An Overview of the Biology, Epidemiology and Control of *Uncinula necator* (Powdery Mildew) on Grapevine, with Reference to South Africa

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Grape powdery mildew, caused by *Uncinula necator*, is the most widespread and destructive disease of grapevine. The disease can be found in most grape-growing areas of the world, including the tropics. In South Africa, grape powdery mildew was first reported in 1880, and since then has become the most important disease of grapevine. The disease can effect all phases of plant growth, and without necessarily causing obvious symptoms, may have a harmful effect on the vine and its products. The pathogen follows a specific pattern in each part of the world to create an epidemic. This pattern is determined by biological characteristics of the organism, climatic factors, cultivation practices and cultivar choices. Increased world-wide emphasis on the production of disease-free grapes with minimal fungicide input provides a sound reason for exploring more efficient disease management strategies through a better understanding of *U. necator* epidemiology and population genetics. Knowledge of these aspects is available for various parts of the world, but little is known about its relevance to South African vineyards. In this article a South African perspective of the pathogen and its control is outlined, based on recent local findings, and considered in the light of knowledge available in other parts of the world.

INTRODUCTION

Grape powdery mildew, caused by *Uncinula necator* (Schw.) Burr., is the most widespread and destructive disease of grapevine (Pearson & Gadoury, 1992). The disease was first described (Schweinitz, 1834) in 1834 in North America. In 1845 an English gardener called Tucker observed grape powdery mildew for the first time in Europe and the botanist Berkeley described the fungus under the name of *Oidium tuckeri* (Bulit & Lafon, 1978). Today powdery mildew can be found in most grape-growing areas of the world, including the tropics (Kapoor, 1967; Bulit & Lafon, 1978). In South Africa grape powdery mildew was first reported in 1880 by Jacob Cloete from Constantia near Cape Town, although it is believed to have been present since 1860 (Du Plessis, 1948). Since then powdery mildew has become the most important disease of grapevine in South Africa (Halleen, 1999).

ECONOMIC IMPORTANCE

The disease can affect all phases of plant growth. The mass of canes pruned from infected vines is lower than that from healthy vines, indicating lower yield (Pool *et al.*, 1984; Wicks *et al.*, 1988; Reuveni & Reuveni, 1995a). Infection reduces the winter hardiness of canes, expressed as a reduction in bud survival (Pool *et al.*, 1984). Levels of photosynthesis and transpiration of infected leaves are reduced (Pool *et al.*, 1984; Shtienberg, 1992). Due to decreased photosynthetic area the chlorophyll content in diseased leaves is reduced. This leads to low activity of chloroplasts and low efficiency of carbon dioxide fixation (Dhillon *et al.*, 1992). Fruit infection lowers wine quality, both as a result of increased acid concentration and as a direct result of the fungus itself producing off-flavours (Ough & Berg, 1979; Pool *et al.*, 1984). Wines made from infected grapes have a lower colour intensity associated with a higher tonality index and a lower concentration of total anthocyanins. Since colour characteristics are correlated with the quality of red wines, the lower concentration of anthocyanins and the lower colour intensity of the wines produced from infected grapes will have a negative effect on vinification (Amati *et al.*, 1996). Wineries in New York State therefore specify that the fruit may not contain more than 3% (by weight) powdery mildew infected berries (Pool *et al.*, 1984). In South Africa it is specified that bunches may not contain more than 5% infected berries (J.H.S. Ferreira, personal communication). Infection of immature fruit causes tissue scarring and berry splitting (Chellemi & Marois, 1992a). Infected berries may also crack and provide entry sites for pathogens like *Botrytis cinerea* and organisms associated with sour-rot (Pearson & Gadoury, 1992). Since such fruit is unmarketable, the disease is of great economic importance for the South African industry. Capespan (Capespan, personal communication) stipulates that consignments delivered for the table-grape export market must have less than 50% infected bunches per carton with no more than three infected berries per infected bunch.

In 1998 the annual cost of chemical control of powdery mildew in South Africa amounted to approximately R30 million, which accounted for 50% of the total fungicide cost spent on vineyards (J.H.S Ferreira, personal communication).

SYMPTOMS

Powdery mildew may be observed on all the green parts of the vine: leaves, branches, inflorescences, rachises, pedicels and berries (Bulit & Lafon, 1978). Mildew colonies are usually found on either the lower surface of exposed leaves or on both sides of well-shaded leaves. The fungus forms a white to ash-grey, web-like mat of mycelial strands over the infected tissue. Haustoria grow from the mycelium into the epidermal cells from which they

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derive nutriment. Chains of conidia borne on short conidiophores arise from the mycelium, giving a dusty or powdery appearance to the surface (Sall & Teviotdale, 1982).

Infected berries develop web-like blemishes and are eventually covered with powdery growth. Severely infected berries are scarred, distorted and often split. The maturity of severely infected bunches is retarded and dark fruit varieties may colour unevenly (Emmett *et al.*, 1992). Berries are susceptible to infection until sugar content reaches about 8%, although established infections continue to produce spores until the berries contain 15% sugar (Delp, 1954). However, recent studies have shown that substantial ontogenic resistance is expressed in fruit long before sugar accumulation begins. Fruit of *Vitis labruscana* 'Concord' and many cultivars of *V. vinifera* become resistant to powdery mildew within 2-4 weeks after fruit set (Gadoury *et al.*, 1998). Petioles and cluster stems are susceptible to infection throughout the growing season, and once infected they become brittle and prone to breakage as the season progresses, with serious implications for the storage life of table-grapes (Pearson & Gadoury, 1992). This is also of great concern to the South African table-grape industry, where producers are severely penalised when cluster stems of export table-grapes are infected (Capespan, personal communication).

When green shoots are infected, the tissue appears dark-brown to black in feathery patches, which later become reddish-brown on the surface of dormant canes. Severely infected canes mature irregularly and die back from the tips.

CAUSAL ORGANISM

Powdery mildews (Erysiphaceae) are a clearly defined family of obligate parasitic ascomycetous fungi (Yarwood, 1957). *Uncinula necator* is a parasite of genera of the Vitaceae. The fungus is heterothallic and populations consist of two mutually exclusive mating types (Gadoury & Pearson, 1991; Evans *et al.*, 1997; Délye & Corio-Costet, 1998). The superficial but semi-persistent septate, hyaline hyphae (4-5 µm in diameter), develops characteristic multilobed appressoria, from which penetration pegs are formed (Pearson & Gärtel, 1985). After penetration of the cuticle and cell wall, a globose haustorium is formed within the epidermal cell. Conidiophores form perpendicularly on the prostrate hyphae at frequent intervals. The multiseptate conidiophore is attached to the mycelial hypha by a cylindrical foot-cell (25-40 µm), which is flexuous at the base. Conidia are hyaline, contain inconspicuous fibrosin bodies, are cylindro-ovoid in shape, are formed singly but accumulate in chains and measure 27-47 x 14-21 µm. The oldest conidium is at the distal end of the chain (Boesewinkel, 1977).

