**Please note that the Proceedings will be published on BIO Web of Conferences journal and the minimum length is three (3) pages**

**Modelling the life cycle of *Erysiphe necator* [Title: Times 12, Bold]**

Legler1, S.E., Caffi1, T., Rossi1, V., and S. Giosuè2 [Authors: Times 10]

1Università Cattolica del Sacro Cuore, Istituto di Entomologia e Patologia Vegetale, I-29122 Piacenza, Italy

2Horta Srl, spin off company of Università Cattolica del Sacro Cuore, I-29122 Piacenza, Italy [Affiliation: Times 9]

\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

[Running text: Times 9]The fungus *Erisyphe necator* (syn. *Uncinula necator*) (Schw.) Burr. is the causal agent of powdery mildew, a major grapevine disease throughout the world. Because this disease causes serious economic losses, the life cycle of the fungus has been extensively studied. *E. necator* is a polycyclic pathogen that exhibits polymorphism in its spore forms, with sexual (i.e., the ascospores) and asexual (i.e., the conidia) reproduction. Its life cycle is characterised by a chain of primary and secondary infection cycles that partially overlap in time (Fig. 1).

*E. necator* overwinters as sexual fruiting bodies, the chasmothecia (formerly cleistothecia), on the vine bark (Pearson and Gadoury, 1987; Gadoury and Pearson, 1988; Cortesi *et al.*, 1997; Jailloux *et al.*, 1998; Füzi 1999). In spring, ascospores are repeatedly released from chasmothecia (Fig. 2), and once they reach the leaf surface, they germinate and cause the primary infections though a biotrophic relationship with the epidermal leaf cells. The fungal colony grows on the leaf surface and forms the conidia, which cause new, secondary infection cycles and cause the epidemic development from late spring to summer (Gadoury and Pearson, 1988; Cortesi *et al.*, 1997; Gee *et al*., 2000). In late summer, the pathogen forms new chasmothecia; because *E. necator* is heterothallic, these fruiting bodies form on the affected host tissue when two opposite mating types are in proximity and their antheridium and oogonium combine (Schnathorst, 1965; Gadoury and Pearson, 1988). Mature chasmothecia can either be dispersed by rain splashes to the vine bark, where they overwinter, or they can release ascospores in autumn; the role of ascospore release in the late season is not clear. Increasing genetic diversity of the fungal population may increase the probability that two opposite mating types mate and, according to Gee et al. (2000), the infection efficiency of ascospores. In some cases, the pathogen can overwinter as mycelium in the dormant vine buds. Shoots developing in spring from affected buds are known as flag shoots, which represent a source of inoculum for secondary infections.

The processes described in the previous paragraph can be grouped into five stages: i) development and maturation of chasmothecia, ascospore dispersal, and ascospore infection; iii) growth of fungal colonies; iv) latency and clonal sporulation; and v) conidial infection.All five stagesare strongly influenced by weather.

Control of powdery mildew is traditionally based on the management of secondary infections. According to a survey by the European Commission, in 2007 growers in Europe used 70000 tons of fungicides for grape protection, 53000 tons of which were used against *E. necator* (EC, 2007). Despite this large use of fungicides, powdery mildew epidemics are frequently difficult to control because of the explosive nature of the infection cycles caused by clonal reproduction. Modelling plant diseases is a key approach for rationalizing disease management actions, including fungicide sprays (Rossi *et al.*, 2010). Weather-driven models have been developed for simulating parts of the life cycle of *E. necator* to evaluate the risk of powdery mildew infection and thereby to schedule fungicide applications. Some of these models are briefly described in the following paragraphs.



Figure 1: Life cycle of *Erysiphe necator*, the casual agent of powdery mildew of grapevine.

**Models for chasmothecia development and maturation.** Rossi et al. (2009a) developed a mechanistic dynamic model to predict the maturation of the *E. necator* chasmothecia in the vineyard. In this model, the chasmothecia advance from one stage of maturation to the next (i.e, from white, yellow, brown, and dark) at specific maturation rates that depend on air temperature Mature black chasmothecia are finally dispersed by rain splashes.



Figure 2: Presence of *Erysiphe necator* chasmothecia, ascospores, and conidia during the year in northern Italy. The arrow indicates the average time of grapevine bud break.

**Models for primary infections or disease onset.** Kast (1995) developed an empirical model for timing the first application of fungicides against powdery mildew based on data collected over more than 50 years in the wine regions of Wuerttemberg and Rheinhessen in Germany. This date is calculated as a time lag with respect to the development stage “three leaves unfolded”, using a rough indexing of the disease severity for a vine site in the preceding year and the mean of the lowest temperatures in the two previous winters.

