Epidemiology and Control of Grape Black Rot in Southern Switzerland

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ABSTRACT

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Guignardia bidwellii, the causal agent of black rot of grape, appeared in 1988 in a restricted area in Switzerland. Ascospores discharged mainly at the beginning of and during flowering in correspondence with initiation of rain. Leaf infections had little correlation with disease on bunches. Secondary infections seemed to play no major role in disease on bunches. Loads of primary inoculum must be consistent to cause problems. We suggest that in the vine growing systems used traditionally (hand pruning), black rot disease can be avoided by sanitation measures.

Additional keyword: *Vitis vinifera*

Black rot is described as one of the most economically important diseases of grape. Losses may range from 5 to 80%, depending on the severity of the epidemic, which is thought to be governed by inoculum level, weather, and cultivar susceptibility (11). The pathogen, *Guignardia bidwellii* (Ellis) Viala & Ravaz, originates in North America and was first introduced into Europe around 1885. It first appeared in Switzerland in 1988, only in a restricted region south of the Alps (Canton Ticino), where 170 ha of Merlot grapes are grown. The disease caused great losses in individual vineyards (10). The disease cycle, epidemiology of the pathogen, role of the ascospores (5) and conidia (6) in infection, correlation between leaf wetness duration and temperature to determine infection periods (12,14), and incubation and latency periods and their relation to temperature and humidity, have been described (3,13, 14), as well as the role of precipitation on the liberation and diffusion of the conidia (6,13). Biomathematical models have been formulated to determine the infection periods and aid the scheduling of fungicide treatments (4,9,14). Disease severity in the first year of appearance in Ticino led to widespread fear and a demand to establish a protocol for preventive fungicide treatments. This study was initiated, therefore, to obtain the necessary data under the particular pedoclimatic conditions in Ticino with the grape cv. Merlot and the particular training and planting system, so as to formulate appropriate control strategies. Since the disease was expanding from a few sites, the spread of the pathogen from a focus was also studied.

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MATERIALS AND METHODS

Maturation of the perithecia and ascospore discharge. At harvest, berries with clear symptoms of black rot were collected and incubated over winter in a wire mesh box in the test vineyards. In spring at weekly intervals some berries were examined under a dissecting microscope (20 to 40×) for the presence of perithecia containing asci with differentiated ascospores. Once found, ascospore discharge was continuously monitored through a suction spore trap with the air inlet placed at 7.5 cm above the overwintered berries, with one revolution in 24 h. Rainfall (>0.2 mm), humidity, and temperature were monitored (datalogger [Campbell Scientific Ltd.] with sensors placed close to the overwintering berries). The spore capture strip from each day was stained in 0.5% safranin and washed and cut into 24 portions, which were mounted on a glass slide and observed in a microscope. Ascospores of *G. bidwellii* were determined by their size (5 to 7 × 12 to 17 µm) and shape (oval, oblong with two strongly refracting droplets on each end) (6). Their number was estimated according to the following scale: 0 = 0 ascospores; 5 = 1 to 10; 25 = 11 to 40; 70 = 41 to 100; 500 = 101 to 1,000; 5,000 = 1,001 to 10,000; and 20,000 = >10,000.

Experimental plots. In 1989, the evaluation of disease progression was made in a vineyard on a hillside at Gudo that in 1988 presented heavy losses due to black rot. It was planted with *Vitis vinifera* cv. Merlot on rootstock *Riparia* × *Rupestris* 3309. Two thousand vines were planted at 1.3 m distances in horizontal rows and trained as double Guyot. To avoid the interference of downy mildew and Botrytis rot, which are the main problems in the region, a typical spraying program was followed (6 treatments, with total of 6 kg of folpet, 4 kg of aethylphosphit of aluminum, 0.3 kg of metalaxyl, 1.2 kg of copper, and 1 kg iprodione). In 1990, we chose a commercial vineyard (at Cugnasco) that the year before presented losses and had basically the same characteristics. This vineyard was on flat ground with 614 vines planted at 1.3 m distances on the row and 1.8 m between the rows. The vineyard was treated eight times against the above mentioned diseases (total of 8 kg of folpet, 10 kg of aethylphosphit of aluminum, 0.3 kg of metalaxyl, and 1 kg of iprodione).

