Research report

Anthracnose of grapevines, a review

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Summary

Grapevine anthracnose (also known as grapevine black spot) is found in most grape growing regions of the world. The causal agent is the ascomycete *Elsinoe ampelina*, although the sexual stage appears to be absent in many countries. The pathogen persists as sclerotia on diseased vine canes for 3–5 years and these germinate to produce conidia, the primary inoculum in most regions. Only actively growing tissues are infected and rain splashed conidia require a certain duration of surface wetness at a given temperature for infection. Rainfall is the main environmental determinant of disease severity. In Australia, the dithiocarbamate fungicides have provided effective control since the 1950s. Control of the disease is critical when frequent periods of rainfall occur in the first two months after budburst. In climates with relatively low spring rainfall the prospects for reactive spraying based on a disease forecasting system are good. The system would use weather data to identify infection periods and in conjunction with climatic data, cultivar susceptibility, vineyard observations and vine growth stage produce forecasts of the likely rate of disease increase, the risk of crop loss and the need for control treatments.

Distribution and importance

Anthracnose or black spot of grapevines, caused by *Elsinoe ampelina* Shear, probably originated in Europe (Shear 1929), but is now present in most grape growing regions of the world (Anon 1978). Countries where the disease has been reported include Australia (de Castella and Brittlebank 1918), New Zealand (Brook 1973), USA (Loucks 1936), India (Suhag and Grover 1972), South Africa (du Plessis 1940), China (Zhang and Huang 1990), Japan (Ozoe *et al.* 1972), Uruguay (Mazzei Patrone 1950), and Argentina (Anon 1964).

The incidence of the disease varies greatly and is mainly influenced by the amount of precipitation during the growing season. In the USA for example, anthracnose is of economic importance in the wet eastern states such as Florida, but does not occur on the drier west coast (Mirica 1988). On the other hand in subtropical India, the disease is widespread in most grape growing regions (Suhag and Grover 1977) while in South Africa it occurs in summer and winter rainfall districts (Boelema 1968). The historic importance of anthracnose is highlighted by the extreme measures used for its control e.g., swabbing vines with sulphuric acid (Taylor 1954). In the past, anthracnose has caused widespread damage to grapevines in many regions and is still an important disease in countries such as India (Suhag and Grover 1972).

In Australia, anthracnose was first recorded in New South Wales in the late 18th century (Gregory 1988) and has since been reported in all mainland States (de Castella and Brittlebank 1918, Gay Brereton and Hamblin 1922, Coombe 1953, Shea 1961, Harvey 1965). Outbreaks have caused serious crop loss in Sunraysia (Victoria and NSW), the Murrumbidgee Irrigation Area (NSW), and the Riverland (South Australia), as well as other regions (de Castella and Brittlebank 1918, Manuel 1928, Coombe 1953). In 1918, 100% crop loss was reported on some properties in Mildura and injury to canes resulted in poor crops in the following season (de Castella and Brittlebank 1918). In Victoria serious outbreaks occurred in 1916–17, the early 1930s, 1947, the early 1950s and 1975–76 (de Castella and Brittlebank 1918, Taylor 1954, Emmett unpublished data). In Australia control measures introduced during the 1950s have been highly successful in reducing the severity and occurrence of outbreaks. In commercial vineyards in Western Australia the disease has almost been eliminated (Harvey 1965).

Symptomology

Leaves

Leaf symptoms first appear 3–7 days after primary infection (Brook 1973). On young leaves, the first signs of disease are small, faint circular chlorotic lesions. These quickly turn brown, increase in size and develop a reddish margin. As the leaf matures, the centres of lesions dry out and become ash coloured. With time, lesions develop a characteristic "shot hole" appearance.

Shoots, petioles and tendrils

Lesions on the shoots, petioles and tendrils of grapevines are more appropriately classified as cankers. They begin as tiny dark brown indentations which darken, increase in size, and elongate. The margins of the cankers are raised and black, while their centres become sunken and ash coloured. Shoots, petioles, or tendrils can be girdled and killed by large cankers. Diseased canes are easily broken when they are wrapped onto trellis wires during pruning. On hardened vine tissues, solitary cankers appear swollen and knuckle-like while the surface of cankers that have coalesced is dark and rough like charred cork. During sporulation, the centre of cankers becomes pinkish-white.

Berries

Infected berries develop small round purple-black lesions that increase in size with berry growth. The centres of these lesions turn pinkish-white following sporulation. This symptom is called birds-eye spot in some countries. Severe crop loss can occur if bunch stems are girdled and killed.

