Final report

Project title:

Diagnosis and management of dieback in persimmon trees

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Public summary

Persimmon dieback is an increasing problem affecting orchards in Australia and overseas. The disease is commonly observed in Australian-grown trees 6 to 36 months after transplanting, causing tree decline and, in severe cases, leads to tree mortality, reducing tree health, productivity, and grower returns. In Australia, persimmon dieback was observed across multiple regions, but its causes remain poorly understood. This knowledge gap made it difficult for growers to diagnose the problem and to apply effective management strategies. This project aimed to understand the causal agents of persimmon dieback in Australia, and to identify available options for potential management of the disease.

Field surveys were conducted across major persimmon-growing regions, with samples collected from cultivars exhibiting dieback and other symptoms, such as leaf, bud, and fruit damage. Fungi associated with these symptoms were isolated and identified using both traditional morpho-cultural characterisation and DNA-based techniques. The study also assessed the pathogenicity of the different fungi, their potential spread within persimmon trees, how environmental conditions affect their growth, and whether they can infect other crops. In addition, fungicides and biological products were evaluated to identify effective management options.

The research found that persimmon dieback in Australia is not caused by a single pathogen, instead it is a complex disease involving several fungi. Some of these fungi are more aggressive than others, with certain species causing severe damage. The study also showed that the pathogens can infect multiple parts of the plant, including stems, leaves and fruit, and have the potential to infect other crops such as grapevine, olive and citrus. Environmental conditions, particularly temperature, were found to strongly influence the rate at which these fungi grow and infect plants.

The project outcomes provide important benefits for the persimmon industry by improving understanding of dieback disease and its risks. It highlights the need for accurate diagnosis, as different pathogens may require different management approaches. The findings also show that preventing infection is critical, as treatments applied after pathogen establishment are much less effective. Several fungicides and biological control products showed strong potential when applied preventatively, providing growers with practical options to reduce the impact of disease.

New knowledge generated by this work includes the identification of key fungal species in persimmon, their behaviour under different conditions, and their ability to infect a range of hosts. The project also identified nurseries as a possible pathway for disease spread, highlighting the importance of disease-free propagation and planting material. These findings provide a foundation for improved disease management strategies and can support future guidelines for growers to improve orchard practices.

Keywords

Persimmon, dieback, fungal pathogens, disease management, fungicides, biological control, cross-host infection, Australian horticulture

Introduction

Persimmon production is an emerging horticultural industry in Australia. However, its productivity is increasingly affected by dieback, a disease that causes tree decline, reduced vigour, and, in extreme cases, tree death. While dieback diseases have been extensively studied in other perennial woody crops such as grapevine and walnut (Antony et al., 2022; Billones-Baaijens & Savocchia, 2018), comparatively little was known about dieback in persimmon prior to this project (Taguiam et al., 2024).

Growers across Australia had reported symptoms consistent with dieback, but the cause, distribution, and contributing factors had not been comprehensively investigated (George et al., 2017; Taguiam et al., 2024). This lack of knowledge limited the ability of the industry to accurately diagnose the problem and implement effective management strategies.

Research on other crops has shown that dieback is commonly associated with fungal pathogens, particularly those belonging to the Botryosphaeriaceae family, which are known to infect a wide range of hosts (Antony et al., 2022; Pitt et al., 2010). These diseases are often caused by a complex of multiple fungal species rather than a single pathogen, making diagnosis and control more challenging. In addition, the prevalence and behaviour of these pathogens can vary depending on environmental conditions, crop variety, and geographic location. However, the biology, diversity, and impact of these organisms in persimmon systems were not well characterised.

The project was undertaken to address these critical knowledge gaps by providing a comprehensive investigation into persimmon dieback in Australia. The research aimed to identify the pathogens involved, determine their relative importance and aggressiveness, understand how environmental conditions influence disease development, and assess their potential to infect other crops. It also sought to evaluate management options to support improved disease control. The outcomes of this project demonstrate that persimmon dieback is a complex disease system involving multiple fungal pathogens with differing levels of virulence, environmental preferences, and host ranges.

The findings highlight the importance of accurate pathogen identification, consideration of environmental factors, and the need for preventative disease management strategies. This research is significant for the Australian persimmon industry as it provides a clearer understanding of the causes and risks associated with dieback. It also supports the development of more effective, evidence-based management practices, helping growers reduce disease impact, protect orchard productivity, and improve long-term sustainability.

Methodology

The causes, behaviour and management of persimmon dieback in Australia were investigated using several approaches. The work combined orchard surveys, laboratory isolation and identification of microorganisms, *in vitro* pathogenicity testing, establishment of persimmon trees in the glasshouse for *in vivo* trials (**Figure 1**), epidemiology studies, nursery investigations, and laboratory and glasshouse evaluation of chemical and biological management options. Most experimental work was conducted using the cultivars 'Jiro' and 'Fuyu', the predominant persimmon cultivars grown in Australia.

Orchard surveys and sample collection

Field surveys were conducted during 2023–2024 in commercial persimmon orchards across the major production regions of Australia, including New South Wales, Queensland, South Australia, Victoria and Western Australia. A total of 168 samples were collected from symptomatic and asymptomatic plant material. These included stems, leaves, buds, calyces and fruit, with a particular focus on woody tissues showing symptoms typically associated with dieback, such as internal vascular staining and necrosis (**Figure 2**). Sample metadata, including orchard location, cultivar, tissue type and symptom description, were recorded to support comparisons across regions.