Initiation of cleistothecia occurs within 48 h of hyphal contact between compatible isolates (Gadoury & Pearson, 1988). At first cleistothecia appear as hyaline spheres approximately 10-20 µm in diameter. They consists of undifferentiated spherical groups of pseudoparenchymatous cells attached to the mildew colony by two parent hyphae. As the ascocarp increases in diameter, the hyaline cleistothecia becomes yellow due to the intracellular accumulation of lipids. Anchorage hyphae are produced by the cells of the outer ascocarp wall as the diameter of the ascocarp approaches 30 µm. These anchorage hyphae become intertwined in the surrounding mildew colony, but they do not anastomose or form appressoria. Anchorage hyphae are distinct from appendages, which appear much later when the ascocarp reaches about 75 µm in diameter. The equatorial band of 8-31 appendages is more robust and directed upward and away from the mildew colony (Gadoury & Pearson, 1988). Appendages are 1-6 times as long as the diameter of the ascocarp, septate, thin walled and brown towards the base with uncinate hooked tips, from which the genus derived its name (Kapoor, 1967). Asci (4-6, rarely 6-9, wedge- or pear-shaped, 50-60 x 25-40 µm) are formed once the ascocarp reaches 75 µm in diameter, and at 20°C, ascospores (4-7, ovate to ellipsoid, 15-25 x 10-14 µm [Kapoor, 1967]) are formed within 7 days of the first appearance of asci. Mature asci are distributed in the centrum similar to the arrangement of carpels in an orange. As the ascocarp approaches a diameter of 100 µm, the outer ascocarp wall darkens. The final event in development is the necrosis of the anchorage and parental hyphal connections to the mildew colony. This is followed by a rapid loss of turgor and the formation of a basal concavity in the ascocarp wall; therefore cleistothecia are concavo-convex when mature. The wall of a mature ascocarp consists of an outer layer (1-2 cells thick) of thickened, dark coloured cells devoid of cytoplasm. Concavo-convex ascocarps are now ready to be dispersed to bark or soil by rain (Gadoury & Pearson, 1988). Mature cleistothecia measure between 90 and 300 µm in diameter. Most viable cleistothecia are between 106 and 180 µm in diameter (Cortesi *et al.*, 1995). One study with tissue culture plants and detached leaves showed that cleistothecia are produced within 25-36 days of inoculation (Gadoury & Pearson, 1988). In another study with tissue culture plants, cleistothecia were produced within 45-60 days after inoculation and viable ascospores after 80 days (Gadoury & Pearson, 1991). Ascospores germinate with a short, single germ tube and form appressoria within 12 h. This is followed by progressive growth, branching of the primary hyphae and formation of lobate appressoria at irregular intervals along the hyphal branches. Conidiophores form and sporulation may occur within 6 days (Pearson & Gadoury, 1987).

DISEASE CYCLE

The two principal sources of primary inoculum for the initiation of a powdery mildew epidemic in grape are mycelium within dormant buds, and ascospores borne in cleistothecia (Bulit & Lafon, 1978). In New York (Pearson & Gadoury, 1987) and some Italian vineyards (Cortesi *et al.*, 1997), cleistothecia are the only source of primary inoculum. In California (Gubler *et al.*, 1988), Germany (Hill *et al.*, 1995), Australia (Wicks *et al.*, 1985; Magarey *et al.*, 1993a), France (Bulit & Lafon, 1978), Romania, Iran (Banihashemi & Parvin, 1995), Peru (Pearson & Gadoury, 1992), Russia and other Italian vineyards (Cortesi *et al.*, 1997) cleistothecia are not the main source of primary inoculum. There the fungus also overwinters as hyphae inside buds of the grapevine, where it stays in a dormant state until the following season (Sall & Wrysinski, 1982). Although powdery mildew has been present in the Western Cape province since 1880, cleistothecia were first observed during 1996 in three vineyards in the main grape-growing areas of Stellenbosch (Halleen & Holz, 2000a). Previously Van der Spuy and Matthee (1977) demonstrated with potted plants kept in isolation that the fungus overwinters locally in the buds of grapevines as dormant mycelia or conidia.

Cleistothecia can form on all infected tissues (Gadoury & Pearson, 1990a). In New York State cleistothecia are produced from late July (pre-veraison) to frost (Pearson & Gadoury, 1987). In Germany the first yellow fruiting bodies can be detected as early as mid-July (early summer) to mid-August with mass formation at the beginning of September (late summer). Ascocarp formation stops at the beginning of October. The frequency of cleistothecial formation seems to depend mainly on disease level at véraison (Hill *et al.*, 1995). The formation of cleistothecia in vineyards is dependent on the amount of disease, not host or environmental factors as previously reported. The more disease, the earlier one can observe cleistothecia as colonies of compatible mating types merge (Pearson, 1990; Cortesi *et al.*, 1995). In Australia cleistothecia are formed during late April and early May (late summer to autumn) (Wicks *et al.*, 1985). In South Africa cleistothecia were also observed during April to May on severely infected leaves (Halleen & Holz, 2000a).

Cleistothecia are washed by late summer and autumn rain to the bark of the vine where they overwinter (Pearson & Gadoury, 1987; Cortesi *et al.*, 1995; Cortesi *et al.*, 1997). Although disease incidence and severity may determine the potential population available for dispersal, rain events determine the actual efficiency of transfer from infected organs to the bark of the vine. Insufficient autumn rains may affect the complete transfer of physiologically mature ascocarps from the leaves to the bark, resulting in a residue of viable cleistothecia that survive on fallen leaves (Cortesi *et al.*, 1997). Studies have shown that cleistothecia were dispersed from leaves to the bark of grapevines in the Stellenbosch area, although in very small numbers (Halleen & Holz, 2000a). In New York rain-dispersed ascocarps accumulate on bark during a 10-week period, where they are retained through subsequent rain events between leaf abscission and budbreak the following spring (Cortesi *et al.*, 1995). Appendages of the ascocarp, which are upright and bristle-like when first formed, are closely appressed to the bark and anchor the cleistothecium to the bark (Gadoury & Pearson, 1988). In New York the density of these ascocarp populations ranges from ± 9,000 to 290,000 cleistothecia per kg bark on unsprayed vines (Gadoury & Pearson, 1989). Most of the cleistothecia are found on the bark from cordons of grapevines with successively lower densities occurring on the bark of the upper and lower trunks. Density and viability of populations on bark do not change substantially during overwintering, despite frequent and heavy rains (Cortesi *et al.*, 1995). Cleistothecia on bark can be found singly or in clumps (Pearson & Gadoury, 1987).