Causal organism

References to anthracnose date back to the writings of Theophastrus and Pliny in ancient Rome, making it one of the oldest known diseases of plants (Vialla in du Plessis 1940). However, it wasn't until 1874 that the conidial stage of the fungus was described in detail as *Sphaceloma ampelinum* by de Bary (Shear 1929). Later, Gouirand and Bergeron, in 1897 illustrated the formation of conidia and sclerotia (Jenkins and Bitancourt 1943). During the late nineteenth century studies of the fungus resulted in a variety of synonymous names such as *Torula meyeni* Ber. and Trev., *Ramularia meyeni* Gar. and Catt., *Gloesoporium ampelophagum* (Pass.) Sacc., *Manginia ampelina* V. and P. (Shear 1929, Sivanesan and Critchett 1974, de Castella and Brittlebank 1918). Nevertheless, the conidial stage of the fungus is still widely recognized as *Sphaceloma ampelinum*. Some authors considered the fungus polymorphic (Vialla and Pacotet in Anderson 1956) although Shear (1929) was unable to confirm this.

The sexual stage of the fungus was de-

scribed as *Elsinoe ampelina* de Bary (Shear). It was considered to belong to the genus *Elsinoe* since it is very similar to the sexual stage of the fungus causing anthracnose of raspberries and blackberries which was described by Burkholder (Shear 1929). The sexual stage does not occur as regularly as the conidial stage and has not been found in countries such as Britain (Butler and Jones 1949), India (Suhag and Grover 1972), South Africa (du Plessis 1940) and Australia. The reason for this absence has not been identified, although it may be related to specific winter conditions (Shear 1929) or the lack of suitable mating types.

Sivaneson and Critchett (1974) and Mirica (1988) provide morphological descriptions of the fungus. Ascospores are hyaline, 3–septate and are 15–16 × 4–5 µm. Asci are eight spored usually globose or elliptical and distributed irregularly in the upper part of the ascoma. Ascomata are globose, separate or aggregated, pseudoparenchymatous, epidermal to subepidermal. The germination of ascospores produces lesions which give rise to the conidial stage. Conidia are hyaline , one-celled and 4–7.5 × 2–3.5 µm. They have mucilaginous walls which allows them to readily adhere to substrates, (e.g., leaves) in the presence of water (Mirica 1988). Conidia are produced most prolifically in acervuli on numerous short cylindrical conidiophores at the edge of lesions. In autumn, sclerotia form when production of acervuli ceases. Different strains of the fungus have been reported (Cheema *et al.* 1978, Kore and Gurme 1979, Suhag *et al.* 1982).

Epidemiology

Anthracnose is most destructive in humid hot climates, and is devastating in parts of India. Free water is required for most of the processes involved in disease development; without it primary sporulation, spore dispersal and infection does not occur. Although sporulation on active lesions does not require free water, it increases with humidity. Anthracnose can develop across a broad temperature range, but it is rarely a problem in regions where spring rainfall is relatively low (Mirica 1988).

Overwintering

The fungus persists on shoots, petioles and bunch stalks as sclerotia which are formed from late summer to winter as plant tissue hardens (du Plessis 1940). In Australia, formation of conidiophores ceases in January or February, and sclerotia are produced instead (de Castella and Brittlebank 1918). It is not known exactly what factors are responsible for this change although it is probably a combination of hot dry weather and the hardening of canes. Suhag and Grover 1982 found that the fungus remained viable on diseased canes, whether present on the vine or as prunings on the ground or 3–5 cm under the ground. Sclerotia on live canes had a higher percent viability than those on prunings on the ground, which in turn was higher than sclerotia on prunings 3–5 cm under the ground. Sclerotia on canes can be sources of inoculum for up to 2–5 years if conditions for primary infection fail to occur (Paufilova 1950, Brook 1992). The fungus may also overwinter on infected berries on the vineyard floor (Mirica 1988). Infected leaf debris is also a source of overwintering inoculum, however persistence of *E. ampelina* is greater on canes (Suhag and Grover 1972).

Primary sporulation

Overwintering sclerotia produce conidia that can cause primary infection as soon as susceptible host tissue is present. More rarely, ascospores may also cause primary infection (Anderson 1956). Thorough wetting of cankers is required in spring for sporulation, and as this can occur at low temperatures, the disease may spread in early spring even in cool climate vineyards (Anderson 1956). At temperatures above 2°C, 24 hours of wetness results in abundant sporulation, although 20°C is the optimum (Mirica 1988, du Plessis 1940). The conditions required for primary sporulation are unlikely to be critical since overwintering sclerotia from field vines produce conidia with little or no moist incubation (Brook 1973, Magarey unpublished data).