Isolation and identification of associated fungi

Plant tissues collected during surveys (**Figure 3**) were processed in the laboratory for microbial isolation. Fungi were isolated from both symptomatic and asymptomatic tissues to capture pathogens as well as possible latent or endophytic species. Isolates were purified and preserved for further study. Initial identification was based on colony appearance and microscopic characteristics, including spore shape, septation and size. To confirm species identity, DNA was extracted from representative isolates and key barcode regions were sequenced. Multi-locus phylogenetic analyses were used to accurately identify the major fungal species associated with persimmon dieback.

Pathogenicity testing

A series of pathogenicity assays were conducted to determine which fungal isolates were able to cause disease symptoms and to compare their aggressiveness. Initial screening was undertaken using detached stem assays on the cultivars 'Jiro' and 'Fuyu'. Representative isolates from the major fungal groups recovered during the survey were inoculated into healthy persimmon stems and lesion development was measured after incubation. Re-isolation of the inoculated fungi was carried out to confirm pathogenicity. Based on these results, selected pathogenic isolates were further tested on 2-year-old potted persimmon trees under glasshouse conditions. These experiments were maintained for 12 months to allow disease development and assessment of internal lesion spread within woody tissue. Re-isolation and molecular confirmation were conducted to verify that symptoms were caused by the inoculated pathogens.

Cultivar susceptibility, epidemiology and host range

Additional detached stem assays were used to compare the susceptibility of six persimmon cultivars to key pathogens identified in the study. To investigate environmental influences on disease development, experiments were conducted to determine the effect of temperature on mycelial growth, sporulation and spore germination of the main pathogens. Cross-inoculation studies were also carried out to assess whether pathogens isolated from stems could infect the leaves and fruit. Host-range assays were undertaken on a range of woody hosts, including grapevine, olive, pistachio, citrus, acacia, jacaranda and rose, to assess the potential for cross-host infection.

Nursery samples

Symptomatic nursery planting material was sampled and examined for fungi as nursery materials may play an important role in disease spread. Fungi were isolated from affected tissues, identified using morphological and molecular methods, and representative isolates were screened for pathogenicity on detached persimmon stems. This component was included to assess whether nursery materials could function as a source of infection for new plantings.

Evaluation of disease management options

Potential disease management strategies were evaluated using both laboratory and plant-based assays. The bacterium *Pseudomonas poae* BCA17, previously assessed as a potential biological control, was tested *in vitro* for its ability to inhibit the growth of major persimmon dieback pathogens (Niem et al., 2023). A range of commercial biofungicides and fungicides was also screened *in vitro* to compare their activity against the main fungal species. The most promising products were evaluated further using detached stem assays and glasshouse trials (**Figure 4**). These experiments compared preventative and curative application timings to assess their potential practical use for disease management in persimmon production systems. Across all experimental components, treatments were replicated, and data were

statistically analysed to compare pathogen identity, pathogenicity, host susceptibility, environmental responses, and treatment effects.

Figure 1. Establishment of persimmon trees (cvs. Jiro and Fuyu) in a controlled environment at Charles Sturt University, Wagga Wagga, NSW, for experimental work on persimmon dieback. Photo by J.D. Taguiam, 9 December 2022.

Figure 2. Persimmon stem samples from the field showing typical dieback symptoms: (a) external appearance, (b) bark removed revealing internal staining, and (c) stem cut longitudinally showing vascular discolouration. Photo by J.D. Taguiam, 30 January 2023.

Figure 3. Sample collection in a persimmon orchard in Barooga, NSW (a) Chris Stillard (President, Persimmons Australia Inc., cstillard@gmail.com) [left] and J.D. Taguiam [right], (photo by R.B. Baaijens, regbaaijens@gmail.com, 11 January 2023); and (b) J.D. Taguiam collecting budwood during dormancy. Photo by J.D. Taguiam, 4 July 2025.

Figure 4. Fungicide trial in persimmon trees in the glasshouse at Charles Sturt University, Wagga Wagga, NSW, incubated for 10 months. Wounds were covered with plastic for 24 h to promote pathogen development, either prior to or after fungicide application. Photo by J.D. Taguiam, 20 February 2024.

Results and discussion

This project provided the first comprehensive investigation of persimmon dieback in Australia and demonstrated that the disease is best understood as a complex disease system involving multiple fungal pathogens, rather than a problem caused by a single organism.

Orchard surveys and sample collection

Surveys of commercial orchards across the main persimmon-growing regions of Australia (**Figure 5**) collected 168 plant samples from symptomatic and asymptomatic stems, leaves, buds, calyces and fruit. From these samples, 326 fungal isolates were recovered.

Isolation and identification of associated fungi

The most frequently associated fungal groups were *Neopestalotiopsis* (*Np.*), *Diaporthe* and *Neofusicoccum* (*Nf.*), and these were isolated across multiple regions and plant tissues. Molecular analyses confirmed several important pathogenic species, including *Nf. parvum*, *Nf. luteum*, *Nf. cryptoaustrale*, *Lasiodiplodia iranensis*, *Np. vheenae*, *Np. maddoxii*, and several *Diaporthe* species, particularly *D. diospyrina*.