In Australia and Italy cleistothecia may also overwinter on leaf litter under vines. The dense aggregations of cleistothecia on leaf litter make them a potentially important source of inoculum, despite the relatively low percentage of viable ascocarps (Cortesi *et al.*, 1997; Magarey *et al.*, 1997). In New York, however, only dead cleistothecia were recovered from the upper 1 cm of vineyard soils in spring. Earthworms are responsible for leaf burial and leaf litter decomposition (Gadoury & Pearson, 1988). Most (79-97%) of the cleistothecia borne on leaves, canes and cluster stems die during winter and spring without releasing ascospores, but 45-90% of the cleistothecia found in bark crevices are viable (Pearson & Gadoury, 1987; Gadoury & Pearson, 1988; Cortesi *et al.*, 1995). The survival of cleistothecia on leaf litter has not been investigated in South African vineyards.

Cleistothecia dehisce circumscissilely at the junction of the concave and convex surfaces of the outer ascocarp wall, near the site of a thin zone in the ascocarp wall, immediately beneath the equatorially attached appendages. Following ascocarp dehiscence, ascospores are discharged through a slitlike rupture of the apex of the ascus (Gadoury & Pearson, 1990a). In New York ascospores are released in spring between budbreak and bloom. The first-formed leaves (basal leaves) of shoots growing near bark are infected first, presumably due to their proximity to the overwintered cleistothecia. These ascospores germinate and infect all green tissue, resulting in colonies that produce conidia for secondary infections (Gadoury & Pearson, 1990a). Where cleistothecia are the principal source of primary inoculum, the pattern of disease development will often occur as a random distribution throughout the vineyard (Pearson & Gadoury, 1987).

After primary infections colonies of the powdery mildew fungus spread over the surface of the vine tissues. The fungus enters the tissue, absorbs nutrients and slowly kills surface cells. After 5-32 days (usually 5-12 days), depending on temperature, the fungus sporulates. These spores are spread by wind and may cause secondary infection within 24 h if conditions are favourable. The fungus can go through at least three cycles of infection before leaf symptoms are first detected in vineyards (Emmett *et al.*, 1992).

Perennating bud infections occur early in the season rather than later in autumn (Pearson & Gärtel, 1985) and infected buds open slightly later than uninfected ones in spring (Sall & Wrysinski, 1982). Shortly after budbreak, the fungus is reactivated in infected buds and developing shoots, which are called "flag shoots", become covered with white mycelium. Conidia produced on these flag shoots infect neighbouring shoots and vines (Emmett *et al.*, 1992). Flag shoots are most easily detected 3-8 weeks after budbreak, before the canopy closes (Magarey *et al.*, 1994). The incidence of flag shoots in vineyards is usually very low, ranging from 0 – 0.2% (Emmett *et al.*, 1990). If flag shoots are present they will cause disease foci centred on the location of the flag shoot. If flag shoots are numerous, the pattern of disease development might be a more random distribution of disease throughout the vineyard (Pearson & Gadoury, 1987). Most of the flag shoots originate from buds borne on spurs. Spurs are short branches (2-3 buds long) of one year's growth retained after pruning (Sall & Wrysinski, 1982). Most flag shoots also appear on the same vines year after year (Bleyer *et al.*, 1998). Flag shoots appear to be most prevalent on vines of more susceptible varieties (cultivars Carignane and Thompson Seedless) that were heavily infected early in the previous season (Emmett *et al.*, 1990). Pruning methods and grape cultivar seem to be two important factors determining the occurrence of typical flag shoot symptoms. The observation (Halleen & Holz, 2000a) that they occur in relative abundance on Carignane vines at node position two is of great importance, since previous studies concluded that flag shoots are seldom observed at nodes one or two, but most frequently at node positions 3-5 (Pearson & Gärtel, 1985). Most wine-grape cultivars are pruned to two bud spurs in South Africa, while table-grapes cultivars are pruned to two bud spurs, 4-6 buds, 6-8 buds or 12-16 buds (Anonymous, 1998). The fact that heavy powdery mildew infections occur in most vineyards without the presence of typical flag shoot symptoms and cleistothecia

further highlights this point. In the absence of cleistothecia, cultivars less susceptible to bud infection might delay the onset of powdery mildew infections early in the season. It is therefore important to investigate the role of cultivar and pruning method on the occurrence of bud infections and typical flag shoot symptoms, since the typical symptoms might not be so evident in less susceptible cultivars.

EPIDEMIOLOGY

Cleistothecium formation

Early studies were confusing and contradictory regarding the effects of host and environmental factors on initiation, development and survival of cleistothecia. Host factors included host resistance and host nutrition, where both poor nutrition and vigorous growth were implicated. Environmental factors that supposedly triggered the initiation of cleistothecia included drought, cold, heat or an environment generally unfavourable for the parasite. Cleistothecia have also been reported to form primarily, or exclusively, on senescent foliage and on mid-cane leaves (Gadoury & Pearson, 1988; Chellemi & Marois, 1992b; Hill *et al.*, 1995). Several studies in the past concluded that ascospores did not germinate or infect grapevines. In moderate climates the pathogen can overwinter successfully in the vegetative state as mycelium in dormant infected buds, and cleistothecia are not necessary to ensure perpetuation of the disease. All these factors contributed to the relegation of cleistothecia as unimportant in the disease cycle (Gadoury & Pearson, 1990b).

It was only recently shown that cleistothecia are the source of primary inoculum in New York (Pearson & Gadoury, 1987). *In vitro* studies proved that initiation of cleistothecia only requires hyphal contact between compatible isolates. Environmental factors such as temperature, day length, humidity, leaf age and host resistance did not affect cleistothecium initiation and, once initiated, only temperature and host resistance affected their growth. No growth occurred at 4°C or 32°C. At 10°C cleistothecia increased in diameter, but will not advance beyond the stage of early ascus formation. On detached leaves or tissue culture plants, mature cleistothecia can form within 25-36 days of inoculation, and incubation at 16-25°C. Cleistothecia grow and mature more rapidly on susceptible cultivars than on resistant cultivars (Gadoury & Pearson, 1988).

