The Present Status of Chestnut Blight in Italy

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ABSTRACT.— Chestnut blight was first noticed in Italy in 1938. The disease spread rapidly throughout the country. Since 1950 healing cankers have been found in all areas having diseased trees for 15 years or longer. Because of the natural spread of the hypovirulent strains of the fungus, healing cankers are now prevalent in all the chestnut stands so that the disease is no longer a problem in the cultivation of chestnut in Italy.

MAIN FEATURES OF CHESTNUT CULTURE IN ITALY

Italy is the most important chestnut growing country in the world with its 680,000 hectares of chestnut and an annual production of more than 60,000 metric tons of nuts. European chestnut *(Castanea sativa* Mill.) is naturally found in a wide area of southern Europe surrounding the Mediterranean Sea. It needs a moderately humid and warm Mediterranean-type climate, and a moderately acid and rich soil. Chestnut is located in the Piedmont zone of the Alps in north Italy and in the middle-mountain zone of the Apennines in central and southern Italy ranging from an elevation of 400 to 1,300 meters.

The European chestnut is mainly a fruit tree because of its valuable nuts and the tendency for early branching of the trunk. Wood products are also obtained from the European chestnut, including poles, ties, beams, lumber for construction and furniture manufacture. Chestnut is also used as a source of tannin. It ranks second only to poplar among the broad-leafed species in Italy as a source of lumber. It is usually grown in pure stands utilizing the coppice technique rather than as a high forest.

As a fruit tree, chestnut has been very important in Italy for centuries. Chestnut flour has been a staple food material for many mountain populations. The tree has about 300 uses including flour, fire wood, food for cattle and leaves for litter. The pure stands are excellent grazing ground.

Since the first quarter of this century, chestnut stands have been neglected and the number of stands has been reduced because of the decrease in the mountain population and because of damage caused by the blight (Table 1).

At present there are good prospects for new development of chestnut culture on the better sites and with high value cultivars.

The first 15 years of blight epidemic

Chestnut blight was first noticed in Italy in 1938 near Genoa, 34 years after the discovery of the disease in North America. In 1940 the disease was found in the Udine district and in 1943 in the Avellino district. It is very likely that the presence of chestnut blight in Italy occurred before 1934 because large branches found on chestnut trees in Tuscany appear to be associated with blight attacks (Biraghi, 1966).

The development of the disease was followed by Professor Biraghi of the University of Florence until 1965. We are indebted to him for his studies and observations. Biraghi (1966) stated that in comparison with the American epidemic, the Italian disease spreads slower and requires a much longer period of time for the pathogen to kill fully grown trees. Ten years after the discovery of the disease, approximately 10 percent of the chestnut area in

Table 1
Areas in chestnut plantations and fruit production in Italy during the last 40 years (data from Bolletine Mensile di Statistica, Roma).

	Year		
Forest type	1935	1955	1974
High forest	483,000[a]	440,000	324,000
Coppice	316,000[a]	292,000	359,000
Total	799,000	732,000	683,000
	1933-35	1953-55	1972-74
Fruit Production	340,000[b]	237,000	63,500

[a]In hectares.
[b]In metric tons, mean of three years.

Italy was diseased (Biraghi, 1950). Thirty years later nearly all the country was affected by the disease, but in some provinces spread was limited (Buccianti and Feliciani, 1966). In the majority of cases, spreading of the diseases was rapid, but in many cases it was strangely slow. A positive correlation was found between vigor and intensity of the disease.

In order to curb the spread of the disease, sanitation was attempted, but as in the United States, the attempts failed. Many high forests attacked by blight were cut at main branch or at ground level. Therefore, after World War II there was an increase of the chestnut coppice area. In a few cases severely damaged chestnut stands were planted with other fast growing species. In other cases, stands were naturally invaded by new species.

In 1950, 12 years after the discovery of the disease, a very important phenomenon was noticed in the area of the earliest infections near Genoa. Healing cankers were found along with typical virulent cankers. Even though branches were girdled by the healing cankers, they had normal vegetative growth. This same phenomenon was soon observed in other areas of early infection. In reporting this at an International Congress, Biraghi noted that the healing cankers had appeared only in areas of early infection, i.e., approximately 15 years after the onset of the disease (Biraghi, 1953).

One of the hypotheses put forward by Biraghi to explain the appearance of healing cankers was the loss of pathogenicity by the fungus. This hypothesis was supported by Grente (1965) in identifying a "hypovirulent" white strain of *Endothia parasitica* (Murr.) P. J. & H. W. And. from healing cankers in the province of Como, north Italy. More types of hypovirulent strains were also obtained by Bonifacio and Turchetti (1972) from diseased chestnut stands in central Italy. Hypovirulent strains show a reduced pathogenicity which can be overcome by the reaction of the host so that the canker heals. Hypovirulence is transmitted by hyphal anastomosis to virulent strains of *Endothia* which in turn lose their virulence.

Generally, healing cankers are characterized by more or less pronounced swelling of the bark which is not killed and by a low pycnidia production, which stops quickly. By slicing the bark away it is possible to observe that the mycelium is confined to the outer layers of the bark and that very often it is not organized into compact fans as in normal virulent cankers.