Pathogenicity Testing

These findings demonstrated that persimmon dieback in Australia has a complex aetiology and that similar symptoms may be associated with multiple pathogens. Pathogenicity testing showed that these fungi differ in their ability to cause disease. Detached stem and glasshouse assays confirmed that several of the identified fungi could induce typical dieback symptoms, including internal vascular staining and lesion development (**Figure 6**). Among the pathogens tested, *Nf. parvum* was consistently the most aggressive, producing the largest lesions in both detached stems and potted trees. *Np. vheenae*, *Np. maddoxii* and *D. diospyrina* were also pathogenic, although generally less aggressive than *Nf. parvum*. Additional species, including *Nf. luteum* and *L. iranensis*, also showed strong pathogenicity in detached stem assays. These results are important for the industry because they reveal that persimmon dieback is not necessarily due to one causal pathogen. Diagnosis and management strategies, therefore, should consider that different pathogens may be involved with dieback symptoms, with differing levels of aggressiveness and potentially different responses to management.

Cultivar susceptibility, epidemiology and host range

Testing across six persimmon cultivars (Astringent: 'Nightingale', 'Macari'; Non-astringent: 'Suruga', 'Yang Fang', 'Jiro', 'Fuyu') showed that disease severity varied among pathogens, but no cultivar displayed strong resistance to the dieback pathogens. *Neofusicoccum luteum* and *L. iranensis* caused severe lesion development across most cultivars, while *Nf. parvum* showed some cultivar-dependent variation. *Neopestalotiopsis vheenae* caused moderate and relatively consistent disease, and *Np. maddoxii* caused generally smaller lesions. However, none of the cultivars tested could be considered resistant. This suggests that cultivar choice alone is unlikely to provide effective control of persimmon dieback under Australian conditions. At best, cultivar susceptibility may influence the degree of risk, but it should be considered only one part of a broader disease management strategy.

The outcomes from this project provided an improved understanding of how dieback develops and spreads. Pathogenicity assays showed that infection is strongly associated with wounds, particularly fresh pruning wounds. After infection, pathogens spread primarily along the vascular tissue rather than across the stem. In potted tree experiments, lesion development was greater in the longitudinal direction than in radial cross-sections, and pathogen recovery was usually confined to sections close to the inoculation point. This suggests that pathogen spread within woody tissue may initially be more localised than symptoms in mature orchards might imply. However, this localised infection remains important because many trunk disease pathogens can persist internally without causing obvious early symptoms. As a result, trees may be infected for long periods before symptoms of external decline become visible. For growers, this means disease is often established before it is visually recognised, which limits the value of reactive management.

Results from the environmental studies showed that disease risk is likely to be influenced by temperature and moisture. The main pathogens grew best between 25-27 °C, with some differences among species. Species of *Neopestalotiopsis* had slightly lower optimum growing temperatures than those from the Botryosphaeriaceae. Spore germination was rapid at warm temperatures, particularly between 25 and 30 °C, and could occur quickly even on nutrient-poor artificial growing media in the laboratory. *Neofusicoccum parvum* germinated the fastest, followed by *Neopestalotiopsis* and then *Diaporthe*. These findings suggest that under warm, moist conditions, fresh wounds can rapidly become infected. The pathogens also survived cooler temperatures and resumed growth when conditions became favourable again, indicating

that inoculum can persist in orchards across seasons. For the persimmon industry, this means infection risk is likely to vary across regions, seasons and orchard microclimates, and disease management should consider weather conditions and timing of operations such as pruning.

The results showed that the pathogens associated with persimmon dieback are not restricted to woody stems. Cross-inoculation studies demonstrated that pathogens recovered from dieback-affected stems were able to infect wounded leaves and fruit, causing necrotic lesions and rot symptoms. This indicates that non-woody tissues may also act as sites of infection and potential sources of inoculum. If infected fruit, leaves and pruning's remain in the orchard, they may contribute to pathogen survival and increase disease pressure in later seasons. This has practical implications for orchard sanitation, as management should not focus only on visibly affected branches. Host range studies further showed that several of the persimmon dieback pathogens can infect other woody plant species commonly found in agricultural and peri-urban landscapes. *Neofusicoccum parvum* had the broadest host range and highest virulence, infecting hosts including grapevine, olive, pistachio, lemon and acacia. Other Botryosphaeriaceae species also infected hosts such as grapevine, olive, rose and jacaranda, while *D. diospyrina* showed a narrower host range. These findings suggest that nearby crops and ornamental hosts such as these may act as reservoirs of inoculum, allowing pathogens to persist and potentially move across landscapes. This is particularly relevant in mixed production areas where several different crops are grown together or in close vicinity.

