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Towards commonly agreed answers to some basic questions on esca

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Summary. The present paper focuses on some basic questions about what we still call esca, such as: What is esca? Is esca a complex of diseases including young esca, white rot and esca proper, while *Pch* causes decline on its own? Is the concept of microbial succession to be retained, and what is its exact meaning? Can *Pch, Pal* and *Fop* be considered primary pathogens? What should the decline associated with *Pch* (*Pal*, *Pin*?) be called? These and other questions will be commented.

Key words: grapevine, esca proper, young esca, brown wood streaking, *Phaeomoniella chlamydospora*.

Research on esca has been divided by Chiarappa (*Phytopathologia Mediterranea* 39, 11–15, 2000) into three time periods, the last of which started in France at the end of the eighties. I think we must give our colleagues Philippe Larignon and Bernadette Dubos credit for renewing research on esca and, most important, for bringing new ideas and information to the debate on the etiology of esca and on its development. New fungi have been discovered and old fungi rediscovered: the fungi that once were known as *Acremonium* sp., *Cephalosporium* sp. or *Phialophora parasitica* are now classified into the new genera *Phaeoacremonium* or *Phaeomoniella* thanks to the work of plant pathologists and taxonomists. On the basis of experimental results, a theory has been formulated explaining the process of esca as due to a succession of micro-organisms. Further research then added more information, and this led to a new theory being proposed: that esca is not a single disease, but a complex of different diseases. After a number of pathogenicity assays, the three most important fungi in esca aetiology, *Phaeomoniella chlamydospora* (*Pch*), *Phaeoacremonium aleophilum* (*Pal*) and *Fomitiporia punctata* (*Fop*), have been elevated to the rank of primary pathogens. The structure of the new genus *Phaeoacremonium* has turned out to be more complex than originally suspected. A new syndrome of grape has also been reported, first in South Africa, then in the USA and elsewhere: grapevine decline or "black goo". We now know more about the epidemiology of *Pch*, *Pal* and *Fop*, their sources of inoculum, and their spread, and we are learning something about how to control these fungi. But, despite these unquestionable advances in the study of esca, we must admit that, at this stage, there is still disagreement on the interpretation of some results and their implications in esca, and on the interpretation to be placed on certain findings from surveys in the vineyard.

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The present paper focuses on some basic questions about what we still call esca, questions that arise out of observations made in these last few years and that must now be submitted to a larger group of researchers to see if they can be generally accepted.

The first question is: how can esca be defined today? In other words: what is esca?

We are or were used to considering esca as:

- a disease of mature plants;
- which is characterised by chloro-necrotic symptoms on the leaves and black spots on the berries;
- and which is sometimes accompanied by apoplectic strokes.

For a long time it was believed that the leaf chlorosis and necrosis were caused by *Fomitiporia punctata* (*Fop*), the white-rot fungus in the wood of the trunks. Some researchers postulated that the fungus causing wood rot (*Fop* or other wood-rotting basidiomycetes) produced toxins that accumulated in the leaves and caused the foliar symptoms.

Starting from the above definition we can ask: what are the peculiar characteristics of esca? In general the following characteristics have been considered to be significant indicators of esca:

- a mature plant age;
- leaf and berry symptoms;
- apoplectic strokes;
- white rot caused by *Fop*.

Can these characteristics still be considered significant in the light of the most recent findings on esca?

Let us consider some of these new findings:

- vine age: the foliar symptoms of esca are now being noted on young (6–12 years) and even very young (2–6 years) vines much more frequently than in the past. The reason for this early onset is unknown;
- leaf symptoms: the expression of these symptoms is strongly characterised by discontinuity from year to year. Reason unknown;
- internal symptoms: the most remarkable is white rot of the wood. However, other internal symptoms also occur: various types of necrosis, brown-red wood, black stripes.

Moreover, surveys in various vineyards in Italy and other countries have revealed different phytopathological situations:

- some young plants colonised by *Pch*/*Pal* show black stripes in the wood and also foliar symptoms, though not white rot. This suggests that the white rot agent *Fop* is not necessary for foliar symptoms to occur or at least, that *Pch*/*Pal* can cause foliar symptoms without *Fop*;
- some young plants with *Fop* show white rot and foliar symptoms, though not the black stripes normally associated with *Pch*/*Pal*. This suggests that the agents causing black stripes (*Pch*/*Pal*) do not cause foliar symptoms, or at least, that *Fop* is here causing the foliar symptoms as well as white rot;
- some young plants have only black stripes and no foliar symptoms. This suggests that black stripes are not a disease symptom and consequently that *Pch* is not a disease agent at all; but it could also be simply a consequence of the known discontinuity in foliar symptoms that is a characteristic of esca;
- some young plants have only white rot and no foliar symptoms. This may suggest that *Fop* causes only white rot but not foliar symptoms; but again, as above, it could simply be due to seasonal discontinuity in foliar symptom expression: these young vines happened to be without visual symptoms when they were examined;
- some young vines with symptoms of decline and black stripes in the wood are colonised by *Pch*/*Pal*;
- some rooted cuttings with black stripes in the wood are colonised by *Pch*/*Pal*;
- the canes of some adult plants are colonised by *Pch*.