In New York (Gadoury & Pearson,1988; Cortesi *et al.*, 1995), but not in Italy and Australia (Cortesi *et al.*, 1997), sufficient autumn rains are necessary to disperse physiologically mature cleistiothecia from leaves to the bark where they overwinter. Retention of cleistothecia on the bark is therefore a prerequisite for survival in New York (Cortesi *et al.*, 1995). In this region ascospores are released in spring between grapevine budbreak and bloom (Gadoury & Pearson, 1990a). Ascospores are released only during or immediately following rains or overhead irrigation (Emmett *et al.*, 1992) of more than 2.5 mm (Gadoury & Pearson, 1990a). Fog and dew (February-June) are also associated with waves of ascospore release in California (Thomas *et al.*, 1991). Rainfall is therefore a critical event in the release of ascospores and the initiation of powdery mildew epidemics in areas where cleistothecia are sources of primary inoculum. Ascospores germinate in water as well as at relative humidities as low as 54% (Pearson, 1990). The optimal temperature for infection by ascospores is between 20°C and 25°C. Infection is significantly reduced at 15°C or below. No infection occurs at or below 5°C, nor does it occur at or above 31°C (Gadoury & Pearson, 1990b; Pearson, 1990; Jailloux *et al.*, 1998).

In the Western Cape province environmental conditions are favourable for the production of cleistothecia during late summer and autumn, dispersal of cleistothecia during autumn, and release of ascospores and initiation of powdery mildew epidemics in spring (Halleen & Holz, 2000a).

In addition to providing primary inoculum, the genetic variability made possible by the sexual stage may be exhibited as variation in the population, such as increased virulence on currently resistant cultivars or development of tolerance to highly selective fungicides, such as fungicides that inhibit the C-14 demethylation of sterols (DMIs) (Gubler *et al.*, 1996; Steva & Cazenave, 1996).

Wind

During the growing period the fungus spreads by asexual conidia (Fessler & Kassemeyer, 1995). Wind speed as low as 2.3 m/s instantaneously triggers dispersal of conidia from leaves, although greater wind speed will result in more conidia being dispersed. The fraction of conidia dispersed at a given wind speed increases with colony age from 12 to 24 days, while conidia from a 27-day-old colony are less easily dispersed (Willocquet *et al.*, 1998). Conidial dispersal also occurs at the onset of rainfall due to leaf movement. Other cropping practices causing leaf shaking, such as pruning or high pressure sprays (wind speeds up to 30 m/s) applied against other pests or diseases may enhance spore dispersal and powdery mildew epidemics (Willocquet & Clerjeau, 1998). Relative humidity has no effect on dispersal of conidia at different wind speeds (Willocquet *et al.*, 1998).

Temperature

Temperatures of 20-27°C (optimum 24-25°C) are favourable for conidial germination and disease development (Fessler & Kassemeyer, 1995; Willocquet *et al.*, 1996), although germination can occur between 6 to 33°C (Delp, 1954). Temperatures above 32°C (Fessler & Kassemeyer, 1995), or 35°C (Delp, 1954), inhibit germination of conidia and temperatures above 40°C will kill conidia (Delp, 1954). Germination is completed within 30 h after inoculation at temperatures between 12 and 30°C (Delp, 1954). The fungus can adjust to temperature and a shift in germination rates occurs according to the temperature during conidial development. For example, if conidia develop at low temperatures, their germination rate at low temperatures is higher than that of conidia which develop at high temperatures. If conidia develop at high temperatures, their germination is reduced at low temperatures. At very high temperatures germination occurs, but at a low rate (Fessler & Kassemeyer, 1995).

Under favourable conditions established colonies begin sporulating after 5 days. At 22°C and 26°C sporulation will stop after 35 and 25 days respectively (Chellemi & Marois, 1991a). *Uncinula necator* has tremendous potential for rapid reproduction. The net reproduction rate per individual conidium ranges between 2272 and 1300 conidia per generation at 22°C and 26°C respectively. Mean generation times range from ±23 days at 19°C to ±14 days at 30°C (Chellemi & Marois, 1992b).

Free water

The conidial stage of *U. necator* is generally considered to be a xerophytic plant pathogen (Gadoury & Pearson, 1990b) and rain is generally considered to be deleterious to development of epidemics (Gadoury & Pearson, 1990a). Conidia are adapted to germinate under dry conditions by their hygroscopy, which seems to be due to a high amount of soluble substances contained in the conidial vacuoles. These are unusually large compared with those in saprophytic fungi (Blaich *et al.*, 1989).

Free water from rain, dew or irrigation can cause poor and abnormal germination of conidia as well as bursting of conidia (Blaich *et al.*, 1989). Free water can also wash conidia from the surface of vines and disrupt mycelium. However, fungal growth usually continues because the fungus is slightly water repellent and mostly sheltered within the vine canopy (Emmett *et al.*, 1992).

Application of water by gently misting leaves to runoff in a glasshouse experiment reduced sporulation. However, the magnitude of the reduction depended on temperature. A large reduction in sporulation occurred when water was applied to colonies growing at 19°C and 30°C, but not those growing at 22°C and 26°C. This may explain why rainfall seems to enhance mildew development during some years but suppresses it during others (Chellemi & Marois, 1991b). In vineyards water lowers the temperature under the canopy and may even enhance the rate of development of surviving infections (Sall & Teviotdale, 1982).

Relative humidity

Normal development of the disease can occur over the full range of relative humidities. Conidia are relatively insensitive to vapour pressure deficit (VPD) at temperatures below 25°C (Delp, 1954). At humidities of 40-100% the level of germination remains more or less a constant average value of 65% (Bulit & Lafon, 1978). Humidity seems to have a greater effect on sporulation than on germination (Pearson & Gadoury, 1992).

Leaf maturity

Leaf maturity does not greatly influence the germination of conidia and formation of appressoria. However, growth and sporulation are adversely affected by advancing leaf maturity (Singh & Munshi, 1993). *Uncinula necator* develops best on young grape leaves and will not usually infect leaves more than 2 months old (Doster & Schnathorst, 1985).

Cultivar

There seems to be no difference in conidial germination on susceptible and more resistant cultivars. However, germinated conidia frequently produce less hyphae on leaves of more resistant cultivars (Singh & Munshi, 1993).

Light

Shaded conditions are more favourable to conidial germination and mycelial growth than sunny conditions (Bulit & Lafon, 1978). Radiation, particularly ultraviolet B, reduces conidial germination and mycelial growth, especially at high temperatures (Willocquet *et al.*, 1996).