Nursery samples

An important additional finding of the project was the potential for nursery planting material to contribute to the spread of disease. Sampling of symptomatic nursery stock identified multiple *Neopestalotiopsis* species associated with dieback symptoms around the graft union. These included known species such as *Np. iberica*, *Np. nebuloides* and *Np. zakeelii*. Preliminary pathogenicity tests confirmed that representative nursery isolates could cause lesions in persimmon stems. This suggests that nursery material may act as a pathway for introducing dieback-associated fungi into new orchards. From an industry perspective, this highlights the need to consider nurseries as a critical part of disease management, including improved hygiene, monitoring, and the use of clean planting material.

Evaluation of disease management options

The management studies showed that several biological and chemical options have potential for reducing disease risk, but they also clearly demonstrated that preventative treatment is much more effective than curative treatment. The biological control bacterium *Pseudomonas poae* BCA17 strongly suppressed all major pathogens in laboratory assays, particularly through the activity of its culture filtrates, which reduced fungal growth by more than 90% in all experiments. Commercial *Bacillus*-based biofungicides, including Serenade® Opti, Serenade® Prime and Serifel®, also consistently reduced fungal growth *in vitro* across the pathogen complex. Among fungicides, Score® (difenoconazole), Seguris® Flexi (isopyrazam), Luna® Experience (tebuconazole + fluopyram), Cabrio® (pyraclostrobin), and Emblem® (fluazinam) were among the strongest performers in laboratory assays, with some additional products also showing useful activity depending on the pathogen.

When these products were tested in detached stem and glasshouse systems, applications applied before infection were significantly more effective than those applied after infection. Curative applications failed to reduce pathogen recovery once infection had established. In contrast, preventative applications reduced pathogen recovery, especially for *Nf. parvum*. In glasshouse trials, Score® provided the strongest preventative suppression of *Nf. parvum*, while Serenade® Opti also showed promise. However, *Np. vheenae* was less responsive to the treatments tested, indicating that management efficacy can vary across the pathogen complex. These findings are highly relevant for growers because they show that dieback disease management in persimmon should focus on protecting fresh wounds, rather than attempting to treat infections after colonisation of the pathogen has already occurred.

Overall, the results of this project support an integrated disease management approach for persimmon dieback in Australia. Such an approach should include accurate diagnosis of the pathogens present, use of disease-free nursery stock, reduction of inoculum through orchard hygiene, disinfection of pruning and grafting tools, protection of fresh wounds with effective fungicides or biological products, and consideration of nearby host plants that may act as inoculum reservoirs. The work also highlights the need for flexible, risk-based management that takes account of seasonal conditions, labour constraints and the realities of mixed production landscapes. This project has provided the Australian persimmon industry with the first evidence that dieback is a multi-pathogen disease system with important implications for orchard productivity, planting material, and long-term management. It also provides a strong foundation for future work, including field validation of management practices, improved nursery diagnostics, evaluation of new cultivars and scion–rootstock combinations, and development of practical guidelines for growers and industry stakeholders.

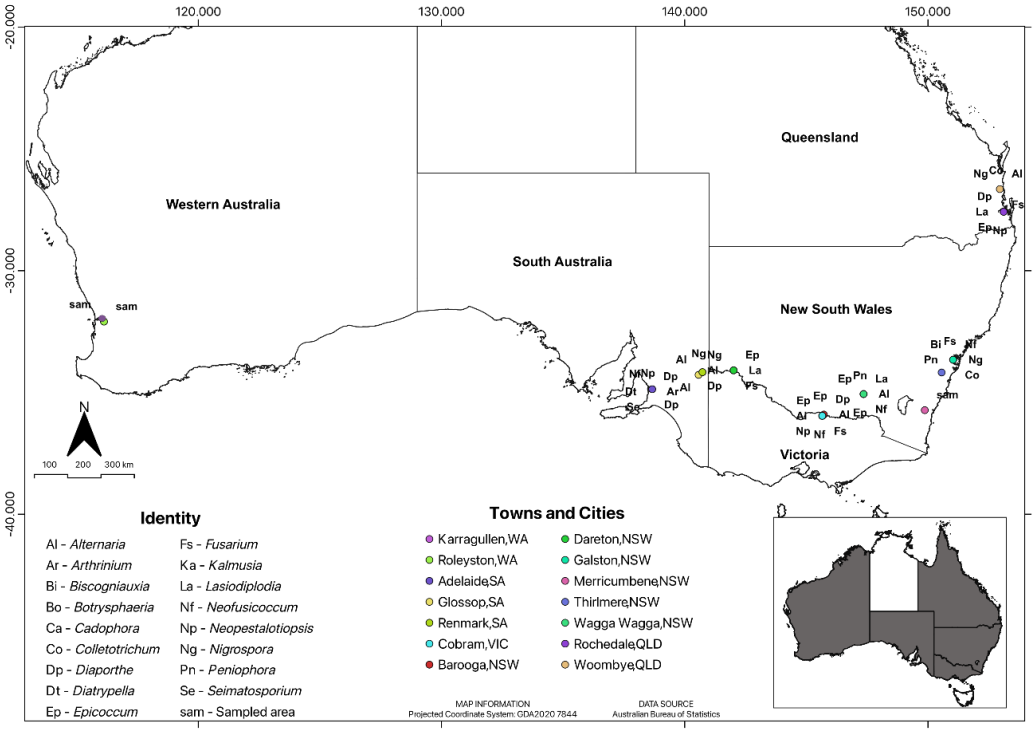

Figure 5. Geographical distribution of fungal genera isolated from persimmons in Australia. Photo by C.E. Gregorio-Taguiam (cgregorio-taguiam@csu.edu.au) generated using QGIS 3.34, 19 January 2026.

