INVITED REVIEW

CANKER STAIN OF PLANE TREES: A SERIOUS DANGER TO URBAN PLANTINGS IN EUROPE

A. Panconesi

Istituto per la Patologia degli Alberi Forestali del C.N.R.
c/o Istituto di Patologia e Zoologia Forestale e Agraria, Università di Firenze, P.le delle Cascine 28, I-50144 Firenze, Italy

SUMMARY

A brief synthesis is given of what is known about canker stain of plane trees caused by *Ceratocystis fimbriata* f.sp. *platani.* This knowledge mainly concerns the morphology, taxonomy and biology of the fungus and relations between host and parasite. The epidemiological data highlight the destructiveness of the disease and its potential danger to all street plantings of *Platanus acerifolia* in Europe. In the matter of control the effectiveness of chemical and biological control measures is underlined, as well as the serious difficulties encountered in genetic improvement, owing mainly to the genetic uniformity of the host tree and the great variability of the pathogen. Prevention, which in Italy has now been made a legal requirement, also has not produced the desired results because of technical difficulties in raising and tending street trees and the problem of finding funds quickly when an insect pest or an epidemic strikes. Nevertheless, prevention, if carried out systematically and rigorously, can reduce the incidence of canker stain in infection centres and prevent its spread to unaffected areas. Meanwhile genetic improvement may succeed in producing *C. fimbriata* resistant individuals of the plane tree that are adapted to European environmental conditions.

RIASSUNTO

IL CANCRO COLORATO DEL PLATANO: UN PERICOLO CHE MINACCIA LE ALBERATURE EUROPEE. Viene fatta una breve sintesi delle conoscenze finora acquisite sul cancro colorato del platano causato da *Ceratocystis fimbriata* f.sp. *platani*. Queste riguardano principalmente la sistematica (morfologia e tassonomia), la biologia e i rapporti fra ospite e parassita. I dati epidemiologici rilevati hanno permesso di mettere in risalto le capacità distruttive della malattia e di sottolineare la potenziale pericolosità per tutte le alberature di *Platanus acerifolia* del continente europeo. Per quanto riguarda i mezzi di lotta vengono evidenziate l'inadeguatezza dei mezzi chimici e biologici e le grandi difficoltà che si incontrano nel miglioramento genetico, dovute principalmente all'omogeneità dell'ospite alla quale si contrappone una grande variabilità del patogeno. Anche la lotta preventiva, benché in Italia sia stata resa obbligatoria, non ha dato i risultati sperati. Ciò è dovuto alle difficoltà tecniche insite nel sistema di allevamento e manutenzione delle alberature cittadine, nel coordinamento delle varie competenze territoriali, nonché nelle difficoltà di reperimento immediato di fondi da impiegare nelle infestazioni e nelle malattie di natura epidemica. Comunque, quando la lotta profilattica viene condotta con metodo, con rigore e nel quadro di un'azione generale concertata, si può gradualmente ridurre l'incidenza della malattia nelle zone infette e impedire al parassita di contaminare le zone ancora indenni. Ciò in attesa che il miglioramento genetico riesca a selezionare dei platani resistenti alla *C. fimbriata* e adatti alle condizioni pedoclimatiche del continente europeo.

Key words: plane tree, *Ceratocystis fimbriata* f.sp. *platani,* canker stain.

Correspond to: A. Panconesi
Fax: +39.055.354786
E-mail: ragazzi@ipaf.fi.cnr.it

HISTORY AND SPREAD

ORIGINS AND SPREAD IN THE USA. The first confirmed report of canker stain was in 1935 on some oriental plane trees (*Platanus orientalis* L.) in Delaware County, PA, USA (Crandal, 1935; Jackson and Sleet, 1935) even though the disease seems to have occurred in street-tree plantings of Gloucester, NJ, since perhaps 1926 (Walter, 1946). However, it is now thought that the tree species first affected by the disease was not oriental plane but rather London plane (*Platanus acerifolia* (Ait.) Willd. = *P.* x *hispanica* Muenchh. = *P. cuneata* Willd. = *P. hybrida* Brot.), one of the most common trees in large cities along the Atlantic coast, from Canada to Florida (Marvin, 1939). The extensive use of London plane for urban plantings is due to its vigour, its rapid growth, its

resistance to pruning, pollution, and, not least, anthracnose (*Gnomonia platani* Kleb.), a disease to which the native plane tree, or sycamore (*Platanus occidentalis* L.), is very susceptible.