Fungicides

Application of fungicides to established colonies significantly reduces sporulation, but does not eradicate colonies. However, temperature also seems to have an effect on the efficacy of fungicides on sporulation. At 26°C colonies treated with triadimefon permitted some sporulation 3 and 8 days after application (Chellemi & Marois, 1991b). DMI fungicides generally have no effect on the germination of conidia (Steva, 1992).

DISEASE MANAGEMENT

Disease resistance

There are great differences in susceptibility within *Vitis* species to powdery mildew. For example, *V. vinifera* is considered highly susceptible, whereas the American species, such as *V. labrusca, V. riparia* and *V. rupestris,* are much less susceptible. However, cultivars within a species vary considerably in susceptibility (Pearson & Gadoury, 1992). The presence or absence of pathogenic specialisation of isolates of *U. necator* from grape is also poorly understood. There is therefore a great need for studies on disease resistance in powdery mildew of grapevine.

Fungicides

Sulphur was the first effective fungicide used for the control of powdery mildew on grapes (Bulit & Lafon, 1978). It is applied as a dust (sublimed sulphur, triturated sulphur) or as a wettable powder (wettable micronised sulphur) and exerts its effect through vapour action. It is a protectant multisite fungicide which acts as a general inhibitor of many fungal enzymes (Wicks *et al.*, 1997). Although sulphur is still used worldwide, it cannot be used throughout the season due to unfavourable weather conditions, such as wind and rain, which make application impossible. A 58% reduction in sulphur residues can occur within 3 days due to natural weathering. Sulphur residues return to near pretreatment levels within 6 to 9 days (Leavitt & Martin-Duval, 1997; Leavitt & Martin-Duval, 1998). Sulphur exhibits good activity from 18°C upwards with an optimum at 25-30°C. High temperatures can result in the burning of leaves. Therefore applications at 32-35°C are not recommended (Bulit & Lafon, 1978). There is, furthermore, a worldwide tend to move away from sulphur applications because of the danger of environmental pollution and allergic reactions to sulphur.

Studies with aqueous solutions of lime sulphur in New York showed that overwintering cleistothecia could be killed on the bark during spring, which led to the delay in the development of epidemics. Lime sulphur at a concentration of 120 mL/L applied as over-the-trellis sprays at 2 800L/ha (33 L/ha of lime sulphur) to dormant grapevines gave good results, but the high rates of application required and the volume of water delivered weigh against the commercial use of these eradicant treatments (Gadoury *et al.*, 1994).

Dinocap is another non-systemic fungicide registered for powdery mildew control in South Africa (Nel *et al.*, 1999). Although Dinocap is active at a lower temperature than sulphur, it also presents a risk of phytotoxicity at temperatures above 34-35°C (Bulit & Lafon, 1978).

The development of systemic fungicides, which maintain activity over a wide temperature range and exhibit less phytoxicity than sulphur, has alleviated the problems encountered with sulphur and dinocap. Consequently benomyl (a benzimidazole) became a very popular fungicide during the 1970s. Unfortunately three to four years of intensive use led to the selection of resistant strains of the pathogen and benomyl resistance became widespread in commercial vineyards (Pearson & Taschenberg, 1980). The benzimidazoles act by inhibition of tubulin biosynthesis, a

process under simple genetic control with frequent mutation to resistance (Russel, 1995). Several point mutations within the tubulin gene are responsible for monogenic resistance (Köller, 1996). Pathogen populations show a discontinuous distribution, therefore each population consists of at least two distinct subpopulations, one sensitive and one resistant. High-level resistance may be obtained in one step. This is called qualitative, discrete or disruptive resistance (Brent, 1995). Resistant strains tend to be as fit as sensitive strains and will survive in the population even after fungicide use is stopped (Russel, 1995; Ypema *et al.*, 1997).

Fungicides with a different chemical structure and mode of action were developed. At present the sterol demethylation inhibiting fungicides (DMIs), which include the triazoles, imidazoles, pyrimidines and pyridines, are very important in the control of grapevine powdery mildew worldwide (Scheinpflug & Kuck, 1987). The triazoles are by far the biggest group and include triadimefon, triadimenol, tebuconazole, myclobutanil, flusilazole, hexaconazole, cyproconazole and penconazole. The pyrimidine nuarimol and the pyridine pyrifenox are also regularly used on grapevines (Scheinpflug & Kuck, 1987). These fungicides are all effective as sterol biosynthesis inhibitors and have a common site of action within the fungal sterol biosynthesis pathway. It is an inhibition of demethylation at position 14 of lanosterol or 24-methylene dihydrolanosterol (eburicol), which are precursors of sterols in fungi (Scheinpflug, 1988). Other main metabolic routes such as respiration and protein or nucleic acid synthesis are not affected at inhibitor (DMI) concentrations sufficient to block sterol biosynthesis (Köller, 1988). Ergosterol is the major sterol in many fungi and is a basic component of fungal membrane synthesis (Scheinpflug, 1988). In the case of *U. necator*, ergosta-5,24 (24')-dien-3β-ol is the final sterol. It is a 4-desmethylsterol and represents 85-95% of total sterols (Debieu *et al.*, 1995). Lanosterol is formed by cyclisation of 2,3-epoxysqualene which originates from mevalonic acid via the isoprenoid pathway. Lanosterol must undergo considerable modifications before the final end products are formed. The first modification step in the fungus is a methylation in the C-24 position, followed by oxidative removal of the methyl groups in the C-14 and C-4 positions (Köller, 1988).

The C-14 demethylation starts with a hydroxylation of the C-14 methyl group, a step mediated by a cytochrome P-450 monooxygenase. Two subsequent oxidation steps and the release of formic acid lead to an intermediate characterised by a double bond in the C-14 position. The reduction of this double bond is the final step of the demethylation sequence. Late steps in sterol synthesis consist of the rearrangement of several double bonds and lead to the end product, ergosterol (Köller, 1988).

No oxygenated intermediates accumulate in DMI-treated fungal cells, indicating that the first hydroxylation step, catalysed by the cytochrome P-450 enzyme 14α-demethylase (P-450$_{14DM}$), is most effectively blocked by DMIs. Pyridines, pyrimidines and azoles bind to a site of the cytochrome P-450 normally occupied by the enzyme substrate lanosterol or 24-methylenedihydrolanosterol. Therefore, the essential nitrogen atom of the inhibitor is bound to the sixth coordination site of the cytochrome heme iron and consequently blocks the binding of an oxygen molecule that normally would be activated and transferred to the C-32 carbon of the sterol precursor (Köller, 1988).

