APR15

Pathogen of the month – April 2015

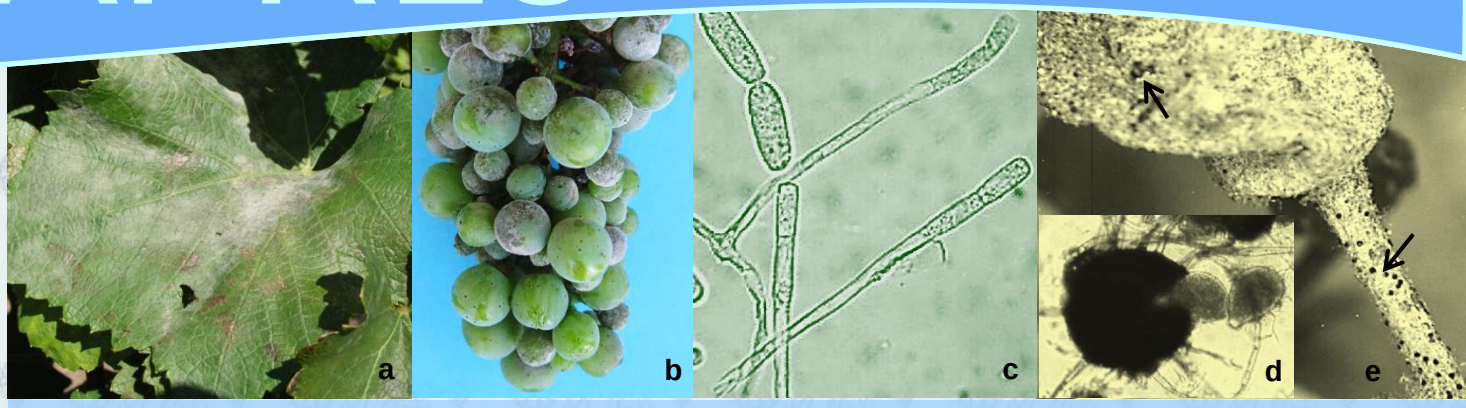

Fig. 1. Powdery mildew symptoms. White powder on upper leaf surface (a) and grey on bunch (b); Conidiophores and conidia (c); Chasmothecia with immature asci (d) on rachis and berry (e). Photo credits: Suzanne Leong-Scott (a) and Tijana Petrovic (b, c, d, e).

Disease: Powdery mildew
Classification: K: Eumycota, D: Ascomycota, C: Leotiomycetes, O: Erysiphales, F: Erysiphaceae.

Powdery mildew (PM) is a ubiquitous disease of grapevine that can reduce yield and quality of grapes and wine. It is estimated to cost the Australian grape and wine industry $76 million per annum. Wine made from PM-affected grapes may have elevated acidity, phenolics, flavonoids and hydroxycinnamates. Total soluble solids and spectral colour may be affected. Sensory attributes of white wines can be modified (e.g. fungal, earthy, cooked tomato aromas). Many Australian wineries use a rejection threshold of 3-5% surface area affected by PM, based on visual assessment.

Erysiphe necator Schwein on grapevine

The Pathogen: *Erysiphe necator* was described on grapes in North America (1834) and was introduced to Europe in the mid-1850s and to Australia in 1866. The fungus is an obligately biotrophic pathogen and grows as superficial hyphae which give rise to haustoria in epidermal cells of green grapevine tissue. Two genetic groups, A and B, have been documented in Australia, Europe and USA. Group A is associated with flag shoots, is found early in an epidemic and subpopulations are somewhat diverse. Group B is associated with flag shoots and ascospore infection, is found later in an epidemic and subpopulations are more diverse.

Host Range: Many genera within the Vitaceae; *Vitis, Cissus, Parthenocissus* and *Ampelopsis.* The European grape (*V. vinifera*) is highly susceptible to *E. necator*.

Biology and ecology: *E. necator* overwinters as mycelia and conidia (asexual stage) in dormant buds, and as chasmothecia (sexual stage) on the bark on the trunk and cordons. Mycelia are activated at bud burst and infected emerging shoots are deformed (flag shoots) and covered with mycelia and conidia. Chasmothecia release ascospores when precipitation (2-2.5mm) coincides with temperature above 10°C. Ascospores infect leaves adjacent to cordons, forming PM colonies. Conidia are wind-dispersed and initiate multiple infections on green tissues throughout the growing season. Clusters are highly susceptible in the first few weeks of berry formation. Ontogenic resistance is expressed about 3-4 weeks after the completion of bloom.

Symptoms: Disease appears on all green parts of the vine; leaves, inflorescence and berries (Fig. 1a-b). Young and senescent colonies are powdery white and grey, respectively. Young colonies contain conidiophores and conidia (Fig. 1c). Chasmothecia develop within senescent colonies (Fig. 1d-e).

Disease management: Measures to produce an open canopy, such as training system and removal of leaves, reduce PM development. Inorganic (e.g. S, mineral oil, salts) fungicides, organic oils and synthetic (e.g. strobilurins, demethylation inhibitors) fungicides may control PM.

Further Reading: Brewer & Milgroom (2010) *BMC Evolutionary Biology* 10:268; Gadoury et al. (2012) *Molecular Plant Pathology* 13:1-16; Scott et al. *(*2010*)* In *Viticulture and Wine Quality* (Vol 1), Woodhead Publishing, Cambridge, UK, 481-514; Scholefield and Morison (2010) http://www.gwrdc.com.au/wp-content/uploads/2012/09/GWR-08-04.pdf.

Key Contact: Eileen Scott, University of Adelaide; e-mail: eileen.scott@adelaide.edu.au;
Tim Zanker, University of Adelaide; e-mail: timothy.zanker@adelaide.edu.au;
Tijana Petrovic, University of Adelaide; e-mail: tijana.petrovic@adelaide.edu.au