Ecology and management of grapevine trunk diseases in New Zealand: a review

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Abstract Grapevine trunk diseases threaten the longevity of vineyard production in New Zealand. This paper provides a summary of the knowledge of the most common fungal grapevine trunk diseases, as well as identifying gaps that require further research. Current knowledge of symptoms, causal organisms, etiology and disease control of botryosphaeria die-back, esca, eutypa die-back and Petri disease are discussed. Additional information about how these diseases may be influenced by different vineyard practices common in New Zealand viticulture is provided.

Keywords grape, ecology, symptoms, integrated management, trunk disease.

INTRODUCTION

With an increased awareness of grapevine trunk diseases that may affect the longevity of vineyards in New Zealand, it was considered timely to present a summary of the current knowledge of the diseases and the methods of control. Selected diseases covered are botryosphaeria dieback, esca, eutypa dieback and Petri disease. While esca has not yet been recorded in New Zealand, this disease has been included as it is a major disease in other grape-growing regions of the world and some of the fungi involved in this complex are present in New Zealand.

BOTRYOSPHAERIA DIEBACK

In some early reports on botryosphaeria dieback (also known as black dead arm) mixed isolations of *Phomopsis viticola* and *Sphaeropsis malorum* (syn. *Botryosphaeria stevensii*) were reported. *Phomopsis viticola* was initially described as the pathogen responsible for symptoms (Chamberlain et al. 1964). *Botryosphaeria* species associated with the dieback disease have a wide host range, can be both saprophytic or endophytic and have been isolated from a number of symptomatic grapevine tissues (Amponsah et al. 2008).

Symptoms

Early signs of infection include mild leaf chlorosis or leaf wilt as the water transport system becomes blocked. Following invasion of the xylem by pathogens, vascular occlusions form (Sun et al. 2008) resulting in vessel blockage. In vines with botryosphaeria dieback the blocked xylem often results in black stains forming in the necrotic

xylem tissue. The stains expand longitudinally and laterally within the wood. The cambium over the non-functional xylem also dies, resulting in a canker that is visible on the exterior of the trunk or arm. Affected vines wilt suddenly during the growing season or fail to break dormancy (Pearson & Goheen 1988). Bunch rot symptoms of shrivelled brown berries with small black spore-bearing structures (Nicholas et al. 2003) have also been reported in South Africa (Pearson & Goheen 1988) and New Zealand (Buchanan & Beever 1987).

Causal organism

Field diagnosis of the causal organism, *Botryosphaeria* spp., is often difficult, as both phomopsis cane and leaf spot and eutypa dieback can have similar symptoms. The causal agent for black dead arm in Hungary has been reported as *Botryosphaeria stevensii* (Pearson & Goheen 1988). In New Zealand, *Botryosphaeria* spp. reported as the causal agent of bunch rots in Auckland and Te Kauwhata (Buchanan & Beever 1987) were later confirmed as *Botryosphaeria lutea* (*Neofusicoccum luteum*) (Phillips et al. 2002). New Zealand isolates of *B. lutea, B. australis* and *B. parva* are reported as infecting wounded green shoots of Pinot noir, Chardonnay, Riesling, Cabernet Sauvignon and Sauvignon blanc varieties (Amponsah et al. 2009a).

Disease cycle and epidemiology

Botryosphaeria stevensii overwinters in diseased woody parts of vines, with pycnidia developing during rainy periods in spring and autumn (Pearson & Goheen 1988). Mechanical injuries such as pruning wounds are the suspected entry points for tissue infection (Pearson & Goheen 1988). Experiments conducted on potted vines using New Zealand isolates show that wound age is important for infection, with a greatly reduced incidence of vine infection 30 days following wounding, compared with the infection incidence up to 3 days after wounding (Amponsah et al. 2009a). Timing of wounding may also be important, with the presence of sap in spring keeping the wounds wet – potentially a factor that increases the risk of infection (Pearson & Goheen 1988). Rainwater run-off may be important in the release and dispersal of conidia under New Zealand vineyard conditions (Amponsah et al. 2009b). Year-round trapping of conidia in rainwater traps has been reported in Canterbury, with the highest recorded trap catches during December, January and February when high mean daily temperatures (15 to 20°C) were recorded (Amponsah et al. 2009b). Optimal temperature for infection is between 23 and 26°C, but infection can occur at a range from 15 to 26°C (Pearson & Goheen 1988).

Control

In the absence of effective chemical control, removal and destruction of diseased vine parts is recommended (Pearson & Goheen 1988).

ESCA

Esca (also known as black measles) is the disease complex that results in a trunk rot of older grapevines. The disease has been widely reported in viticultural regions throughout the world (Rooney-Latham et al. 2005). The symptoms that characterise this disease have not been described in New Zealand, although some of the fungi involved have been reported (Clearwater et al. 2000; Whiteman et al. 2002; Mundy et al. 2009a). The disease has been highly destructive in vineyards in California (Gubler et al. 2005; Rooney-Latham et al. 2005) and Europe (Surico et al. 2008), so the New Zealand industry needs to remain vigilant.