A study with triadimenol-sensitive and -resistant strains showed that they contained similar sterol compositions, although triadimenol treatment led to accumulation of 14α methylsterols, such as eburicol and obtusifoliol, and a decrease of the final 4-desmethylsterol. This suggests that sterol C-14 demethylase is the target for triadimenol in sterol biosynthesis in *U. necator* (Debieu *et al.*, 1995). Another study showed that triadimenol treatment may or may not lead to the accumulation of eburicol. It is only in isolates highly resistant to triadimenol where there is no accumulation of eburicol. PCR amplifications showed that all isolates with a resistance factor to triadimenol greater than five displayed a point mutation in the C_{14}-demethylase gene, confirming that a point mutation in the target enzyme of DMI fungicides is a major cause of resistance (Corio-Costet *et al.*, 1998). A single mutation, leading to the substitution of a phenylalanine residue for a tyrosine residue at position 136, was found in all isolates exhibiting a resistance factor higher than five. Further allele-specific PCR experiments revealed that the P-450$_{14DM}$ gene of most, but not all, resistant isolates with a resistance factor of at least five contained the same nucleotide substitution at codon 136. Therefore, it is possible that some resistant isolates of *U. necator* possess another point mutation(s) in the P-450$_{14DM}$ gene that may result in different levels of DMI resistance (Délye *et al.*, 1997).

The new strobilurin (azoxystrobin, kresoxim-methyl and trifloxystrobin) and spiroketalamine (spiroxamine) fungicides represent an important new tool available for the control of grape powdery mildew. Spiroxamine is a new multisite ergosterol biosynthesis inhibitor (Tiemann *et al.*, 1997). Strobilurins are synthetic analogs of the natural lead molecule, strobilurin A, which is an antifungal secondary metabolite of the agaric *Strobilurus tenacellus*. Strobilurin fungicides are inhibitors of mitochondrial respiration. They accomplish this by blocking the electron transfer at the cytochrome bc_1-complex (Ammermann *et al.*, 1992). The strobilurins, which inhibit mitochondrial electron transport at the cytochrome bc_1-complex, fall into a different cross-resistance group from all other commercially available fungicides (FRAC, 1997).

"Soft chemicals"

There is a growing interest in reducing the reliance on conventional fungicides for the management of diseases, not only due to the ever-increasing resistance problems, but also to produce a "purer quality" product. To enhance this trend, many environmentally safer products or safer chemicals are being tested. These include plant oils, mineral oils, phosphates and other inorganic salts, wetting agents and fertilisers. It is clear that the results depend on the concentrations used, time and number of applications and alternation or combination with other fungicides. However, many of the results are positive and some of these materials have the potential to be useful additional tools for the management of grape powdery mildew. They are also cost effective and not detrimental to fruit and wine quality or yield.

Plant oils include a canola oil derivate Synertrol (Azam *et al.*, 1995; Wicks *et al.*, 1995; Northover & Schneider, 1996) and soybean plant oil (Northover & Schneider, 1996). Mineral oils include Sunspray UFO, Safe-T-Side (Northover & Schneider, 1996), Stylet oil (Wicks *et al.*, 1995; Northover & Schneider, 1996; Dell *et al.*, 1998), DCT Plus (Ampol DC Tron Plus) (Wicks *et al.*, 1998), Codicide and Shell Spray (Wood *et al.*, 1995). Shell

spray also gave good results when used in combination with sodium or potassium bicarbonate. Phosphates include K_2HPO_4 and KH_2PO_4 + KOH, both with Triton X-100 (Magarey *et al.*, 1993b; Reuveni & Reuveni, 1995b). Other inorganic salts include $NAHCO_3$ (Reuveni & Reuveni, 1995b; Führ & Hill, 1998) and $KHCO_3$ (Reuveni & Reuveni, 1995b). Wetting agents such as Cittowet alone (Magarey *et al.*, 1993b) or in combination with sodium or potassium bicarbonate gave good results (Wood *et al.*, 1995). Liquid dishwashing detergent (Sunlight) plus sodium or potassium bicarbonate also gave good results (Wood *et al.*, 1995). Fertilisers such as Vigor Cal™, a saccharated calcium product (Culver & Rajamannan, 1997) and soluble silicon sprays make plants more resistant to powdery mildew infections (Bowen *et al.*, 1992; Reynolds *et al.*, 1996). Excessive N-fertilisation, however, increases the susceptibility of grapevines to powdery mildew infections (Bavaresco & Eibach, 1987).

Biological control

To date the only biological agent commercially available for control of grape powdery mildew is the mycoparasite *Ampelomyces quisqualis*, formulated as AQ10™. It is produced using large-scale fermentation and is formulated as a stable water-dispersible granule (WDG) containing conidia as the active ingredient (Daoust & Hofstein, 1996). Germinating conidia form hyphae that are capable of attacking and penetrating the propagating hyphae of the powdery mildew pathogen via a very specific host-parasite interaction. The antagonist forms pycnidia in hyphae, conidiophores, conidia and immature cleistothecia of *U. necator* (Falk *et al.*, 1992). This process will eventually lead to suppression and/or complete elimination of the powdery mildew pathogen. Results obtained in grape-producing regions throughout the world have shown that powdery mildew can be effectively controlled with AQ10™ (Daoust & Hofstein, 1996). In New York, where *U. necator* overwinters as cleistothecia, *A. quisqualis* has the potential to affect the levels of overwintering cleistothecia substantially (Falk *et al.*, 1995).

The only other organisms that show potential as biological agents against powdery mildews are species of the ballistospore-forming yeast *Tilletiopsis* (Knudsen & Skou, 1993; Urquhart *et al.*, 1994).