From the mid-1920's to the early 1940's canker stain spread to the larger cities of the East Coast, causing very serious losses to urban plantings of *P. acerifolia.* Walter *et al.* (1952) call the disease 'highly infectious' and write: 'The literature on tree diseases presents very few definite statements concerning the destructiveness of otherwise well-known contagious pathogens. So far as the writers are aware, it affords no record of mortality rates that could be classified with canker stain on the basis of transmission of the causal organism'.

Canker stain is probably indigenous to the forests of North America, where it is not very dangerous because it seems to have few natural means of diffusion, and because the native sycamore, as will be seen, is fairly resistant to it, perhaps as a result of selection pressure exerted by the pathogen in the past. The disease began to spread in epidemic fashion only when the parasite was introduced into an anthropogenically stressed environment on a species such as *P. acerifolia* that is both susceptible and used intensively in urban tree plantings. By 1949 in one of the earliest centres of infection, where the disease had arisen in the mid-twenties, 87.1% of the original plane trees had already died and an additional 4.3% were infected (Walter *et al.,* 1952). In the second half of the 1970's canker stain was still active on *P. acerifolia* and had also begun to make a worrying appearance in some natural forests in Arkansas (McCracken and Burkhardt, 1977).

SPREAD IN ITALY. In the 1940's canker stain therefore ravaged urban plantings of plane trees in the larger American cities along the Atlantic coast (Walter, 1946). During the second World War wood from infected trees was used to package materials for the war effort, and this is probably how the pathogen was introduced to the European continent, giving rise to the processes of infection there (Panconesi, 1972; Cristinzio *et al.,* 1973; Panconesi, 1973). This supposition is borne out by the fact that the first centres of infection in Europe were in or near the major port cities: Naples, Livorno, Syracuse, Marseilles and Barcelona, which were very active during and after the second World War. It is likely that at first only one of these ports became 'infected' with the parasite from overseas, and that from this single point it spread out to other Mediterranean port cities. The first epicentre of the disease in Italy is thought to have been the Naples-Caserta area (Cristinzio *et al.,* 1973); this hypothesis is confirmed by the fact that, when the disease was detected for the first time at Forte dei Marmi in Tuscany (Panconesi, 1972), it had already killed many of the 200 year-old planes lining the Vialone Carlo III, the broad avenue that leads to the Palace of Caserta.

Canker stain did not assume an epidemic pattern until several years later, but then it spread in a dramatic manner through Italian cities, as it had done in North America before. A survey from Forte dei Marmi revealed that in the 20 year period 1972-1991, 90% of all planes there became infected and died. Subsequently the disease spread out from Forte dei Marmi to much of Versilia and to many towns of Tuscany: Massa Carrara, Lucca, Pisa, Livorno and Florence (Panconesi, 1986). At the same time it also made its appearance in the Padua area (1975) and Verona (1978), and somewhat later spread from Udine to Trieste (Mutto Accordi, 1987). In Lombardy the first report of canker stain was from the Park of Monza (1980), after which it spread throughout the whole Region of Lombardy, from Valtellina (Sondrio) to the Lake area (Varese) (Gervasini, 1996), and from there it passed to nearby Switzerland (Canton Ticino). In Piedmont the disease was first reported in Turin (1980), from where it spread to almost all the other provinces of that Region (Cravero *et al.,* 1992; Gianetti, 1996). In Emilia Romagna centres of infection have now been reported at Piacenza, Reggio Emilia, Modena, Ferrara, Parma (Marchetti and Vai, 1996), and more recently also in Bologna. In Campania after the initial reports of 1973 the parasite spread to the areas around Caserta, Avellino and Salerno. After a first report some years ago, there has also been a worrying resurgence of the disease in Rome (Biocca and Annesi, 1996). Lastly, serious canker stain infections have been noted on the oriental planes that regenerate along the banks of streams in the Syracuse area (Granata and Guastella, 1986). In these areas the occurrence of the disease is unexpected since it was assumed that the ability of the pathogen to spread in natural forests is limited. Unfortunately the Italian natural riverside forests are under much greater anthropogenic stress than those in the USA: many of those forests have now been transformed into coppices consisting of plane trees with 5-7 suckers each. In 1991 canker stain also appeared in street plantings of the city of Palermo (Sammarco and Torta, 1997).