Figure 6. Pathogenicity assessment in glasshouse grown persimmon trees at Charles Sturt University (a) Pathogens were inoculated into artificially wounded sites on the stems and covered with Parafilm® to prevent desiccation and cross-contamination. (b) Stems showing internal dieback symptoms after the incubation period. Photo by J.D. Taguiam, 20 February 2024.

Outputs

Table 1. Output summary

Output	Description	Detail
PhD research project completed	A PhD research project investigating persimmon dieback in Australia was successfully completed through Charles Sturt University.	Confirmation of candidature was completed, and the thesis was submitted to the University for external examination on 27 March 2026.
Experimental plant material established	Persimmon plants were established and maintained for controlled pathogenicity and glasshouse experiments.	Persimmon trees were grown in controlled research facilities for use in pathogenicity and fungicide/biofungicide assays.
Documented collaboration with PR17000	To establish partnerships with project leader for PR17000.	Samples collected in June 2023 from 'Jiro', 'Fuyu', 'Rojo Brillante', 'Sunami', and 'Shinsu' varieties grown at NSW DPI Dareton, NSW were incorporated into the isolate collection.
Raise awareness of dieback pathogens, their assessment and collection via communication with Persimmon Australia and PR20000 Persimmon industry communication program and including in-field hands-on workshops with growers.	To establish partnerships under PR2000 for dissemination of research outputs.	Partnership established with the Executive Officer of Persimmon Australia, who became part of the PhD supervisory team. Five articles were published in the Persimmon Press newsletter. Two oral presentations were delivered at national persimmon conferences (Mildura, VIC in 2023; Toowoomba, QLD in 2026). The PhD candidate also regularly attended and presented project updates at the Project Reference Group and Persimmon SIAP meetings.
Dieback-associated pathogens identified	Multiple fungal pathogens associated with persimmon dieback in Australia were identified and characterised.	*Neofusicoccum*, *Neopestalotiopsis*, and *Diaporthe* species were identified, including the first report of several pathogens associated with this pathosystem in Australia.
Orchard survey and pathogen distribution data determined	Surveys of Australian persimmon orchards were conducted to determine pathogen incidence and distribution.	Overall, 168 samples were collected and 326 fungal isolates were obtained. Incidence patterns were analysed across regions, with results incorporated into the PhD thesis and supporting datasets. An incidence map was also developed.
Molecular identification and data generated and shared in public databases	DNA-based identification of pathogens was conducted, and resulting sequences were contributed to public databases.	ITS, TEF1-α, and TUB2 sequence data were generated and lodged in NCBI GenBank (**Table 4**).
Herbarium accession of isolates obtained	Representative fungal isolates were preserved and accessioned.	Specimens were lodged with the NSW Plant Pathology & Mycology Herbarium (DAR collection) in Orange, NSW.
Nursery samples assessed	Nursery materials were assessed as potential sources of infection and pathogen introduction.	Nursery sampling was conducted, and multiple *Neopestalotiopsis* spp. were identified.

Table 1. continued

Output	Description	Detail
Cultivar susceptibility initially assessed	Relative susceptibility of commercially important persimmon cultivars was evaluated.	Susceptibility of 'Jiro' and 'Fuyu' cultivars was documented using both detached stem and glasshouse assays. Susceptibility of additional cultivars ('Nightingale', 'Macari', 'Suruga', and 'Yang Fang') was also assessed using detached stem assays.
Epidemiological insights into disease development assessed	Key biological and epidemiological drivers of persimmon dieback were characterised.	Infection via pruning wounds was confirmed, temperature–growth relationships were established, and cross-infection to leaves, fruit, and other hosts was demonstrated.
Host range assessed	Ability of pathogens to infect alternative hosts was assessed.	Pathogens were shown to infect grapevine, olive, pistachio, citrus, acacia, and ornamental hosts, among others, with implications for landscape-level disease spread identified.
Biocontrol agents assessed	Biological control agent were assessed for activity against dieback pathogens.	*Pseudomonas poae* BCA17 was evaluated, with strong inhibition (>90% suppression) observed *in vitro* and in culture filtrate assays.
Fungicide/Biofungicide efficacy evaluated	Control options were evaluated under laboratory, detached stem, and glasshouse conditions.	Multiple commercial fungicides were tested; Score® (difenoconazole) showed the greatest efficacy among fungicides, while Serenade® Opti was the most promising biofungicide.
Scientific outputs and publications generated	Scientific knowledge was generated and disseminated through publications.	PhD thesis was submitted; one paper was published in a Q1 journal, with additional chapters in preparation for submission to peer-reviewed journals.