To determine the capacity of the pathogen to expand from a point source, we used a plot of 384 vines trained as double Guyot in flat ground in 16 rows (distance between rows 1.80 m, inside rows 1.40 m) of the experimental vineyard of Cugnasco, isolated from other vine-growing areas, where black rot had never been observed. Inoculum consisting of 140 mummified berries with mature perithecia was placed in a mesh box under a vine on 30 April 1991. Four treatments of folpet (with a total of 11.4 kg) to control downy mildew were also made.

Disease assessment. At the first detection of disease 10 vines were randomly chosen. During the season, these 10 vines were observed once weekly. Total number of leaves and bunches with and without symptoms (incidence) were counted. Severity on bunches was individually assessed: 0 (no symptoms), and 10, 25, 50, 75, and 100% of the berries of the bunch presenting symptoms. Disease severity on bunches was calculated as the average of all bunches present. Rot due to other causes was not considered.

To follow the spread of disease from a point source in 1991, on these 10 vines the total number of leaves per vine with spots of black rot was counted once each week.

RESULTS AND DISCUSSION

Maturation of the perithecia and ascospore liberation. On 30 April 1990, the first differentiated ascospores were noted in the perithecia. This coincided with stage D of shoot development (tip of leaves visible) (1). Ascospores were first captured with the first rainfall after that date (6

May) (Fig. 1). From 6 May until the last capture on 3 July, 36 days with rain were registered and ascospores were captured on all these days. The period was covered by 22 distinguishable ascospore liberation periods of a mean length of 17.3 ± 3.0 h, with the shortest covering 4 h and the longest 60 h (Fig. 1). Captures usually occurred within the hour in which the first rain was registered (14 of 22 periods). In five cases, capture anticipated the registration of rain, this probably was an artifact of the measuring system, which needed at least 0.2 mm of rain before the start of a rain event was registered. In one instance, captures initiated 2 h after a rain event began. In two other instances, an unexplainable 4-h delay in capturing ascospores was noted. We can assume that ascospores usually will be liberated as soon as a rain event starts and berries are wetted. Light has no influence on this pathogen, contrary to other comparable pathogens such as *Venturia inaequalis* (8). All ascospore liberation periods showed a similar pattern; the first hour over 90% of ascospores were captured, but in the following 4 to 5 h captures decreased rapidly, to less than 10 ascospores per h. As long as rain continued, ascospores were detected but always at an insignificant level. Evidently, drying between two rain periods allowed more ascospores to mature than did continuous wetting as, after each dry period, ascospores could be captured in great numbers even if that interval was short (data not shown). It seems that the strategy of the pathogen is to liberate its spores right at the beginning of each rain event, then conserve them until the next rain period.

Ferrin and Ramsdell (5) reported that the liberation can continue up to 8 h after cessation of rain. Our data showed a great variability in this respect. Most often captures ceased with the end of rain, but if high humidity continued, few spores were captured 14 h later. The two data sets can only be compared at high numbers of ascospore release as Ferrin and Ramsdell captured airborne ascospores with a Burckard volumetric spore trap set in the vineyard without deposit. The liberation periods in which most ascospores were captured were between 23 May and 8 June (Fig. 1), which corresponded phenologically to flower button separated (H) to full flowering. Ferrin and Ramsdell (5) also indicated that most ascospores were liberated shortly before and during flowering and concluded that this period was when grape bunches showed the highest susceptibility (5).

Disease progression. In 1989, differentiated ascospores were first observed on 19 April. First symptoms (necrotic flecks with

Table 1. Correlation coefficient (R^2) between leaf disease incidence (percent leaves with symptoms), severity (no. of spots per leaf), bunch incidence (percent bunches with symptoms) and black rot severity on bunch (percent rotted berries) in a vineyard with natural infection, calculated from the data of 3 August 1989 and 27 July 1990 (see Figure 2)[a]

	Dependent variable		
Independent variable	Leaf incidence	Bunch incidence	Bunch severity
Leaf severity (Number of spots on leaves)	91.0 / 71.7	49.13 / 28.3	48.0 / 47.8
Leaf incidence		47.6 / 25.2	49.11 / 35.9
Bunches severity (percent infected berries)		89.8 / 91.9	

[a] Sampling size was 10 vines.