It is fair to say that canker stain has now spread to the whole of Italy from the Alps to Sicily with the sole exception of Sardinia and some other southern Regions, where however plane trees are uncommon.

SPREAD THROUGH EUROPE. After its detection in Italy (Panconesi, 1972) the parasite was identified on urban plane plantings in other European countries, first

in France (Marseilles), where it is now moving from Provence northwards along the Rhone valley (Avignon and Lyons) (Ferrari and Pichenot, 1974, 1976; Vigouroux, 1986); then in Spain where it has been identified and is spreading to some cities along the Mediterranean coast (Barcelona, Tarragona and Valencia) and further inland (Cuenca) (Fernandez *et al.,* 1977; Ruperez and Muñoz, 1980; Cadahia, 1983); and subsequently also in Switzerland (Canton Ticino), where it probably arrived via Italy. There are also some unconfirmed reports of canker stain in Belgium (Anselmi *et al.,* 1994). The disease has not been detected in Germany or Portugal, at least until now (Wulf*,* 1995; Levi Pires, 1998). It has however been found in the Erevan region of Armenia (Simonian and Mamikonyan, 1982).

THE CAUSAL PATHOGEN (MORPHOLOGY AND TAXONOMY)

The cause of canker stain of plane trees is the fungus *Ceratocystis fimbriata* Ell. *et* Halsted, f.sp. *platani* Walter (=*Cfp*) (*Ascomycotina, Ophiostomatales*). This *forma specialis* is adapted only to planes, whereas other *formae*, though morphologically indistinguishable from it, attack other hosts such as sweet potato (*Hypomaea batatas*), coffee (*Coffea arabica*), cocoa (*Theobroma cacao*), the rubber-tree (*Hevea brasiliensis*), aspen (*Populus tremuloides*) etc., but not planes (Walter, 1946; Webster and Butler, 1967a).

TELEOMORPH (PERITHECIA AND ASCOSPORES). The sexual stage of *Ceratocystis* is characterised by perithecia that are dark-brown in colour, superficial or partly immersed in the substrate, with a globose base (120-330 µm), adorned with hyphal filaments (sometimes consisting of the conidiophores themselves) and provided with a long neck (400-1000 µm) that is somewhat dark and wide at the base, lighter and narrower at the tip. The tip of the neck is adorned with erect hyaline hyphae (48-102 µm) among which is the ostiole from which the ascospores are expelled at maturity. The asci form and disintegrate very early; for this reason the organ that was presumed to be a pycnidium (*Sphaeronema fimbriatum*) was actually a perithecium with evanescent asci, and the species was accordingly named *Ceratostomella fimbriata* (Elliott, 1923, 1925). Successively *Ceratostomella* was transferred to *Ophiostoma* H. and P. Sydow by Melin and Nannfeldt (1934) and to *Endoconidiophora* Munch. by Davidson (1935). Finally Bakshi (1950) and Hunt (1956) revised the generic name *Ceratocystis* and considered *Endoconidiophora* and *Ophiostoma* to be synonyms, while *Ceratostomella* Sacc. (*sensu* Höhne) was excluded since the genus comprised only species with persistent asci. *Cfp* ascospores (3.5-8 x 2.5-6 µm) lack a germ pore, are elliptical in shape, flattened at one end, and are coated in the mass with a continuous gelatinous sheath that gives them a characteristic hat-shaped appearance when viewed in profile. The ascospores are contained in a water-repellent mucilaginous matrix, yellowish-white in colour, which emerges from the body of the perithecium through the neck and is deposited among the ostiolary hyphae. Because the spore mass is higher than its substrate, spore dissemination is favoured, especially by insect vectors. The perithecia form abundantly on pruning cuts made on infected organs, or on wood cut to size and placed in storage prior to use. More rarely the perithecia develop on infected wood exposed due to cracking of the bark following death of the underlying cambial tissue.