What does this complicated picture suggest? A number of possibilities:

- foliar symptoms are probably determined by environmental parameters as well as by the fungus or fungi that colonise the wood, and physiological factors within the plant;
- foliar symptoms do not indicate the exact disease status of a vine. When a vine has foliar symptoms we cannot be sure which fungus or fungi have actually colonised the wood;
- the theory of microbial succession is useful but is not strictly necessary to explain how esca develops over time.

In this context we can say that the traditional definition of esca is no longer tenable.

What, then, is esca?

In a way, "esca" as such does not exist. In its place we find a number of different syndromes:

- brown-wood streaking of rooted cuttings (this could simply be the start of decline, and not a specific disease);
- decline of young vines (variously named: black goo, *Phaeoacremonium/Phaeomoniella* grapevine decline, young grapevine decline, Petri grapevine decline, etc.);
- young esca;
- white rot;
- esca proper, the classical disease affecting generally mature vines, in which different syndromes are still completing or have already completed their evolution.

In that case esca proper would be the last step in a series of pathological events; this series starts in the nursery, continues in the young vineyard and is completed when the plant is mature. Each of these events is determined by:

- a particular fungus or a combination of fungi;
- environmental factors (rain, temperature, etc.);
- the particular physiological status of the plant.

This assumption relies on the fact that *Pch* and *Fop* are primary pathogens, i. e. they attack the vines, invade the tissues and cause disease symptoms without the help of other organisms.

Are there still doubts about the primary role of *Pch* and *Fop*?

Are pathogenicity tests on *Pch* and *Fop,* carried out in many laboratories and on vines of various ages valid?

Are studies on the biology of *Pch, Pal* and *Fop* valid? These studies show that:

- *Pch* produces at least two classes of virulence factors: phytotoxic pullulans and pectic enzymes;
- *Pal* produces smaller amounts of pullulans, phytotoxic metabolites (scytalone and isosclerone) and pectic enzymes;
- *Fop* produces phytotoxic pullulans and wood-degrading enzymes;
- *Pal* antagonises *Fop in vitro* and *in vivo*.

On the other hand we must also remember that:

- *Pch* is not always isolated from black stripes;
- rooted cuttings with *Pch* do not always become vines with decline.

I will now outline how, according to some of us (G. Surico, L. Mugnai and A. Graniti), esca develops from the nursery to the mature vineyard (Fig. 1). This model incorporates almost all that has been discovered in the last few years about the epidemiology and biology of the fungi involved in esca, but first and foremost it is based on an assumption, namely that *Pch* and *Fop* are primary pathogens; and on a series of findings: that *Pch* occurs in the canes of mature plants; that the spores of *Fop* are mainly introduced from outside the vineyard; and that *Pch* has its sources of inoculum on the host.

According to this model, the reason why rooted cuttings become infected by *Pch* and/or *Pal* is that: 1. cuttings are already infected when they are taken from the mother plants or, 2. the infection started in the nursery. The infected rooted cuttings so obtained will sometimes, though not always, give rise to young vines with decline or young esca. The particular syndrome shown by these young vines will depend on a number of factors: the fungus or fungi involved, the vegetative vigour of the plant, the cultivar, the rootstock and factors external to the plant (soil, temperature, water, etc.).

Of course, healthy propagating material grown under the appropriate sanitary conditions will produce healthy young vines. In that case, any infections that arise will occur later, mainly through pruning wounds. Depending on the fungus or fungi involved, the young plant will then become affected by: 1. decline; 2. young esca, or 3. white rot. The next step after this will be additional infection with *Fop*, for those vines that already suffer from decline or young esca, or infection with *Pch*/*Pal* for vines that already have white rot. In both cases the result will be esca proper. Alternatively, if a healthy young vine escapes one or another of these three syndromes, it will go directly toward esca proper after infection with first *Pch*/*Pal* and then *Fop*; or first *Fop* and then *Pch*/*Pal,* or *Pch*/*Pal* and *Fop* simultaneously.