Spread

Spread of the disease is mainly by conidia which are dispersed in water during rainfall or overhead irrigations of 2 mm or more (Mirica 1988). As a result, the spread of the disease is primarily confined within a vine or to nearby vines. However, rain-splashed conidia may cause infections up to 7 m away from the source of inoculum (Brook 1973). The disease may also spread by conidia carried on the feet of birds or on agricultural machinery, or by diseased planting material. Disease spread may also occur from wind-borne ascospores where the ascomata of the fungus are present. However, little is known about the range of dispersal by this means.

Infection

Tissue wetness is essential for infection. Conidial germination and the infection of vine tissues occurs at temperatures ranging from 2°C to 40°C, the optimum temperature being 30–35°C, (Suhag and Grover 1977, Virk and Grover 1979, Mirica 1988). The influence of light and humidity on infection is unclear. The duration of tissue wetness required for infection varies with the temperature. For severe infection 1.5 hours of tissue wetness is required at 30°C, 3–4 hours at 21°C, 4–7 hours at 16.5°C, and 7–10 hours at 12°C (Brook 1973, Magarey unpublished data).

Conidia of *E. ampelina* will survive interruptions to leaf wetness of at least two hours (Magarey unpublished data). This is important when considering the combined effects of two or more wetness events. Ascospores germinate and infect vine tissue at temperatures ranging from 2–32°C (Mirica 1988). However little else is known about ascospore behaviour and periods of surface wetness required for infection.

Incubation

The incubation period for anthracnose varies with temperature. After infection, expression of symptoms requires about 13–14 days at 2°C, 7 days at 12°C, 5 days at 16°C, and as little as 3–4 days at temperatures above 21°C (Brook 1973, Suhag and Grover 1977, Mirica 1988).

Secondary sporulation

Disease spread can be rapid, because new conidia are produced within 1–5 days after lesions become visible (Brook 1973). Sporulation reaches a maximum when the diameter of the lesion is about 1 mm (Brook 1973). Leaves infected early in the season cease to produce conidia by summer and early autumn, but cankers on stems and recently infected leaves continue to sporulate (Brook 1973). In culture, the optimum temperature for growth and sporulation of the fungus is 30°C (Kore and Gurme 1978, Virk and Grover 1979). High humidity greatly increases sporu-lation. Kore and Gurme (1978) found that in culture, optimum sporulation occurs above 80% RH but limited sporulation still occurs at 30% RH. Light has little effect on sporulation. Kore and Gurme (1978) found conidial formation in culture occurred in continuous light and in diffused light but was greatest in continuous darkness. Nothing is known about the environmental conditions required for ascospore production.

Host susceptibility

Elsinoe ampelina is only known to infect *Vitis* species, although susceptibility to the fungus varies widely between cultivars. Species of *Vitis* native to the eastern United States of America are predominantly resistant to anthracnose and provide a source of resistant germplasm for grapevine breeders (Mortenson 1981). The *V. vinifera* cultivars Sultana, Waltham Cross, Ohanez are highly susceptible, while Shiraz and Cabernet Sauvignon are highly resistant (Jennings 1953, Dang and Daulta 1982, Yadav and Nirwan 1981, Mortenson 1981 and Goyal *et al.* 1971).