REDUCED SENSITIVITY TO DMI FUNGICIDES

Potential risk

DMIs belong to the group of site-specific fungicides, which are in general more prone to resistance than conventional multisite inhibitors. After being used for some years, losses in efficiency of triadimefon were noted during 1983 in South Africa (M. Gordon, Bayer, personal communication), 1984 in Portugal (Steva & Clerjeau, 1990), 1985 in California (Ouimette & Gubler, 1990), 1989 in New York (Pearson, 1990) and 1996 in Austria (Redl & Steinkellner, 1996). Resistance to triadimenol was reported during 1988 in Portugal and 1989 in France (Steva & Clerjeau, 1990). In all the cases resistance was associated with the exclusive use of only one product for several years. This suggested that resistance to DMIs developed as slow shifts in the population. The distribution of sensitivity to a DMI fungicide among individuals in an unexposed *U. necator* population (a wild-type population) is continuous, ranging from highly sensitive phenotypes to phenotypes considerably less sensitive (more resistant) than the population mean (Erickson & Wilcox, 1997). On a logarithmic scale EC_{50} frequency distribution will show a normal distribution. The use of DMIs stimulates the selection process of isolates with elevated resistance levels and contributes to a rapid increase in the population; in other words the frequency distribution will be skewed towards the resistant end of the spectrum (Gubler *et al.*, 1996). This type of resistance is polygenic resistance, where many mutant genes are required, each responsible for only a minor resistance step to achieve the highest level of resistance possible (Dekker, 1993). This is also referred to as quantitative, multi-step, continuous, directional or progressive resistance (Brent, 1995). It is generally accepted that resistance in powdery mildew fungi is not determined by one major gene, but rather by several different genes (Buchenauer, 1987). The question of exactly how many genes are involved in regulating DMI resistance remains controversial (Köller, 1996). The various combinations of resistance genes result in a continuous distribution of sensitivity, so that distinct subpopulations cannot easily be recognised and it may be several years before resistance problems will arise in practice. This will depend on the fitness of resistance strains and the selection pressure by the fungicide (Dekker, 1993).

As with *Sphaerotheca fuliginea*, the cucumber powdery mildew pathogen (Schepers, 1985), there does not seem to be a difference in fitness between DMI resistant and wild-type isolates. Délye *et al.* (1997) found no difference in growth between sensitive and resistant isolates where MICs for resistant isolates ranged from 8.5 µM to more than 50 µM triadimenol. Erickson and Wilcox (1997) also showed a 30-fold increase in median ED_{50} values for *U. necator* populations treated with triadimenol. Although there have not been major changes in activity of and resistance to triadimefon, myclobutanil or fenarimol in Californian vineyards since 1990 (Gubler *et al.*, 1996), reductions in mean EC_{50} values were detected for myclobutanil and fenarimol from 1993 to 1994 and for triadimefon from 1994 to 1995. It is not clear whether this was due to physiological limitations in the ability of *U. necator* to express higher levels of resistance or changes in the mildew control programmes and their effects on further selection for resistance. Sulphur was reintroduced in 1987 and is used in alternation with myclobutanil or fenarimol (Ypema *et al.*, 1997). There also seems to be a difference in sensitivity of *U. necator* subcultures to different DMI fungicides. Reduced efficacy has been reported more frequently for triadimefon than for myclobutanil and fenarimol. This can be explained by the lower inherent activity of triadimefon and its longer history of use (Gubler *et al.*, 1996; Ypema *et al.*, 1997). Cross-resistance does exist among the DMIs used to control *U. necator* (Steva & Clerjeau, 1990; Erickson & Wilcox, 1997; Ypema *et al.*, 1997). A time course experiment indicated a steady increase in mean EC_{50} values for triadimefon, myclobutanil and fenarimol, even though triadimefon was the only fungicide applied. This suggested that selection pressures by triadimefon influenced resistance to myclobutanil and fenarimol as well, indicating that the genetic mechanisms conferring resistance to DMI fungicides were correlated (Gubler *et al.*, 1996). However, the degree of cross-resistance might vary substantially between the DMIs. A much greater degree of cross-resistance exists between triadimenol (triazole) and myclobutanil (triazole) than between either of these fungi-

cides and fenarimol (pyrimidine) (Erickson & Wilcox, 1997). DMI cross-resistance seems to be more restricted in the case of *U. necator* than *Venturia inaequalis,* where generally high levels of cross-resistance occur.

In the Western Cape province grapevines are cultivated predominantly in five regions: the Hex River valley, Tulbagh-Worcester-Roberstson valley, Paarl-Franschhoek valley, Stellenbosch region and Riebeeck Kasteel region. The Hex River region is separated from the Tulbagh-Worcester-Robertson region by mountain ranges. The same is true for the Paarl-Franschhoek, Stellenbosch and Riebeeck Kasteel regions, which are separated from the former group. The regional subpopulations had all been exposed to triadimefon or triadimenol prior to 1989, when these fungicides were phased out and other demethylation inhibiting fungicides (DMIs) were applied. A recent investigation (Halleen *et al.*, 2000b) of the distribution of *U. necator* variants resistant to triadimenol, penconazole and flusilazole showed that populations in these regions all had reduced sensitivity for triadimenol. This indicated an earlier shift in triadimenol sensitivity in the subpopulations and showed that resistant variants were sufficiently competitive to become established in vineyards. Cultivation in each of these geographical regions is extensive. Therefore wind may play a prominent role in the dispersal of conidia of resistant variants within each of the regions. This is substantiated by preliminary data (F. Halleen, unpublished) that *U. necator* in vineyards offered by producers as good candidates for obtaining isolates at baseline sensitivity due to no usage of DMIs, or the fact that only sulphur was applied, displayed slight shifts in triadimenol sensitivity. Wind dispersal of conidia over long distances has not been demonstrated for *U. necator*, but has been reported for *Erysiphe graminis* f. sp. *hordei* in northern Europe (Wolfe & McDermontt, 1994).

The occurrence of resistant variants in the subpopulations was also compared with those in a vineyard in the Ceres Karoo region, which was isolated by two mountain ranges from the viticultural regions and where triadimefon was used prior to 1989 before being abandoned. No other DMIs were applied. Cross-resistance between the triazoles was indicated by the frequency at which resistant variants occurred in subpopulations. The Ceres Karoo population was at baseline sensitivity level for penconazole and flusilazole. However, the four populations (De Doorns, Franschhoek, Riebeeck Kasteel and Stellenbosch) which showed the highest shifts in sensitivity to triadimenol, also displayed a high level of reduced sensitivity to flusilazole. This was in spite of the fact that only the Stellenbosch population was regularly treated with flusilazole. The other three populations were predominantly exposed to penconazole. Reduced sensitivity to penconazole was furthermore most prevalent in the Paarl, Riebeeck Kasteel and De Doorns populations. Of these populations, Paarl received predominantly penconazole, whereas the other populations were treated with a range of DMIs (Halleen *et al.*, 2000b). From the above it appears as if resistance to DMIs is a multigenic trait in *U. necator* and that one or more resistance genes are independent with respect to individual DMIs or groupings thereof. This has significant implications for programmes for the management of DMI resistance (Erickson & Wilcox, 1997).