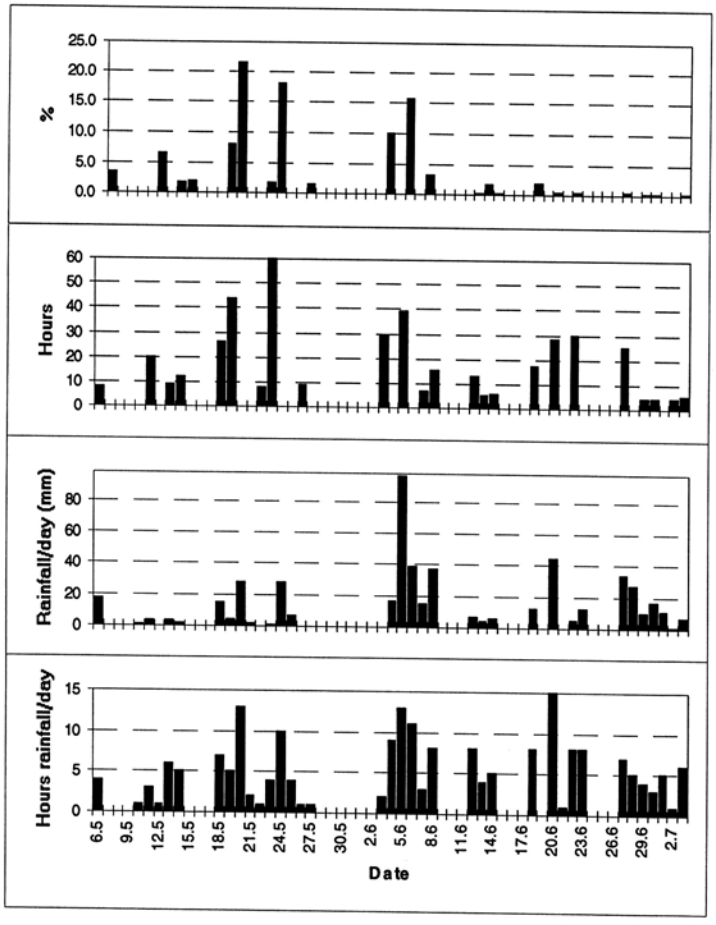

Fig 1. *Guignardia bidwellii* ascospore discharge per event in percentage of all ascospores counted during one season in a spore trap (top graphic), length of event in h (second graphic) (as a single event is considered continuous count or with interruption of less than 6 h of ascospores) and corresponding rainfall in mm and duration per day.

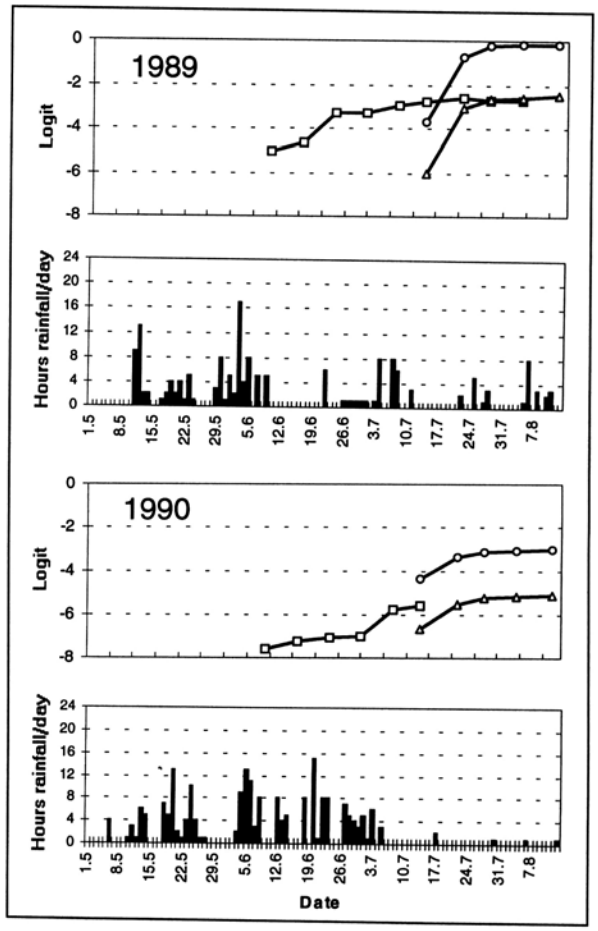

Fig. 2. Incidence of black-rot caused by *Guignardia bidwellii* on leaves (squares) and bunches (circles) in percentage and severity on bunches (triangles) (as average percentage of berries with symptoms of all berries) in 1989 and 1990 with the corresponding rainfall duration. Data transformed in logit as ln (X/(1 – X)).