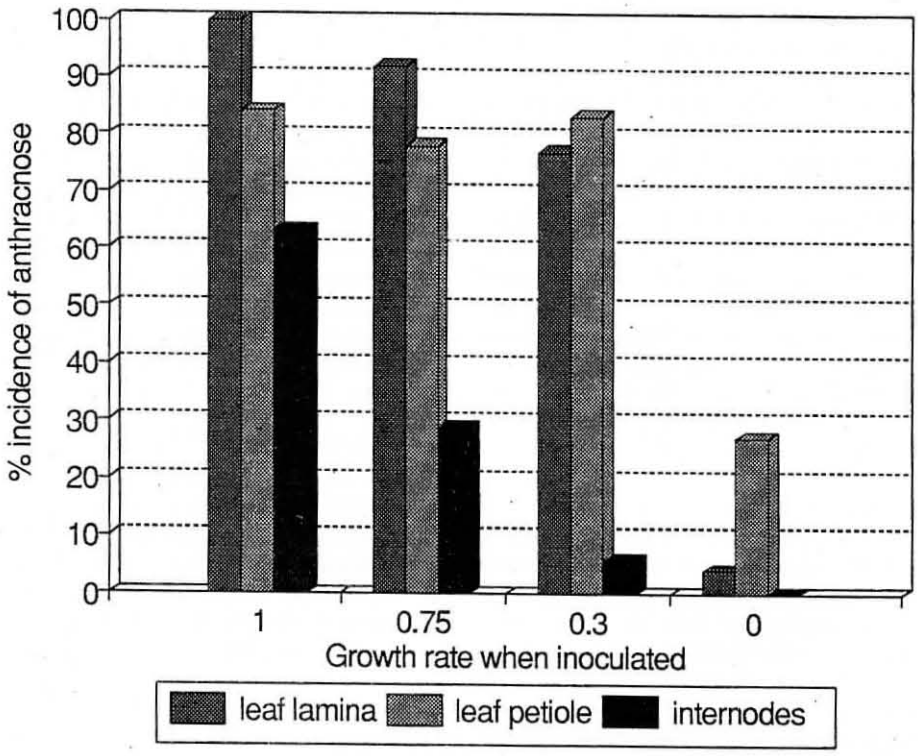

Figure 1. Differential infection of leaf lamina, leaf petiole and stem tissue on *Vitis vinifera* cv Palomino inoculated with *Elsinoe ampelina*, as a function of growth rate. Tissue is highly susceptible to infection when young and rapidly expanding. Susceptibility declines as growth rate decreases and is very low when tissue has fully expanded (after Brook 1973).

Effect of tissue age

The decline in tissue susceptibility with age is another important factor affecting the spread of the disease. Young grapevine tissues are more susceptible to infection while older tissues are highly resistant or immune (Brook 1973, Suhag and Grover 1977). When leaves and internodes are fully expanded, the rate of infection is very low (Figure 1). Also necrotic lesions reach a much greater size on young leaves than they do on older leaves (Suhag and Grover 1977). Similarly, Brook (1973) noted that berries become highly resistant to infection although not immune, when they reach a soluble solid content of 5–7%. The mechanism of resistance to anthracnose remains unclear. Various workers have examined the role of biochemical compounds in disease resistance. Kansal and Lal (1978) found increased levels of phospholipids in diseased leaf tissue, but reduced phospholipid content in the leaf overall. Daulta and Chauhan (1981) correlated disease intensity with concentration of reducing sugars and total phenols for nine cultivars of grapevine. There was only a small positive correlation for reducing sugars but a strong negative correlation for total phenols. Many anti-fungal compounds or phytoalexins are based on phenolic compounds (Salisbury and Ross 1985).

Effect of tissue type

Susceptibility to anthracnose differs considerably with tissue type. Suhag and Grover (1972) found that leaf material was more susceptible to infection than stems of the cultivar Thompson Seedless (syn. Sultana). There appears to be no consistent relationship in susceptibility of types of tissue. Datar and Ashaputre (1985) for example found that fruit of cultivars such as Country Bangalore, Calashil, and Husaini Black Kabuli was susceptible while leaves were moderately resistant. The reverse was found with the cultivars Rose of Peru, Waltham Cross, and Ruby Red.

Genetics of resistance

The inheritance of anthracnose resistance was investigated by Mortenson (1981). Mortenson proposed a trigenic hypothesis for resistance that involved two dominant genes for susceptibility and a single dominant gene for conditioning resistance with independent inheritance of each gene. This work was based on earlier studies by Fennell (1948) who stated that resistance was conditioned by multiple factors. Datar and Ashaputre (1985) also investigated the source of resistance and noted differential reactions to the leaf and fruit infection. They considered that resistance of leaves and berries was governed by two separate genes.

Control

Topography can affect the incidence and severity of anthracnose because of its effect on temperature, humidity and rainfall. Refatti (1949) noticed that vines grown on a hillside in the Pergine district of Italy escaped serious damage while other vines suffered between 10 and 100% crop loss. Moreover, low lying areas within a vineyard where dew persists or drainage is poor may be more prone to disease (de Castella and Brittlebank 1918). Cultivar selection is important in areas or climates prone to the disease.

Canopy management

Vine trellising systems can reduce the incidence of disease and may be a method for reducing anthracnose in some vineyards. Vine cultivar and trellis combinations that allow growing shoots to touch the ground and promote vegetative growth have higher disease incidences (Suhag and Daulta 1981).

Removal of canes with cankers will reduce the amount of overwintering inoculum present in the following season. However sufficient buds should be left for the next crop (Emmett 1976). Minimal pruning techniques can encourage the build up of inoculum of pathogens that persist in canes such as *Phomopsis viticola* Sacc. (Pscheidt and Pearson 1989).