Resistance management

The existing FRAC strategy for effective disease control and resistance management is the use of a maximum of four DMI applications per season. Possible strategies include the use of mixtures or alternation with effective non-cross-resistant fungicides (FRAC, 1997) or alternation with less effective conventional fungicides (sulphur, dinocap, dichlofluanid) (Clerjeau, 1994). The reintroduction of sulphur in Californian vineyards in 1987 has already been linked to reduced DMI resistance in vineyards where resistance had previously been a problem. Alternating sulphur with DMI fungicides in vineyards containing resistant subcultures resulted in better disease control than tank mixing DMI fungicides and sulphur, or DMI fungicides applied by themselves (Ypema *et al.*, 1997). The use of mixtures of DMIs with protective fungicides is not recommended (Wicks *et al.*, 1997). Due to the under-dosage of active ingredients in the mixtures, a more rapid selection of resistant strains has been observed. In addition, an interaction between DMI (triadimenol) and sulphur has proved to be antagonistic (Steva, 1992). Studies with *V. inaequalis* also concluded that low rates of DMIs accelerated the development of resistance (Köller, 1996). DMI fungicides should therefore be used at their maximum registered dose (alone or in mixtures) (FRAC, 1997). DMI fungicides should also not be used in mixtures with copper-based fungicides (cupric hydroxide) used for the control of downy mildew (Anderson & Wicks, 1993). DMIs should only be used in a preventative and not in a curative manner. Therefore applications should start before powdery mildew symptoms occur (FRAC, 1997).

There is a temptation to blame the difficulty of disease control on resistance in all cases. There are many other possible reasons, such as poor application, deterioration of the product or unusually heavy disease pressure (Brent, 1995). Growers should therefore be encouraged to improve their treatment practices and to adhere to recommended timing, application volume and accurate treatment of each row (FRAC, 1997), especially during the most critical period (the first 40 days from budbreak) for disease infection (Magarey *et al.*, 1994).

Disease warning systems (weather stations providing daily powdery mildew risk assessments) have been introduced in the major grape growing areas of the world. This has resulted in a reduction of the number of sprays applied per season (20-50 %) when compared with conventional management practices (Szòke *et al.*, 1998). One of these systems, the AusVit decision support system, does not only make use of climatic conditions, but users are also asked to enter details about grape cultivar, canopy, disease history, the proposed use of the grapes and disease incidence and severity details obtained from monitoring the vineyard site (Magarey *et al.*, 1998). Traditionally South African farmers use a spray programme timed according to grape phenology, with little or no regard for production of primary inoculum by the fungus. A disease warning system was recently evaluated by ARC Infruitec-Nietvoorbij at Stellenboch, South Africa which may help to reduce the number of fungicide applications in a control programme (De Kock, 1995).

A strategy to eradicate overwintering cleistothecia on the bark of grapevines has to be developed. One such a strategy is the use of aqueous solutions of lime sulphur applied as over-the-trellis sprays. Despite good results, the extremely high rates of application required and the volume of water delivered weigh against the adoption of this method (Gadoury *et al.*, 1994). The occurrence of a functional sexual stage creates an opportunity for the devel-

opment of genetic variation in the fungus, specifically the development of pathogenic races and of tolerance to highly selective fungicides. A recent study (Gubler *et al.*, 1996) showed that DMI resistance can be maintained in overwintering ascospores. In South Africa many farmers leave their vineyards unattended after harvest (Feb-May). This can lead to infection of leaves and the opportunity for compatible mating types to produce cleistothecia. In the light of this, farmers will have to be educated in this matter and a method to reduce the mildew infection after harvest will have to be developed.

The potential of resistance development to strobilurins is not known (Ypema & Gold, 1999). It has been recommended that strobilurins should be used as preventative treatments and applications made at the manufacturer's recommended rates. Furthermore, strobilurins should not constitute more than 30% to 50% of the total number of fungicide applications made in one season. They should be used in blocks of one to three consecutive applications. Where blocks of two or three strobilurins are applied they should be separated by a minimum of two applications of a fungicide from a different cross-resistance group (FRAC, 1997).

CONCLUSION

Increased world-wide emphasis on the production of disease-free grapes with minimal fungicide input provides a sound reason for exploring more efficient disease management strategies through a better understanding of *U. necator* epidemiology and population genetics. Although the fungus occurs annually in vineyards in the Western Cape province, relatively little is known of its mode of survival and sources of primary inoculum. Cleistothecia of *U. necator* and typical flag shoot formation were first reported (Halleen & Holz, 2000a) in South Africa in 1996 and 1997, respectively, at least 116 years after the anamorph was reported in 1880 from Constantia, near Cape Town (Du Plessis, 1948). However, the fact that cleistothecia were formed sparsely, sporadically and very late in the growing season, indicates that opposing mating types necessary for cleistothecium production must have been introduced only recently and that opposing mating types are not yet abundant in local vineyards.

In addition to providing early primary inoculum, the sexual stage will contribute to resistance development to highly selective fungicides (Gubler *et al.*, 1996). This is shown by studies indicating that the selection exerted the previous year by the application of DMIs is perceptible at the beginning of the season from the emergence of the first symptoms. When the fungus survives winter as mycelium in the buds, the population is sensitive at the beginning of the season and an evolution in resistance is perceptible on treated plots. However, the decrease of sensitivity is not found at the beginning of the next season (Steva, 1996).

Locally, several triazole fungicides are registered for use on grapevines including triadimefon, triadimenol, penconazole, flusilazole, hexaconazole, myclobutanil, tebuconazole and fluquinconazole (Nel *et al.*, 1999). The pyrimidines nuarimol and fenarimol and the pyridine pyrifenox are also registered DMIs (Nel *et al.*, 1999). Most producers appeared to be adhering to the recommendations of Nietvoorbij (De Klerk, 1988), which advocate five to seven applications per season. It was recently shown (Halleen *et al.*, 2000b) that by following these guidelines resistance developed in *U. necator* to triadimenol, penconazole and flusilazole in South African grapevines. Thus, to ensure that DMIs can remain an effective basis for the control of powdery mildew, a maximum of three applications per season is suggested as recommended by Clerjeau (1994). The introduction of the new strobilurin fungicides, azoxystrobin and kresoxim-methyl (Ypema, 1999) and the biological agent *Ampelomyces quisqualis* formulated as AQ10™ (Daoust & Hofstein, 1996), represent important new tools available for the control of grapevine powdery mildew. Recent studies also indicate that substantial ontogenic resistance is expressed much earlier than previously believed, which narrows the period of susceptibility (Gadoury, 1998). A better understanding of the prevalence of mating types, seasonal distribution of ascospore release and the association of ascosporic infection with crop loss in local vineyards, together with the availability of a wider range of products with different modes of action will eventually lead to better powdery mildew control and management of fungicide resistance.

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