Conclusions

Despite the many advances in the studies on esca, many questions remain. This article has tried to answer some of the questions, or at least to place them in their right context; other questions will be dealt with in some of the articles in the current issue of *Phytopathologia Mediterranea*. Finally, participants at the 2nd International Workshop on

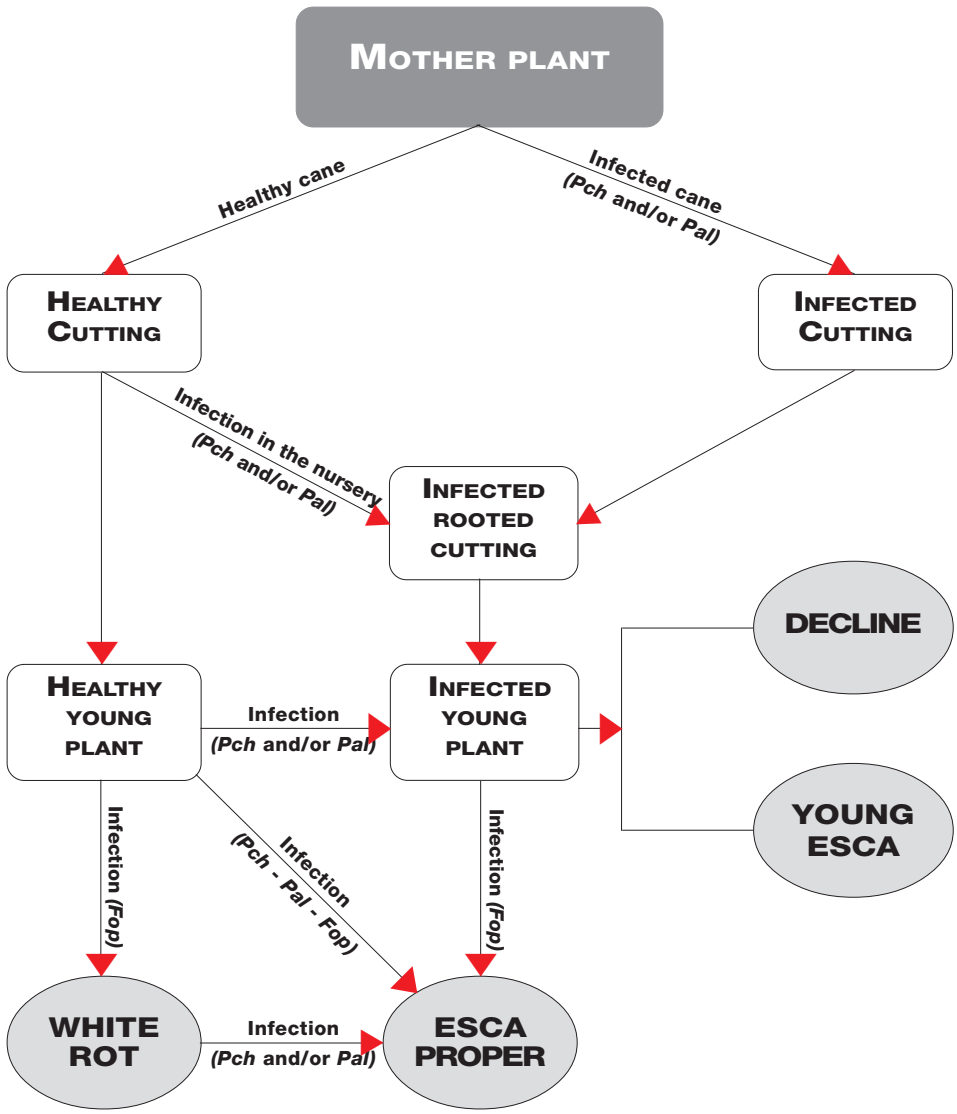

Fig. 1. Proposed scheme of the development of esca and related syndromes.

Esca and Grapevine Diseases are invited to express themselves on three points in particular:

1. the name to be given to the decline of young vines caused by *Phaeomoniella chlamydospora*;
2. the position of *P. chlamydospora*, *P. aleophilum* and *F. punctata* as primary pathogens;
3. the proposed development model of esca proper and its related syndromes;

Lastly, it is superfluous to underline the importance of not diminishing the attention given to the study of esca. Because of its capacity to reduce yield, and its negative impact on the grapevine landscape, esca deserves a concerted effort so that we can protect our grapevines from one of the most serious diseases to affect it. It is high time to devise effective measures to control esca proper and its related syndromes. This is what viticulturists are expecting from us, and we must not disappoint them.