Outcomes

Table 2. Outcome summary

Outcome	Alignment to fund outcome, strategy and KPI	Description	Evidence
Improved understanding of persimmon dieback pathogen complex	SIP outcome 2: The Australian persimmon industry has increased profitability, efficiency and sustainability through innovative R&D, sustainable BMPs and varieties. Strategy 3: Develop and optimise fit-for-purpose sustainable pest and disease management strategies. KPI: Development of pest and disease management strategies that mitigate crop loss in collaboration with growers.	The project established that persimmon dieback in Australia is a complex disease involving multiple pathogens rather than a single causal agent.	Isolation and identification of *Neofusicoccum parvum*, *Neopestalotiopsis spp.*, and *Diaporthe diospyrina*; pathogenicity assays; first reports in Australian pathosystem.
Improved epidemiological understanding of infection pathways		The research identified pruning wounds and grafting sites as primary infection courts, with pathogens colonising vascular tissues and persisting asymptomatically.	Pathogenicity assays and lesion progression data; demonstration of wound-mediated infection and limited internal spread.
Identification of nursery pathways as a disease risk		Differences in disease severity between cultivars ('Fuyu' and 'Jiro') were identified, indicating variability in susceptibility and lack of resistance.	Detached stem pathogenicity assays showing lesion variation among cultivars.
Improved knowledge of fungicide efficacy		The project identified effective fungicides and demonstrated variability in pathogen sensitivity.	*in vitro* growth assays; glasshouse and detached stem validation.
Identification of effective disease management strategy		Preventative fungicide applications were shown to be significantly more effective than curative treatments.	Detached stem and glasshouse trials showing reduced re-isolation with preventative treatments.
Evaluation of biological control potential		The project demonstrated that *Pseudomonas poae* BCA17 has strong inhibitory activity against multiple dieback pathogens.	Dual culture and culture filtrate assays showing 24–96% inhibition.
Increased capacity for evidence-based disease management		The project provides a scientific basis for shifting from reactive to preventative, risk-based management strategies in persimmon production.	Integration of epidemiology + management findings; recommendations on pruning wound protection, sanitation, and nursery hygiene.
Contribution to industry awareness of dieback disease		Engagement with growers and industry improved awareness of dieback pathogens, infection pathways, and management approaches.	Industry communication, collaboration with Persimmons Australia, nursery engagement, and extension activities.

Monitoring and evaluation

Table 3. Key Evaluation Questions

Key Evaluation Question	Project performance	Continuous improvement opportunities
To what extent has the project achieved its expected outcomes?	Successfully improved understanding of persimmon dieback in Australia by showing it is caused by multiple fungal pathogens rather than a single agent. Identified key pathogen groups, their distribution, and pathogenicity, providing a foundation for better diagnosis and management.	Future research should focus on disease epidemiology, environmental drivers, and field validation of management practices. This further work is required to translate these findings into practical management strategies.
How relevant was the project to the needs of intended beneficiaries?	Highly relevant to industry needs which addressed dieback disease as a key priority. Improved capacity through better understanding of the causal pathogens and their impact. Delivered both applied (disease identification and understanding) and academic outcomes (PhD and scientific outputs).	Future projects could enhance relevance by increasing grower involvement through field and nursery trials, as well as in setting priorities and co-developing management strategies.
How well have intended beneficiaries been engaged in the project?	Engagement with growers was achieved through multi-region field sampling, participation in industry events (e.g. Persimmon Conferences), and communication via the Project Reference Group and Persimmons Australia Inc. Regular updates provided via the Persimmon Press Newsletter and presentations at SIAP meetings kept stakeholders informed of progress and preliminary findings.	While engagement was effective within the scope of a PhD program, more regular and structured activities (e.g. workshops, on-farm and nursery trials, and targeted feedback sessions) could strengthen collaboration and knowledge exchange. Participation was strong in some regions, but more limited in others. The results of this study may help encourage broader grower involvement in future projects.
To what extent were engagement processes appropriate to the target audience/s of the project?	Engagement processes were appropriate within the constraints of a PhD project. Extension activities (e.g. conference presentations and field days) provided accessible platforms for grower engagement, and communication through established industry channels ensured information reached relevant stakeholders.	Future projects should emphasise grower participation, particularly through activities such as field and nursery trials, as well as sample submission and on-farm involvement.
What efforts did the project make to improve efficiency?	The project was delivered efficiently by using a PhD program to achieve both research outcomes and skills development. For example, the development and use of a sampling protocol improved efficiency by making it more cost-effective to collect samples, while also encouraging grower participation. This also assisted growers in learning how to identify symptoms and to collect samples for sending to the laboratory for analysis.	Build on existing communication efforts by enhancing the timeliness and regularity of updates to growers through both direct engagement and Persimmons Australia Inc.

Recommendations

This project demonstrated that persimmon dieback in Australia is a complex disease requiring an integrated, preventative management approach. The findings indicated that growers should prioritise protection of pruning and other wounds, maintain strict orchard hygiene, disinfect tools regularly, and use certified disease-free nursery material, as curative treatments were ineffective once infection was established.

Future RD&E should focus on field validation of management strategies that were deemed effective in laboratory, detached stem and glasshouse trials. Also, a targeted investigation of transmission pathways of dieback pathogens in the nursery setting is necessary. Given the diversity of pathogens involved in persimmon dieback infection and the similarity of symptoms they produce, reliable diagnosis based on visual assessment alone is not feasible. Hence, there is a need to develop accessible molecular diagnostic tools for accurate pathogen identification, such as multiplex qPCR assays capable of simultaneously detecting and quantifying the key pathogens associated with persimmon dieback.