pycnidia) were observed on leaves in the vineyard 51 days later, on 9 June (Fig. 2). From that date the disease incidence on leaves increased rapidly for a 14-day period, slowing down until reaching a maximum on 21 July 1989 of almost 7% of all leaves (Fig. 2). First symptoms on berries were observed on 11 July (2.4% of all bunches presented symptoms); disease incidence increased rapidly, reaching 46% of all bunches infected by 26 July 1989 (Fig. 2). After that date shoots were topped and leaves removed in the grape zone. No further increase in disease incidence was noted. The evolution of disease severity as percentage of berries with black rot was similar, but even when the disease was generalized, severity stayed low, reaching a maximum of 7.6%. This generalization is shown by the fact that initially most vines showed no symptoms or had only 0 to 5% of the bunches with black rot, except a single vine with 15% infected bunches. In less than 1 month most vines expressed disease severity greater than 15%.

The length of the incubation period in the temperature range of 15 to 24°C is around 13 to 17 days (4). Therefore, in 1989 infection of the bunches should have taken place sometime between 26 June and 7 July, which corresponded to the end of the flowering time and the beginning of fruit set. The inoculum could have been either ascospores or pycnidiospores from infected leaves. Apparently, the rain periods after 21 July did not cause any more infections.

In 1990, the first perithecia with differentiated ascospores were found on the overwintering berries on 30 April, whereas the first symptoms on leaves were detected 40 days later, on June 9. Even though the spring was rainy with numerous periods favorable to infection, progression of the epidemic was slow, occurring mainly between 10 and 17 July, and disease incidence on leaves was only 0.36% by 14 July (Fig. 2, 1990). The first symptoms on bunches were noted on 10 July, which corresponded to observations made in 1989. The main increase was again in mid of July, reaching a maximum of 4.6% infected bunches by 30 July (Fig. 2, 1990) with a disease severity of 0.58%. Infections of the berries probably took place between 27 June and 5 July, corresponding to the time of fruit set. It can be assumed that the inoculum consisted mostly of ascospores since leaf infection incidence was still very low. Comparing progression of the disease on the leaves between 1989 and 1990, we noted that the disease was slowed down in 1990 even though the weather pattern was favorable. In contrast, disease severity on bunches with any symptoms (proportion of infected on berries) was similar in both years (16.5 and 13%, respectively). The capability of the fungus to infect berries seems to be associated with the phenological state, which would support prior findings (5,6). When favorable weather conditions coincided with berry susceptibility, primary infection of bunches occurred and the final disease severity was correlated with disease incidence on bunches (Table 1). Similarly, disease incidence on leaves was correlated with the final number of spots. On the other hand, no correlation could be detected between disease incidence on leaves on a particular vine and incidence or severity of black rot on bunches (Table 1). The difference in disease severity between 1989 and 1990 can be explained by the quantity of the ascosporic inoculum. The vineyard used for the 1989 experiment was pruned and residues left in the vineyard. In the vineyard used in 1990, the grower was aware of the problem and, instead, the infected mummified berries were collected and removed from the vineyard, thus reducing the initial inoculum decisively. Three comparable vineyards in the immediate proximity, which in 1989 had similar incidences of black rot, were not cleaned and showed bunches with a minimum berry disease incidence of 30 to 60% (two vineyards) and more than 60% (third vine-