Fungicidal control

Historically, the control of anthracnose has involved the treatment of vines during dormancy to reduce the overwintering inoculum. Dormant treatments were applied as either sprays or swabs, just prior to bud burst. Before this time the majority of overwintering sclerotia are small and are protected by layers of host cuticle (de Castella and Brittlebank 1918). Fungicides that have been used as dormant sprays in the past include copper sulphate, lime sulphur, and Bordeaux mixture (du Plessis 1940, Manuel 1928). Swabbing treatments involved painting or daubing the vines with solutions of sulphuric acid, Bordeaux mixture or iron sulphate (Gay Brereton and Hamblin 1922). The high labour costs involved, the hazardous nature of some of the compounds and the availability of safer more effective treatments has made these practices obsolete.

Since young vine tissue is more susceptible to infection than older vine tissue, most modern spray programs start at budburst and continue at 10–14 day intervals for about 4 weeks (Coombe 1955, Harvey 1965, Boelema 1968, Anon 1971, Emmett 1980). If weather conditions at flowering favour disease development, further sprays may be recommended (Harvey 1965, Boelema 1968).

During the growing season, Bordeaux mixture and other copper based fungicides have been used widely in control (Winkler *et al.* 1974, Gupta 1987, Shoi and Sridhar 1972). However, as copper based fungicides can be phytotoxic to young growth, they have been replaced by the more effective dithiocarbamate fungicides which are now used widely in Australia, USA and South Africa (Coombe 1953, Taylor 1954, Boelema 1968). The success of the dithiocarbamates is evident in Australia where routine early season sprays of ziram or thiram have been so effective that the disease is no longer a serious problem. Other multisite or protectant fungicides are also effective but many other fungicides used for other grape pathogens provide little or no control of anthracnose (Table 1).

Concluding remarks

Grapevine anthracnose is readily controlled by a range of protectant fungicides. However, with the increasing emphasis on minimal chemical use and residues in food and food products nowadays, it is important that the number of fungicide applications in control programs is kept to a minimum. Spraying for anthracnose control can be avoided in most years in districts with relatively low spring rainfall as free moisture is required for infection.

A management shift from routine spray to reactive spray programs requires accurate disease forecasting. Reliable forecasts are obtained when accurate epidemiological models are used in combination with site-specific weather data. The components of a forecast model could be as follows:

i. selection of a routine or reactive spray program based on climatic data and cultivar susceptibility;
ii. identification of infection periods using records of leaf wetness, temperature, humidity and rainfall;
iii. estimation of the incidence of disease from calculations of infection periods, vineyard observations and spray records;
iv. calculation of the rate of disease increase based on the estimated disease level and vine growth stage, including leaf and berry susceptibility;
v. assessment of spray need as "low" – where a fungicide for anthracnose control should be included in the tank mix during other spraying operations or as "high" where a spray for anthracnose control should be applied regardless of other spraying operations.

The absence of an effective post-infection fungicide limits the use of a reactive strategy to vineyards with low disease pressure. The prospects of a reliable forecast system are high because the epidemiology of *E. ampelina* is well understood. The system would need accurate vineyard observations and weather data to produce reliable forecasts. The integration of epidemiological models for a range of diseases and pests into a computer driven decision aid device for grapegrowers has the most potential for providing low input control strategies for anthracnose.

An aspect of the biology of the disease requiring further study is the reason for the absence of the sexual stage from many parts of the world. This is important since ascospore production could have considerable influence on the spread of disease and the potential for the pathogen to develop resistance to fungicides.

Table 1. Summary of fungicide efficacy against anthracnose

Fungicide or Fungicide Class	Activity	Reference
dithiocarbamates	Excellent to Good	Emmett *et al.* 1981
captan/captafol	Excellent to Good	Emmett *et al.* 1981
dichlofluanid	Excellent	Magarey and Emmett 1992
fluazinam	Excellent	Magarey and Emmett 1992
chlorothalonil	Good	Hopkins 1974, Magarey *et al.* 1977
DMI*	Fair[P]	Magarey and Emmett 1992b, Magarey unpublished data
copper	Fair to Poor	Coombe 1953, Emmett *et al.* 1981
lime sulphur	Fair to Poor	Coombe 1953
benzimidazoles	Fair to Poor	Coffey *et al.* 1991
sulphur	Poor	Coffey *et al.* 1991
dicarboximides	Poor	Coffey *et al.* 1991, Magarey and Emmett 1991
metalaxyl	Poor	Moore and Schroeder 1983

Excellent - exhibits excellent control
Good - exhibits good control
Fair - exhibits some control
Poor - has little or no controlling effect
[P] - problem associated with phytotoxicity during early season use particularly on sensitive cultivars e.g., Sultana
*DMI - Demethylation inhibiting fungicide.

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