Symptoms

The name esca describes the soft white rotted fibres in the trunk of the grapevine that were used for tinder, a characteristic symptom of this disease (Surico 2008). Other symptoms include "apoplexy" or sudden wilting of the vine with the shedding of some or all leaves and fruit, superficial brown to purple spots on the berry surface (measles), vascular discolouration of wood, typical tiger-striped patterns on leaves and tip die back of shoots (Eskalen et al. 2007).

Table 1 Fungi associated with the esca disease of older grapevines.

Grouping	Genus (and species where known)	Present in New Zealand[2]
Wood staining[1]		
	Phaeomoniella chlamydospora	Yes
	Phaeoacremonium species including *aleophilum, angustius, iranianum, mortoniae, parasiticum, subulatum, venezelense*	*P. aleophilum, P. armeniacum, P. globosum, P. occidentale*
White rots		
	Fomitiporia australiensis	No
	Fomitiporia mediterranea	No
	Fomitiporia polymorpha	No
	Fomitiporella vitis	No
	Inocutis jamaicensis	No
	Stereum hirsutum	Yes

[1]Summary of wood-staining *Phaeoacremonium* species taken from Surico et al. (2008).
[2]Presence in New Zealand according to Landcare Research (2010).

Causal organism
The various fungi associated with the esca complex (Table 1) can be grouped into two classes, wood staining and white rotting. In some cases leaf symptoms are expressed when only the wood-staining fungi are present. No esca symptoms have been reported in the field when only the white rot fungi are present.

Disease cycle and epidemiology
In California, propagules of *Phaeoacremonium* species were detected in soil and standing water, as well as in the air of infected vineyards (Rooney-Latham et al. 2005). *Togninia minima* (the teleomorph of *Ph. aleophilum*) was detected in both laboratory mating experiments and on moist incubated grapevine pieces from naturally infected vineyards, suggesting that in California at least this is the fungus associated with symptom expression (Rooney-Latham et al. 2005). The correlation between trapped propagule numbers and rainfall events suggests that the pathogen moves as airborne inoculum during rainfall. The complete life cycle and hence the importance of propagule dispersal for the fungus is still unknown.

Phaeoacremonium species are detected in wood of vines with either Petri disease (described below) or esca. Vines that do not show symptoms of the diseases can have the fungi growing in the wood and transfer of these pathogens can occur during propagation.

Control
In Europe, esca is a considerable disease for which no chemical control is available. Identification will be important if Esca establishes in New Zealand so that the best possible control measures can be implemented. One of the main methods of control currently recommended is to control the nursery process carefully so that *Phaeoacremonium* species do not establish in the vineyard via infected planting material (Surico et al. 2008).

EUTYPA DIEBACK

Eutypa dieback (also known as dying arm or dead arm) occurs in most grape-growing regions of the world where rainfall exceeds 600 mm annually (Pearson & Goheen 1988). Within New Zealand, the disease is distributed in both the North and South Islands (Mundy & Manning 2007; Mundy et al. 2009b).

Symptoms

According to Pearson & Goheen (1988), symptoms include deformed and discoloured shoots in spring whilst cupped, small and chlorotic leaves are normally the first symptoms on vines older than 8 years. In cordon-pruned vines, symptoms become more extreme in successive years until part or all of the arm fails to produce shoots in spring. Close examination of symptomatic cordons will often reveal cankers. Removal of bark is required to determine the extent of the canker. In New Zealand cane-pruned vines may show a lack of shoot growth from one side of the head or no dieback symptoms before vine death. In the field, cross sections of necrotic sapwood may be found to extend from the origin of the canker in the trunk or arm (Pearson & Goheen 1988) often in a wedge-shaped lesion. Culturing of fungi is required to determine if lesions result from eutypa dieback or from botryosphaeria dieback.

Causal organism

The casual organism of eutypa dieback is *Eutypa lata* (anamorph *Libertella blepharis*). *Eutypa* can be cultured from colonised wood chips placed on agar media and conidiomata may develop from the culture after 6–8 weeks (Pearson & Goheen 1988). However, identification based only on morphological features in culture can be insufficient to distinguish *E. lata* from other ascomycetes possessing *Libertella* anamorphs, including *Eutypella*, *Diatrypella*, *Diatrype*, *Cryptosphaeria* and *Cryptovalsa* (Pitt et al. 2010). Cultures produce a white mycelium growth from infected wood chips after 3 or more days at 20–25°C. A 12-h light-dark regime promotes formation of conidiomata and sporulation (Pearson & Goheen 1988). Cultures of the Diatrypaceae collected from New Zealand have been identified as *Eutypella vitis* or *Eutypa* sp., based on sequences of the internal transcribed spacer (ITS) regions (M.A. Manning, Plant & Food Research, unpublished data). Continued studies in New Zealand will be required to determine the presence and importance of other *Libertella* in symptomatic grapevines.