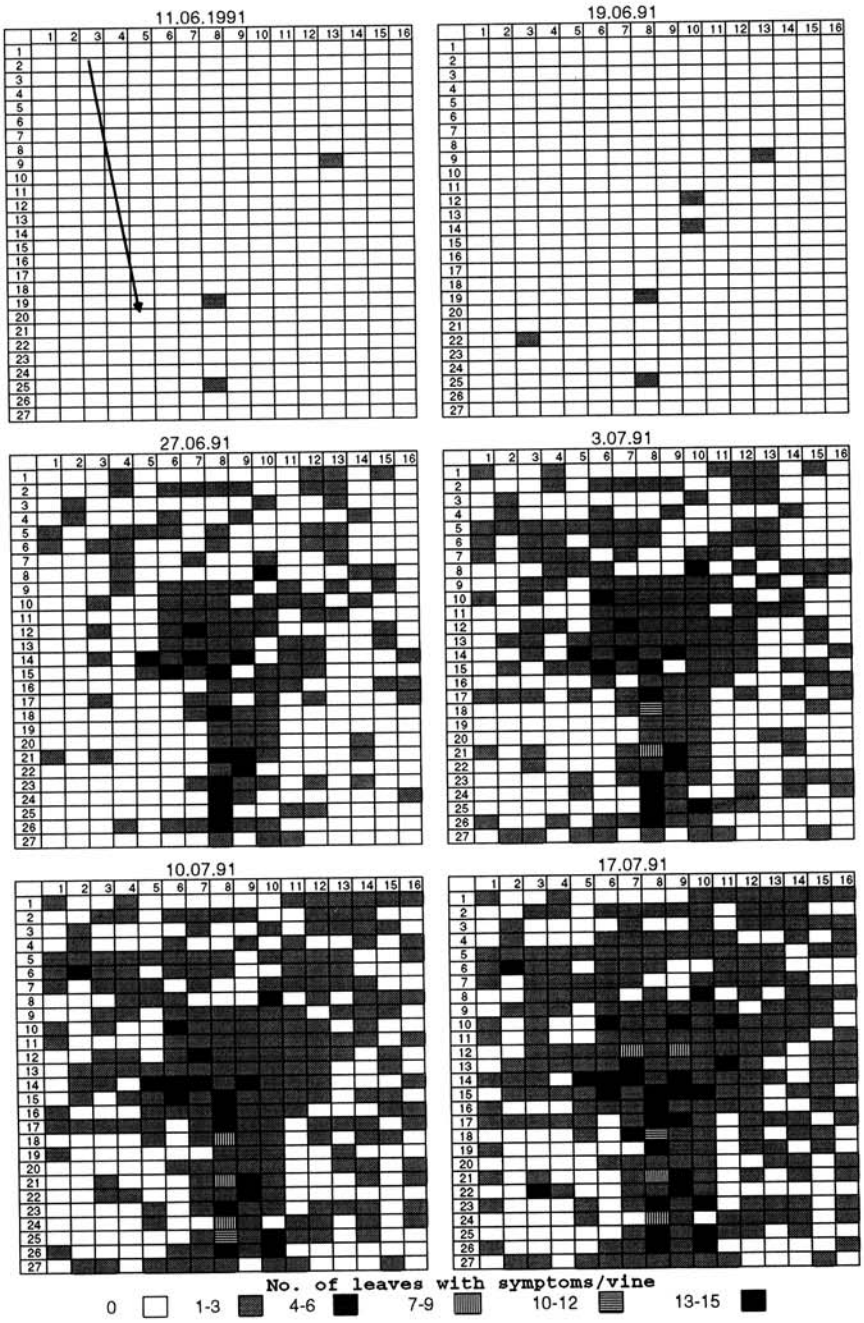

Fig. 3. Spatial evolution of grape black rot caused by *Guignardia bidwellii* from a point inoculum source placed in row 8 vine 14. Evaluation on six different dates. Each small square represents a single vine, which can be identified by number of the row (upper horizontal axes, 1 to 16) and number of the vine in that row (left axes, 1 to 27). Patterns and numbers (at base of figure) indicate number of leaves per vine that present black rot symptoms. Average number of leaves per vine = 240. Prevailing wind direction during rainstorms indicated by the arrow.

yard) in August 1990.

Spatial progression of the epidemic. The spatial progression of the appearance of disease symptoms from a point source of inoculum indicated a scattered expansion but with a higher incidence in the row where the inoculum was placed than in neighboring rows (Fig. 3). However, secondary infections were only apparent after 19 June. The symptoms found in rows 3, 10, and 13 can be attributed to ascospores derived from the introduced inoculum and therefore the gradient of distribution was flat. Moreover, the primary inoculum quantity was not sufficient to cause an alarming disease level. Secondary infections led to locally higher but insignificant numbers of infected leaves with spots on 17 July (Fig. 3). Black rot on berries was not detected in this vineyard. This pattern of disease progression can be attributed to a delayed disease increase as in 1990, after the time of maximum susceptibility of the berries, and to a low primary inoculum level.

This study showed that under low inoculum situation, the black rot fungus does not lead to a vast epidemic. Its capacity to expand is limited to the leaves, as berries are highly susceptible only during a short period. High incidence on leaves, due to disease progression in summer, does not lead to berry loss. Problems arise only if the disease is allowed to build up inoculum over the years. Inoculum quantities have to be consistent to cause problems. In traditional (hand pruning) vine-growing systems, major problems from black rot can be avoided by sanitation measures as shown in previous studies (2,7). Still, if a chemical treatment is considered, it should be applied only during the most sensitive period of the berries (e.g., after flowering up to fruit set).

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