Basically we can distinguish two kinds of healing cankers in nature. The first type is that of a very superficial canker in which the fungus spreads longitudinally over a large vertical portion of the stem, the bark is slightly cracked, looks rough and is dark in color. The mycelium is superficial, rather abundant in the front zone of the advance of the canker, more and more scant in the older zones of the canker, until it is completely corked out in the oldest ones. In most cases, neither fructifications of *Endothia* nor development of epicormic shoots occur below the canker. It seems quite feasible that this kind of healing canker is caused by some kind of hypovirulent strain of *Endothia.* The second type of healing canker starts as a normal virulent canker and kills the inner bark and covers the sapwood with abundant mycelial mats of fungus. Vigorous wound cork barriers are found in the reactive swollen zone that encircles the killed one. Epicormic shoots below the canker occur normally along with fructifications of the fungus in the central zone. Apparently this type of healing canker, which started as virulent, lost virulence because of subsequent infection by a hypovirulent strain. Also, healing cankers occur in which penetration of the fungus, abundance of mycelium, pycnidial fructification and reaction of the host are intermediate between those of the two types described.

Spreading of hypovirulence

Fifteen years after the discovery that healing cankers were prevalent in the coppices of old infection areas, blight ceased to be a problem. The disease was stopped at an early stage and the pathogen seldom reached deeper layers of the bark. Fully grown trees that were not cut after the first heavy

attacks by blight showed healing cankers and began to produce new branches in the crown.

Experimentally, hypovirulence can be spread by inoculating normal virulent cankers with cultures of hypovirulent strains. Very often hypovirulence is passed on to the virulent strains and the canker heals. Grente (1975), who discovered the phenomenon, proposes that the biological control of blight is possible by means of artificial inoculation.

How hypovirulent strains spread in nature is not clear. In fact, one of the most important features of the healing cankers is low production of pycnidia which stops in a short time. If we keep in mind that some of the conidia born by hypovirulent strains give rise to virulent strains, we realize that spread by means of conidia may favor normal strains which fruit abundantly in normal cankers. On artificial media some hypovirulent strains bear only a few pycnidia, but others bear as many conidia as the virulent strains.

One of the possible explanations for the spread of hypovirulence in nature is that hypovirulent strains may be more adaptable to natural conditions than the virulent strains in the saprophytic phase. It is known that *E. parasitica* can live as saprophyte on chestnut and on other forest trees (Boyce, 1948; Hepting, 1971), so in this situation it is possible that the less pathogenic strains may be more fitted to survive and spread. Fructifications of *Endothia* are easily found on old cut poles and sometimes on the outer rough bark of old trees, but the question is to find out if we are dealing with hypovirulent strains of *E. parasitica* or with a saprophytic species similar to *E. fluens,* which is very close to *E. parasitica.* If this hypothesis is true, then, the saprophytic stage of *E. parasitica* may be a way to preserve chestnut and to control the development of epidemics.

Present situation of the blight

Except for small experimental plots no chemical treatment or biological control has been carried on in diseased chestnut groves in Italy. But, in several cases where heavy attack occurred, chestnut trees were cut at ground level so that they would sprout from the stumps. The evolution of chestnut blight in Italy has been natural.

The present situation is a general decrease in disease incidence, i.e., the majority of cankers are reactive and only a few branches are killed by the fungus. Now only weak sprouts, instead of vigorous ones as at the beginning, are killed and groves on poor sites or under adverse conditions are damaged more severely. We experimentally confirmed this observation by inoculating several sprouts of one coppice with one strain of *E. parasitica.* Sometimes we observed that the weakest sprouts of the coppice were killed while the strongest ones would recover.

Following many surveys throughout Italy, I concluded that the presence of the healing cankers and hypovirulent strains were everywhere. Fungus isolation from cankers seldom resulted in identifying pure virulent or hypovirulent strains but strains were found with intermediate features that can be purified by monoconidial subculturing.

At present there are only a few places in the country where damage by blight is still severe. In these areas it is difficult to graft successfully because blight kills the twig at the grafting point or along the weak sprout of the scion. I believe that the disease entered the majority of these areas during the last 15 years. Therefore, I believe that hypovirulent strains have not had enough time to curb the virulent cankers. In a survey of two valleys in the Piedmont in north Italy only active cankers have been found in the upper part of the valley where the disease entered late while the great majority of cankers were healing in the lower part of the valley (Mario Palenzona, personal communication).

Based on a survey in the old diseased stands near Genoa, Udine and Avellino, the incidence of the disease in some cases is very low, but it is still present. In these areas, I have found that from 1 to 20 percent of the group in six-year-old coppices cankered, and from 10 to 30 percent cankered in 10-year-old coppices. In 15-year-old coppices I could count up to 100 percent of the stems cankered, but only 10 to 20 percent were active. Also, I must say that I encountered cases of very low incidence of the disease. For example, in a limited zone of the province of Cuneo, north Italy, it is difficult to find cankers whether active or healing on the huge crowns of the cultivar "della Madonna." This suggests that host susceptibility and environment also affect resulting disease. At the beginning of the epidemic, Biraghi (1950) did not find clear relationships between these two sides of the disease triangle. Now we have indication that some varieties are not as susceptible to attack as others. This fact may be explained if we assume that the epidemic at its peak obscured varietal and environmental differences that now are evident because of the loss of virulence by the pathogen.

It is difficult to foresee whether the disease will be eliminated, or if it will find a balance at a low level of incidence. The second possibility would seem to be likely because of the surveys carried out on the old diseased areas 40 years after the onset of the disease and 25 years after the appearance of hypovirulent strains.

The resistance level of the American chestnut to chestnut blight does not seem to be very different from that of the European chestnut. Therefore I would be optimistic about the evolution of the epidemic in America following the introduction of the hypovirulent stage of the pathogen now existing in Europe.

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