The UC Davis model (Gubler *et al.*, 1999) was developed for use in California. This is a rule-based model that accounts for both ascosporic and conidial stages of *E. necator*. The ascosporic part of the model was designed to estimate the risk of ascospore release from chasmothecia and consequent primary infection. Predictions are based on average temperature during an extended leaf-wetness event; the model uses the 'Conidial Mills Table' at 2/3s value for hours of leaf wetness required at various temperatures. In general, at least 12-15 hours of continuous leaf wetness are required when temperatures are between 10-15°C.

Gadoury and Pearson (1990) developed simple rules for minimal conditions for ascospore discharge from chasmothecia, i.e., 2.5 mm of rainfall and a temperature of 10°C.

Recently, Caffi and Rossi (2009) developed a mechanistic dynamic model for the simulation of *E. necator* ascosporic infections. The model uses air temperature, relative humidity, leaf wetness duration, rainfall, and vapour pressure deficit to calculate: i) ascospore maturation rates in spring; ii) ascospore dispersal events; iii) proportion of ascospores ejected in each discharge event; iv) infection efficiency of ascospores; v) probable onset of the disease symptoms; and v) duration of the latent period, i.e., the period between infection and production of asexual spores.

**Models for secondary infections or disease risk.** The model of Chellemi and Marois (1992) simulates the population growth of *E. necator* on *Vitis vinifera* ‘Carignane’ over time. This model follows the fate of each secondary infection cycle from germination of conidia until sporulation ceases; population size is determined by the number of viable conidia present each day. Equations accounting for the effect of temperature and liquid water on germination, penetration, and daily sporulation rates over the infectious period are included. The probability of conidia being deposited on susceptible leaf tissue is also considered.

Once ascosporic infection has occurred, the model of Gubler *et al.* (1999) switches to a risk index that is based entirely on the effect of temperature on the reproductive rate of the pathogen. The index fluctuates between 0 and 100; it increases by 20 points for each day with at least 6 hours between 21-30°C, while it decreases by 10 points for each day with less than 6 hours between 21-30°C or with a minimum temperature above 35°C. The use of the model allows grape growers to lengthen spray intervals during times of low to intermediate disease pressure and to shorten intervals when pressure is high. An index of 0-30 indicates that the pathogen is functioning minimally and is producing new conidia every 15 days or not at all. An index of 40-50 is considered normal and indicates that new conidia are being produced every 8-11 days. An index of 60-100 indicates the pathogen is producing new conidia every 5 days.

Kast (1995) developed a program named OiDiag that allows the grower to adjust the interval between sprays by calculating a temperature-dependent index accounting for powdery mildew development. Index values are calculated based on temperature, number of hours with humidity between 65 to 80% or >80% per day, duration of leaf wetness, and rainfall within periods of 14 days. The new version OiDiag-2.2 also considers the ontogenetic resistance of grapes in that higher index values are calculated for the period 10 days between the start of flowering and 10 days after the end of flowering.

Carisse et al. (2009) recently developed a risk assessment based on the relationship between incidence of powdery mildew on the leaves of different cultivars and cumulative concentration of airborne conidia in the vine production area of Quebec, Canada. An action threshold of 50 conidia per m3 air per day is used for timing the interval between fungicide sprays.

**Models for epidemic development.** Sall (1980) developed a mathematical model accounting for the influence of seasonal weather patterns and timing of initial infections on fungus colonization of grapevine leaves and bunches. This model is mainly based on Vanderplank’s equation; the powdery mildew infection rate is calculated based on temperature and moisture conditions. The model is linked to a vine growth submodel that describes the increase in the surface area of the susceptible parts of the vine, based on temperature.

Bendek (2002) developed a regression model to describe the development of powdery mildew incidence from flowering to the development of berries 5 mm in diameter in central Chile. The model is based on temperature and relative humidity.

Recently, Calonnec *et al.* (2008) developed a model coupling temporal and spatial vine growth with the development and spread of powdery mildew at the vine stock scale. In this model, the dynamics of the pathogen population on leaves are split into infection, colony or mycelium growth, and sporulation and dispersion. The time between infection and sporulation is described as the latent period, and the duration of sporulation is described as the infectious period. Temperature, wind speed, and wind direction are the main input variables. The environmental variables also dictate growth of the crop (appearance and growth of vine organs).

Despite the large number of relevant models, a holistic approach to quantitative modelling of the *E. necator* life cycle has been lacking. Recently, a model has been elaborated for *Plasmopara viticola* that links quantitative aspects of both sexual and asexual stages in a biologically coherent framework (Rossi *et al.*, 2009b).

An approach like the one used for *P. viticola* is proposed for *E. necator*, as diagrammed in Figure 3. This model considers the entire life cycle of the pathogen, allowing a global view of the pathosystem as a dynamic process. Rates of this model (i.e., discharge of ascospores, deposition of ascospores and conidia, infection by ascospores and conidia, sporulation, mating, and maturation and dispersal of chasmothecia) and relevant periods (i.e., incubation, latency, and infectiousness) must be described mathematically as a function of the influencing weather variables. Several of these mathematical functions have already been elaborated in the previously cited models. This model should facilitate an integrated approach for protecting grapevines against powdery mildew and should guide all management options, from reducing the overwintering inoculum to protecting leaves and bunches.



