Esca (black measles) of grapevine. An overview

LUIGI CHIARAPPA

Plant Pathology and International Agriculture, 4221 Montgomery Avenue, Davis, CA 95616, USA

Summary. Research on the nature of esca started in 1898 in France and continued there until 1926. Two lignicolous basidiomycetes (Stereum hirsutum and Fomes igniarius) were believed to be the causal organisms of an internal wood decay. Studies conducted by Petri in Sicily in 1912 revealed that fungi of the genera Cephalosporium and Acremonium were causing alterations around and far away from wounds. In 1959 in California, the pathogenicity of Cephalosporium species and the wood-rotting ability of Fomes igniarius were proved. In the 90's numerous studies were conducted in France, Italy, South Africa and California, and important advances were made in understanding the etiology and epidemiology of the disease.

Key words: esca, black measles, grapevine.

More than 100 years ago a grapevine disease of unknown etiology was recognized in France and California, where it was called “fleeting” (Ravaz, 1898) and “sunstroke” respectively (Anonymous, 1895). In both cases the disorder was considered to be due to a physiological condition resulting from what was called “abrupt changes in the bottom water level.”

Research on the pathological nature of the problem started in France in 1898 and is still active today on three continents. It has been characterized by three periods of intensive interest in the disease etiology, with two long pauses, sixty and thirty years long respectively, during which little or no research was done.

Ravaz (1909) was the first to observe fungal mycelium in the tissues of diseased vines, suggesting the parasitic nature of the disorder. Sporocarps found on diseased vines were identified as belonging to the fungus Fomes igniarius (L. ex Fr.) Kickx. However, pathogenicity tests were unsuccessful.

Vinet (1909) recorded the presence of another fungus, Stereum hirsutum (Willd. ex Fr.), in vineyards near Anjou. This was confirmed by Viala (1926), who admitted that F. igniarius could attack grapevines, but he considered such an event only exceptional. According to him, the main pathogen was S. hirsutum, or a new species that he named S. necator Viala. Establishing this species was based on what was believed to be parasitic specialization and a few morphologic characters. But again, no conclusive pathogenicity tests were carried out.

The research conducted by Petri in Sicily (1912) was not specifically directed to esca. Petri was concerned with a vine decline problem following phylloxera invasion and replanting with American rootstocks. Petri observed extensive browning of the vascular system of declining vines accompanied by abundant gum formation in woody tissues. From the brown streaks he consistently isolated two species of Cephalosporium and one of Acremonium. All three fungi proved pathogenic in inoculation tests, producing the brown wood streaks. While he considered these organisms as weak pathogens, Petri noted that they were important factors of
wood alteration around and farther away from wounds.

The second period of esca research started with Hewitt (1957) in California. After conducting a six-year study in a commercial 'Red Malaga' vineyard, recording black measles occurrence on individual vines, Hewitt concluded that only very few vines had symptoms every year for the whole period. Instead, most vines exhibited symptoms without continuity, often also changing from leaf to fruit symptoms and vice versa. Hewitt's systematic study and knowledge of the disease history of each individual vine offered an ideal opportunity for testing his hypothesis of toxin production by soil fungi. Later on, the same 'Red Malaga' vineyard was used by Chiarappa (1959) to establish a correlation between internal wood decay and measles (Fig. 1). Several different fungi were isolated from decayed grape tissues. Of these, only *F. igniarius*, later renamed *Phellinus igniarius* (L. ex Fr.) Quélet, and a *Cephalosporium* sp. were consistently isolated, the latter being the most frequent (Table 1).

A total of 64 inoculations of this undetermined species of *Cephalosporium* into the healthy wood of bearing vines resulted in the production of dark brown striations extending 10-15 cm from the site of inoculation. Re-isolations from these dark areas yielded the same fungus as that used for inoculum. However, tissue darkening appeared to form also at some distance from the fungus mycelium. Chiarappa concluded that the *Cephalosporium* sp. did not behave merely as a secondary organism but had some function in the decay process and in the black measles syndrome. As for *P. igniarius*, it produced typical spongy decay *in vitro* on sterilized grape sawdust and wood disks. In subsequent work, Chiarappa (1997) demonstrated that field inoculations of young bearing vines were successful in reproducing *in vivo* the same type of deterioration. Similar findings were reported from Italy by Mugnai *et al.* (1996c).

The third period of esca research initiated with the work of Larignon and Dubos (1987). These authors suggested that esca was caused by a succession of fungi in diseased vines following initial colonization by *Cephalosporium* sp. and *Eutypa lata* (Pers. Fr.) Tul. & C. Tul.