Disease cycle and epidemiology

According to Nicholas et al. (2003), perithecia develop on infected wood and can form 2 or more years after the tissue is killed. Infections of apricots, almonds, plums, apples, pears and a range of other fruit and ornamental trees can also be sources of spores. Spores are released during rainfall and splashed or blown via air currents onto recent pruning wounds. The fungus grows into the xylem and blocks the water-conducting vessels. Infections move at a rate of 10–12 cm/year. Foliage symptoms do not normally occur until at least 4 years after the initial infection occurs. Following infection of fresh pruning wounds, *E. lata* colonises the xylem, cambium and phloem (wood). On infected wood, perithecial stromata are produced firstly at the site of initial infection and then spread on dead wood with the canker development (Pearson & Goheen 1988). The pathogen is disseminated solely by the pale yellow and allantoid ascospores that are released from the perithecia (Pitt et al. 2010).

Control

Current control practices are focused on pruning, with remedial cuts to remove infected wood, timing to avoid the most susceptible period of wounds in early winter, avoiding pruning during or directly after rainfall, and the use of wound protection paints (Pitt et al. 2010). In New Zealand two products are currently registered with label claims for use as a wound protectant against *E. lata*; these are VineVax™ and Greenseal™.

PETRI DISEASE

Petri disease (also known as young esca, young vine decline or black goo) is a concern in the wine industry because of the damage the disease can cause to young plantings. Petri disease is also a concern as it may lead to the establishment in vineyards of fungi that may later be involved in expression of esca.

Symptoms

With Petri disease, growth slows with a reduction in trunk growth, shortened internodes, reduced

foliage and reduced leaf size (Scheck et al. 1998). Vines may grow normally in the first year but decline in subsequent seasons, with leaf chlorosis and early defoliation. Vines with symptoms generally have dark-brown to black staining of the vascular tissue, observed when the trunks are cut in cross or longitudinal section (Whiteman et al. 2007). The symptoms may result in vine death or poor establishment of new plantings.

Causal organism

Phaeomoniella chlamydospora, Phaeoacremonium species and other pathogens can all be isolated from young vines, often from the same tissue, complicating determination of which fungi are responsible for symptoms. In Australia, *P. chlamydospora* was the fungus predominately isolated from vines with Petri disease (Edwards & Pascoe 2004) and New Zealand studies have also indicated a regular occurrence of this pathogen (Clearwater et al. 2000). While *Phaeoacremonium* species may not be as commonly isolated, they are detected in symptomatic plants and their relative importance is yet to be determined.

Disease cycle and epidemiology

Many of the staining fungi isolated in Petri disease vines are the same as those associated with esca, and it is likely that the infection cycle for these pathogens is the same as that described above for esca. In 2009, three new *Phaeoacremonium* species on grapevines in New Zealand were confirmed from non-symptomatic grape rootstock mother-vines (Graham et al. 2009). This report is consistent with the conclusion that rootstock mother-vines were the primary source of *P. chlamydospora* for grafted vines (Whiteman et al. 2007). These records are further supported by findings in Spain (Giménez-Jaime et al. 2006) and South Africa (Fourie & Halleen 2004).

In New Zealand, reported pathways of infection by *P. chlamydospora* include propagules in the soil and infection during grafting due to nursery practices involving repeated exposure to cutting and hydration processes (Whiteman et al. 2002). Therefore the production of young vines and the soils used for nursery plantings have been investigated (Ridgway et al. 2002).

Control

The use of clean planting material without *P. chlamydospora* and *Phaeoacremonium* species would be ideal. However, detection of the fungus in mother-vines is difficult, and identification based on visual symptoms such as black staining has been reported to have a high proportion of false positives (Whiteman et al. 2007). Molecular methods of detection and identification have been investigated (Pottinger et al. 2001; Ridgway et al. 2002; Whiteman et al. 2002; Weir & Graham 2009) but, to date, no commercial service has been provided to allow certification of planting material free of Petri disease-related fungi. Therefore, current control activities are focused on reducing the risk of producing infected grafted plants. Hot water treatment to reduce the chance of fungal infection of new planting material has been investigated as a possible method of control (Graham 2007), but caution has been advised before widespread adoption of this practice (Whiteman et al. 2007). No effective chemical control for the field or nursery has yet been established.

CONCLUSIONS

Knowledge of the fungi responsible for grapevine trunk diseases has increased since the first international workshop on grapevine trunk diseases, but control options remain limited. Good cultural practices, such as removing infected wood and protecting pruning wounds from infection, are still the best advice for the industry to reduce the spread of trunk diseases.

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