Figure 3: Relational diagram of a model describing the entire life cycle of *Erysiphe necator*. Boxes contain state variables, valves are rates, diamonds are switches, while clouds represent ascospores, colonies, or conidia that leave the system. Lines with circles are external variables related to the plant: LAI and BAI are leaf and bark area index, respectively; bb and lf are the time of bud break and complete leaf fall, respectively; k is the carrying capacity of the plant. The variable t is the day of the year; i, l, and p are incubation, latency, and infectious periods, respectively. Inf=1 means minimum conditions for infection are met.

**Literature Cited**

1. Bendek C. 2002. Development a predictive epidemiologist model of grape powdery mildew (*Uncinula necator*) forecast in Chile. Thesis, Magister in Ciencias Vegetales, Pontificia Universidad Católica de Chile, pp. 45.
2. Caffi T. and Rossi V. 2009. A Mechanistic Model for Infection of Grapevines by Ascospores of *Erysiphe necator.* Proceedings of the 10th International Epidemiology Workshop, pp. 23-25.
3. Calonnec A., Cartolaro P., Naulin J.M., Bailey D. and Langlais M. 2008. A host-pathogen simulation model: powdery mildew of grapevine. *Plant Pathology*, 57, 493-508.
4. Carisse O., Bacona R. and Lefebvre A. 2009. Grape powdery mildew (*Erysiphe necator*) risk assessment based on airborne conidium concentration. *Crop Protection*, 28, 1036-1044.
5. Chellemi D.O. and Marois J.J. 1992. Development of a demographic model for *Uncinula necator* by using a microcomputer spreadsheet program. *Phytopathology*, 81, 250-254.
6. Cortesi P., Bisiach M., Ricciolini M. and Gadoury D. M. 1997. Cleistothecia of *Uncinula necator* – An additional source of inoculum in Italian vineyards. *Plant Dis*., 81, 922-926.
7. European Commission 2007. The use of plant protection products in the European Union - Data 1992-2003, Luxembourg: Office for Official Publications of the European Communities, 222 pp. ISBN 92-79-03890-7
8. Füzi I. 1999. The epidemic role of cleistothecia of grapevine powdery mildew at Szekszard vine-growing region. Növényvédelem, 35, 215-221.
9. Gadoury D. M. and Pearson R. C. 1988. Initiation, development, dispersal, and survival of cleistothecia of *Uncinula necator* in New York vineyards. *Phytopathology,* 78, 1413-1421.
10. Gadoury D. M. and Pearson R. C. 1990. Ascocarp dehiscence and ascospore discharge by *Uncinula necator. Phytopathology*, 80, 393-401.
11. Gee L. M., Stummer B. E., Gadoury D. M., Biggins L. T. and Scott E. S. 2000. Maturation of cleistothecia of *Uncinula necator* (powdery mildew) and release of ascospores in southern Australia. *Australian Journal of Grape and Wine Research,* 6, 13-20.
12. Gubler W.D., Thomas C.S., Weber E. and Luvisi D. 1999. Development and implementation of a powdery mildew risk model for grapevines. (Abstr.) Proc. 1st Int. Powdery Mildew Conf., Avignon, France.
13. Jailloux F., Thind T. and Clerjeau M. 1998. Release, germination, and pathogenicity of ascospores of *Uncinula necator* under controlled conditions. *Can. J. Bot*., 76, 777-781.
14. Kast W.K. 1995. A step by step risk analysis (SRA) used for planning sprays against powdery mildew (OiDiag-System). *Viticolture Enological Science*, 52, 230-321.
15. Pearson R.C. and Gadoury D. M. 1987. Cleistothecia, the source of primary inoculum for grape powdery mildew in New York. *Phytopathology,* 77, 1509-1514.
16. Rossi V., Caffi T. and Legler S.E. 2009a. A Model for Maturation and Dispersal of *Erysiphe necator* Cleistothecia.Proceedings of the 10th International Epidemiology Workshop, pp. 138-139.
17. Rossi V., Giosuè S. and Caffi T. 2009. Modelling the dynamics of infections caused by sexual and asexual spores during *Plasmopara viticola* epidemics. *Journal of Plant Pathology*, 91 (3): 615-627.
18. Rossi V., Giosue’ S., Caffi T. 2010 (in press). Modelling Plant diseases for decision making in crop protection. In: Precision Crop Protection - The Challenge and Use of Heterogeneity, Oerke E.C. (ed), Springer Science, Dordrecht (NL).
19. Sall M.A. 1980. Epidemiology of grape powdery mildew: a model. Phytopathology, 70, 338-342.