During the following years research on trunk diseases of grapevine expanded to Italy, South Africa and California. Two reasons probably explained this resurgence of interest. The first was the banning or restriction in use of sodium arsenite for the control of esca and the consequent increase of the disease. The second was the discovery of grapevine decline in many vineyards, especially those where replanting had taken place following phylloxera damage. Brown wood streaking was found to be associated with this decline.

Larignon (1991) observed discoloration in wood of esca-affected vines and recorded differences in the total amount of phenols in grapevine wood at various stages of decay. Three years later Ferreira *et al.* (1994) associated *Phialophora parasitica* Ajello, L.K. Georg & C.J.K. Wang with slow decline of grapevines in South Africa. This was followed by Morton (1995) who uncovered widespread production of a gummy brown sap in young declining vines. For lack of better term, she called this condition "black goo."

In the meantime, significant advances were

---

**Table 1. Comparison of the isolation frequencies of the fungi from esca diseased vines in California, Italy and France. Isolations in average percentages.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Cephalosporium&quot;a</td>
<td>59.2</td>
<td>68.1c</td>
<td>23.6c</td>
</tr>
<tr>
<td>&quot;F. igniarius&quot;b</td>
<td>14.4</td>
<td>61.5d</td>
<td>33.3d</td>
</tr>
<tr>
<td><em>S. hirsutum</em></td>
<td>5.1</td>
<td>-</td>
<td>5.0</td>
</tr>
</tbody>
</table>

*a* Includes strains identified as *Phaeoacremonium chlamydosporum*.

*b* Includes strains identified as *Phellinus igniarius* and *Phellinus punctatus*.

*c* Brown-red wood.

*d* Decayed wood.
Fig. 1. Leaf, fruit and internal decay symptoms of black measles. Lower right, *Phellinus igniarius*, the cause of spongy decay, surrounded by abundant gum formation.

Fig. 2. 139-year-old ‘Shiraz’ vines at Chateau Tabilk, Tabilk, Victoria, Australia. Vines are healthy and still in full production. (Photo courtesy John Mac Cready, Sierra Vista winery, Placerville, CA, USA)
made by Muginai et al. (1996 a, b, c) and Larignon and Dubos (1997) in the isolation and identification of fungi associated with internal wood decay. Their frequencies of isolation, when compared with those of Chiarappa (1959), show a similar pattern (Table 1). In 1996 Crous et al. proposed the establishment of the new genus *Phaeoacremonium* with six species that included grape isolates of *Cephalosporium*, *Acremonium* and *Phialophora parasitica*. The California *Cephalosporium* isolate by Chiarappa (CBS 239.74) was named *P. chlamydosporum*. Du Pont et al. in 1998 showed by molecular biology techniques that *P. chlamydosporum* and *P. aleophilum* were two fungi belonging to different genera.

Progress on the mode of spread of these and other mitosporic fungi have been made during the last two years. Bertelli et al. (1998) observed that rooted cuttings of ‘Sangiovese’ grafted on 1103P rootstock and ready to be planted had dark striations in the wood. These striations were most apparent near the grafting area where *P. chlamydosporum* was most frequently isolated. The hypothesis these authors suggested was that this pathogen came from mother vines already harboring latent infections or entered the cuttings through cuts and wounds during grafting and calusing. Larignon (1999) in spore-trapping epidemiological studies found that both *P. aleophilum* and *P. chlamydosporum* were airborne, that they were both present in the canes before pruning, and that *P. chlamydosporum* also penetrated vine canes through pruning wounds following rainfall.

Relevant to the young vine decline syndrome is the report by Scheck et al. (1998) giving the results of *P. chlamydosporum* inoculations in self-rooted ‘Chardonnay’ vines. After growing in the field for two and half years these vines showed significant growth reduction and discoloration of the pith and vascular elements. The pathogenicity of other species of *Phaeoacremonium* recovered from diseased rootstocks in California still remains to be confirmed.

In conclusion, it is clear that vineyards in Europe, South Africa and California have experienced crippling diseases which have dramatically shortened their productive life and increased management costs. Porter (1999) gives two examples from the North Coast region of California where vineyards had to be uprooted only six years after planting, or they had to be grafted three times for lack of take. This situation is of great concern to the grape industry.

This first international workshop and the establishment of the International Council on Grapevine Trunk Diseases (ICGTD) are an attempt to improve the health, productivity and longevity of vineyards. That this is possible is demonstrated by places in the world where vineyards planted in 1860 are still in good production (Fig. 2). This indirectly suggests where priority research should be directed, including a re-examination of many viticultural practices and related pathological problems that have led to short lived, low-yielding grapevines.

**Literature cited**


