ha each season) for the reasons referred to previously.

The disorder was detected in the majority of crops grown in the late summer and autumn of 2006 and its development was monitored closely. A field walk in infected commercial crops was conducted on 1 June 2006 and attended by around 15 invited researchers and advisers from a range of disciplines. Following this field walk, it was agreed that the symptoms were consistent with those that would be expected from an insect-borne pathogen, and probably viral. This conclusion led to a renewed focus on identifying a pathogen. Around the same time, a review of the literature uncovered reports of similar symptoms in sunflower crops in India. These were subsequently shown to be

caused by TSV. Department of Primary Industries and Fisheries (DPI&F) virologists were engaged to search for the presence of TSV, or other viruses, in infected plants.

Confirmation of TSV as the causal agent

A sunflower sample was submitted with some green leaf tissue and showing necrotic oak leaf patterns suggestive of virus infection. This sample contained isometric particles when examined in the electron microscope. Similar particles were observed in subsequent samples. The morphology of these particles was typical of the genus *Ilarvirus*, of which tobacco streak virus is a member. Typical host reactions for TSV were obtained when the virus from sunflower was inoculated onto test plants. TSV-specific polymerase chain reaction (PCR) primers were designed and gave a positive reaction with samples of sunflower necrosis disease. The PCR products were cloned and sequenced, and the virus confirmed as TSV by comparison with sequences on the GenBank database. Further confirmation was obtained by positive reactions of sunflower samples in a TSV-specific ELISA test. TSV was inoculated onto tobacco, then from tobacco, re-inoculated onto sunflower, where it produced typical sunflower necrosis symptoms. This confirmed not only the presence of TSV, but the fact that it caused the disease.

Subsequent research focused on identifying alternative hosts and transmission vectors associated with TSV. In addition, anecdotal observations in the 2006 sunflower crop suggested that crops grown from seed treated with insecticides had a reduced level of infection. A field trial was conducted in 2007 to confirm this observation.

Crop host range

The following hosts were susceptible to TSV in glasshouse transmission tests and produced severe necrotic symptoms:

- chickpeas
- faba beans
- peanuts
- mungbeans
- azuki beans
- French beans
- soybeans
- sunflowers.

Cotton was also infected, but without necrosis.

In CQ, field infections of TSV were recorded in chickpeas, mungbeans and sunflowers, and typical disease symptoms were reported in peanuts, although TSV was not confirmed as the causal agent in this case. In the 2006-07 mungbean crop, many paddocks in the Emerald district were infected with TSV with the impact on yield varying from mild to severe. Field infection of chickpeas has been observed in only two cases.

Sunflowers and mungbeans have been tested for seed transmission (which is known to occur in some species) but none has been observed.

Crop varietal reactions

No clear differences were observed in the reaction of commercial and breeding lines of sunflowers to TSV in glasshouse screening tests. However, in a commercial planting situation at Clermont, with high natural disease pressure, cv. Aussie Gold was decimated with 100% infection and severe necrotic symptoms, whereas an adjacent planting of Hyleic 41 had a disease incidence of only about 40%.

Weed hosts

P. hysterophorus was demonstrated to be a symptomless, natural host of TSV in CQ. Many severe disease outbreaks in sunflowers and mungbeans were associated with the presence of areas of flowering parthenium in adjacent pasture paddocks. Initial results indicate that the virus is seed transmitted in this host. Many other common weeds have been reported in the literature as being hosts of TSV, although infection of other weed species has not been confirmed in the field in CQ.

P. hysterophorus is a widespread weed of pastures and roadsides in CQ and can occur, and flower, at any time of the year in response to rainfall. It is likely that the association between TSV and parthenium species (which has also been reported overseas) is the major reason that TSV has caused problems in sunflower and mungbean crops in CQ and not other production areas.

Vectors

TSV is known to be transmitted by thrips carrying virus-infected pollen grains. To date, five species have been shown to be vectors. Thrips species recovered from sunflowers, parthenium and other weeds from CQ include *Thrips tabaci*, *Frankliniella schultzei*, *Microcephalothrips abdominalis*, *Tenothrips frici*, *Haplothrips bituberculatus* and *Haplothrips froggatti*, the first three recorded in literature as vectors.

Seed treatment trials

Field observations during the 2006 sunflower crop season suggested that seed insecticide treatments may have a mitigating effect on the incidence and severity of the disorder. Two field experiments were established in early 2007 to test the impact of three seed applied insecticides (Gaucho[®], Cruiser[®], Cosmos[®]) and one plant defence activator (Bion[®]) on the incidence and impact of the disorder. No significant differences in infection rates or severity of infection were recorded in these trials.

Impact on crop yield

The impact of TSV on crop yield depends on the proportion of plants infected and the impact of infection on individual plant yields. Both factors can vary widely. The proportion of plants infected with TSV in crops monitored in this project varied between 0 and 50% and appeared to be related to vector populations, supply of infected pollen and proximity to sources of infection.

The impact of the disorder on individual infected plants ranges between complete plant death and minor visual symptoms and is related to the timing of infection. Infections that occur early in the growing season have greater impacts. Timing of infection appears to be related to fluctuations in vector populations and the abundance of flowering (pollen-producing) weed hosts.

Benefits

The major benefit from this project is the identification of the causal agent of the Central Queensland sunflower disorder, and that there is now a relatively good understanding of sources of infection and vectors involved in its transmission. The susceptibility and potential impact of TSV on other crops is also understood. Preliminary recommendations regarding mitigating the spread and impact of the disorder have been developed and widely disseminated throughout the industry in CQ. Graingrowers in this region now have a better understanding of the risks associated with the disorder and can make more informed decisions regarding if, how, when and where they grow susceptible crops.

Other Research

While this project has been successful in identifying the cause of the sunflower disorder and developing preliminary recommendations to minimise impacts, further research and development is required to develop long-term strategies to minimise the impact of TSV on the grains industry in CQ and elsewhere.

These recommendations have been incorporated in the project specification for the 'Epidemiology and management of tobacco streak virus in sunflower and pulse crops of the Northern Region' project which has been submitted to GRDC.

Recommended research activities include:

1. Search for field weed hosts of TSV which could act as virus reservoirs and examine potential seed transmission of TSV in these species. This will allow identification of points in the infection cycle that can be targeted for control.
2. Survey for TSV on sunflowers, pulses, weeds and other crops that may act as sources of infection.

3. Examine sunflower germplasm and breeding lines to search for tolerance and resistance to TSV.
4. Determine which thrips species transmit TSV.
5. Use of the above information to examine management systems to reduce the impact of the virus.

Additional Information

Murray Sharman, Denis Persley, John Ladewig, John Thomas, Gary Kong and Richard Routley (2006). Tobacco streak virus causes sunflower disease in Queensland Proceedings of the Seventh Australasian Plant Virology Workshop, Perth, WA.