Research should also evaluate integrated control strategies, including fungicides, biological control agents, and biorationals. Initial screening of potential biopesticides and biorationals should be undertaken, with the most promising candidates progressing to field trials. Further work should characterise metabolites of selected products to identify bioactive compounds associated with antifungal activity against dieback pathogens. In addition, profiling of endosphere microbiomes in scion and rootstock tissues from healthy and infected trees is recommended to better understand shifts in microbial communities associated with disease development and treatment responses.

To support industry adoption, project outcomes should be translated into practical management guidelines, extension materials, and targeted training activities. Continued collaboration among growers, nurseries, researchers, and industry stakeholders will be essential. These actions will help maximise the impact of this research and support the development of sustainable, evidence-based management strategies for the Australian persimmon industry.

Refereed scientific publications

Journal article

Taguiam, J. D. W., Billones-Baaijens, R., Stodart, B. J., Steel, C. C., Fuss, A. M., & Savocchia, S. (2024). A threat to the persimmon industry: A review of persimmon (Diospyros species) dieback. Crop Protection, 180, 106672.

PhD Thesis

Taguiam, J.D.W., Stodart, B.J., Steel, C.C., Fuss, A.M., Savocchia, S. (2026). Diagnosis and management of dieback in persimmon trees. Submitted to Charles Sturt University on 27 March 2026, in fulfilment of the requirements for the degree of Doctor of Philosophy; Gulbali Institute, Faculty of Science and Health, School of Agricultural, Environmental and Veterinary Sciences (Under Examination).

Chapter in a book or paper in conference proceedings

Taguiam, J.D.W., Stodart, B.J., Steel, C.C., Fuss, A.M., Savocchia, S. (2026). Plant pathogens down under: An overview of persimmon dieback research in Australia. 2026 Australian Persimmon Industry Conference and Field Days. February 3-4, 2026, Oaks Hotel Toowoomba, QLD, Australia.

Taguiam, J.D.W., Stodart, B.J., Steel, C.C., Fuss, A.M., Savocchia, S. (2025). Preliminary evaluation of fungicide efficacy against key pathogens of persimmon dieback. 2025 Faculty of Science and Health HDR and Honours Symposium, November 18-19, 2025, Charles Sturt University, Wagga Wagga, NSW, Australia.

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Intellectual property

This project generated intellectual property in the form of DNA sequence information (Table 4). There is no commercialization to report.

PRE- EXISITNG IP (Background IP and Third-Party IP) brought into Project							
No	**Name of IP, if any**	**Type of Output**	**Usage**	**Nature of IP**	**Conditions of use**	**Confidentiality**	**Risks identified in relation to the IP**
1	Identification of dieb	Molecular Markers	Dissemination	Confidential Information	Permit	Confidential	Nil
2	Biocontrol agent	Biological Control Agent	Dissemination	Confidential Information	Permit	Confidential	Nil
3		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
4		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
5		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
6		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	

If Type of Output is designated as "other" provide	please provide details here.
Proprietor/Owner/Licensor of IP listed above:	**If the IP listed above is associated with another Hort Innovation Project insert the Hort Innovation Project number here or if not**
Molecular markers - Charles Sturt University, SARDI, Wine Australia, The University of	(Insert details here)

PROJECT IP That is intellectual property developed during the Project							
No	**Name of IP, if any**	**Type of Output**	**Usage**	**Nature of IP**	**Conditions of use**	**Confidentiality**	**Risks identified in relation to the IP**
1	Pathogen sequence	Molecular Markers	Dissemination	Confidential Information	Creative Commons	Confidential	Nil
2		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
3		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
4		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
5		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	
6		Choose an item.	Choose an item.	Choose an item.	Choose an item.	Choose an item.	

If Type of Output is designated as "other" provide details here:	

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Appendices

Table 4. GenBank accession numbers of fungal isolates from the persimmon dieback study

Species	Isolate	GenBank Accession Number		
		ITS	TEF	TUB
Botryosphaeria dothidea	CSU_020-2a	PQ008474	PV649006	N/A
Diaporthe diospyrina	CSU_005-2a	PX700278	PZ049366	PZ049354
Diaporthe diospyrina	CSU_006-1a	PX700279	PZ049367	PZ049355
Diaporthe diospyrina	CSU_023-1a	PX700282	PZ049370	PZ049358
Diaporthe diospyrina	CSU_030-1a	PX700284	PZ049372	PZ049360
Diaporthe diospyrina	CSU_036-1a	PX700286	PZ049374	PZ049362
Diaporthe fraxini-angustifoliae	CSU_032-1a	PX700285	PZ049373	PZ049361
Diaporthe fraxini-angustifoliae	CSU_046-1a	PX700287	PZ049375	PZ049363
Diaporthe fraxini-angustifoliae	CSU_071-1b	PX700289	PZ049377	PZ049365
Diaporthe kongii	CSU_012-1a	PX700280	PZ049368	PZ049356
Diaporthe kongii	CSU_024-3c	PX700283	PZ049371	PZ049359
Diaporthe yunnanensis	CSU_021-2a	PX700281	PZ049369	PZ049357
Diaporthe yunnanensis	CSU_049-1a	PX700288	PZ049376	PZ049364
Lasiodiplodia iranensis	CSU_033-1a	PQ008475	PV649005	N/A
Lasiodiplodia iranensis	CSU_009-1a	PQ008472	PV649007	N/A
Neofusicoccum cryptoaustrale	CSU_107-1a	PQ008481	PV648999	N/A
Neofusicoccum cryptoaustrale	CSU_061-1b	PQ008478	PV649002	N/A
Neofusicoccum luteum	CSU_ 036-1c	PQ008476	PV649004	N/A
Neofusicoccum luteum	CSU_039-1a	PQ008477	PV649003	N/A
Neofusicoccum parvum	CSU_098-2b	PQ008480	PV649000	N/A
Neofusicoccum parvum	CSU_098-1b	PQ008479	PV649001	N/A
Neofusicoccum parvum	CSU_024-1a	OR710781	N/A	N/A
Neofusicoccum parvum	CSU_014-1a	PQ008473	N/A	N/A
Neopestalotiopsis camelliae-oleiferae	CSU_091-1a	PX048499	PZ056231	PZ056192
Neopestalotiopsis camelliae-oleiferae	CSU_102-1a	PX048500	PZ056240	PZ056201
Neopestalotiopsis camelliae-oleiferae	CSU_104-1a	PX048501	PZ056241	PZ056202
Neopestalotiopsis camelliae-oleiferae	CSU_106-1a	PX048502	PZ056243	PZ056204
Neopestalotiopsis camelliae-oleiferae	CSU_107-1b	PX048503	PZ056244	PZ056205
Neopestalotiopsis camelliae-oleiferae	CSU_108-1a	PX048504	PZ056245	PZ056206
Neopestalotiopsis iberica	CSU_105-1a	PX048497	PZ056242	PZ056203
Neopestalotiopsis iberica	CSU_115-1a	PX048498	PZ056247	PZ056208
Neopestalotiopsis maddoxii	CSU_005-1a	PX048493	PZ056229	PZ056190
Neopestalotiopsis nebuloides	CSU_117-1a	PX726396	PZ056248	PZ056209
Neopestalotiopsis sp.	CSU-036-2b	PZ049821	PZ056230	PZ056191
Neopestalotiopsis sp.	CSU_094-5a	PZ049822	PZ056232	PZ056193
Neopestalotiopsis sp.	CSU_096-2a	PZ049823	PZ056234	PZ056195
Neopestalotiopsis sp.	CSU_098-1a	PZ049824	PZ056235	PZ056196
Neopestalotiopsis sp.	CSU_098-2a	PZ049825	PZ056236	PZ056197
Neopestalotiopsis sp.	CSU_100-1a	PZ049826	PZ056238	PZ056199
Neopestalotiopsis sp.	CSU_101-1a	PZ049827	PZ056239	PZ056200
Neopestalotiopsis sp.	CSU_111-1a	PZ049828	PZ056246	PZ056207

Table 4. continued

Species	Isolate	GenBank Accession Number		
		ITS	TEF	TUB
Neopestalotiopsis sp.	CSU_118-1a	PZ049829	PZ056249	PZ056210
Neopestalotiopsis sp.	CSU_120-1a	PZ049830	PZ056250	PZ056211
Neopestalotiopsis sp.	CSU_122-1a	PZ049831	PZ056251	PZ056212
Neopestalotiopsis sp.	CSU_124-1a	PZ049832	PZ056253	PZ056214
Neopestalotiopsis sp.	CSU_127-1a	PZ049833	PZ056254	PZ056215
Neopestalotiopsis sp.	CSU_128-1a	PZ049834	PZ056255	PZ056216
Neopestalotiopsis sp.	CSU_129-1a	PZ049835	PZ056256	PZ056217
Neopestalotiopsis sp.	CSU_131-1a	PZ049836	PZ056257	PZ056218
Neopestalotiopsis sp.	CSU_132-1a	PZ049837	PZ056258	PZ056219
Neopestalotiopsis sp.	CSU_135-1a	PZ049838	PZ056259	PZ056220
Neopestalotiopsis sp.	CSU_136-1a	PZ049839	PZ056260	PZ056221
Neopestalotiopsis sp.	CSU_137-1a	PZ049840	PZ056261	PZ056222
Neopestalotiopsis sp.	CSU_139-1a	PZ049841	PZ056262	PZ056223
Neopestalotiopsis sp.	CSU_140-1a	PZ049842	PZ056263	PZ056224
Neopestalotiopsis sp.	CSU_145-1a	PZ049843	PZ056264	PZ056225
Neopestalotiopsis sp.	CSU_146-1a	PZ049844	PZ056265	PZ056226
Neopestalotiopsis sp.	CSU_148-1a	PZ049845	PZ056266	PZ056227
Neopestalotiopsis vheenae	CSU_004-1b	PX048492	PZ056228	PZ056189
Neopestalotiopsis zakeelii	CSU_095-2a	PX048495	PZ056233	PZ056194
Neopestalotiopsis zakeelii	CSU_099-1a	PX048494	PZ056237	PZ056198
Neopestalotiopsis zakeelii	CSU_123-1a	PX048496	PZ056252	PZ056213