ANAMORPH (*CHALARA* STATES). The genus *Ophiostoma* H. *et* P. Sydow was separated from the genus *Ceratocystis* Ell. *et* Halst. *sensu lato* and established as a distinct genus on the basis of its tolerance to cycloheximide (Harrington, 1981), and differences in the chemical composition of the cell walls (Smith *et al.,* 1967; Spencer and Gorin, 1971; Jevell, 1974; Wejman and De Hoog, 1975), in biological adaptation (Dowding, 1984) and in the morphology of the conidial stages (Nag Ray and Kendrik, 1975; Upadhyay and Kendrik, 1975). In addition, while conidiogenesis is essentially holoblastic in *Ophiostoma*, as it is also in the conidial stages of *Graphium* Corda, *Leptographium* Lag *et* Melin, *Pesotum* Crone and Shocknet and *Sporotrix* Hekt *et* Perkins, in *Ceratocystis* it is phyalidic and is to be referred to the anamorphic state of *Chalara* (Corda) Rabenh. (De Hoog, 1974; Von Arx, 1974; De Hoog and Scheffer, 1984; Harrington, 1987). The further separation of a genus *Ceratocystiopsis* Upad. *et* Kend. from *Ophiostoma,* purely on the basis of their falcate, sheathed ascospores has not been accepted by all researchers. Recent studies have suggested that these falcate sheaths in some species of *Ceratocystiopsis* may represent vestiges of an ascomatal centrum and not distinct sheaths (Wingfield, 1993).

After these subdivisions only a few pathologically important species remain in the genus *Ceratocystis*: *C. fimbriata, C. paradoxa* (Dade) C. Moreau, and *C. fagacearum* Bretz. In *C. fimbriata*, at the anamorphic *Chalara* state, the presence of three types of agamic spores is generally recognised (Davidson, 1935), though not by all researchers (Andrus and Harter, 1933; Hunt, 1956); these spores are: (*i*) cylindrical endoconidia, hyaline, with truncated ends that vary in length (8-43 µm). This variation is probably due to the failure of one or

more septa to form during the process of conidia formation (plasmolysis) in the conidiophore hypha; (*ii*) doliform or barrel-shaped endoconidia, light brown with truncated but lightly rounded tips, 6-17 µm long; (*iii*) thick or double-walled endoconidia, globose or ovoid, more or less dark olive-brown in colour and 9-19 µm in diameter. Andrus and Harter, (1933) state that only the first abstracted conidia are double-walled; conidia produced later are single-walled.

As regards the conidiophores, some researchers think that there is only one type with variable morphology, others believe that each type of conidiospore is formed by its own particular conidiophore. It has been shown that the shape of the conidiophores, as well as that of the cell wall, is changed by age and environmental conditions (Morgan Jones, 1967; Cristinzio *et al.,* 1973; Panconesi, 1973; Nag Ray and Kendrik, 1975; Domsch *et al.,* 1980; Upadhyay, 1981). In young colonies (PDA medium) it is very difficult to find doliform or barrel-shaped endoconidia, but I have sometimes observed that older colonies produced barrel-shaped endoconidia from endoconidiophores producing cylindrical endoconidia when younger. Aging causes widening, lengthening and darkening of the conidiophore, which then begins to produce the shorter, wider, and light-brown barrel-shaped endoconidia.

In the case of *Cfp* it has been found that with few exceptions the three types of conidia produced by the conidiogenous hypha remain united and form long chains**.** This is because the conidial walls are covered by an overlying outside layer that forms a continuous gelatinous sheath which does not dissolve until later (Cole and Samson, 1979). It should be remembered that unlike the ascospores, the three types of phyaloconidia mentioned suspend easily in water. The few exceptions to the conidial types are certain anomalies reported by Marziano (1988) which relate to the holoblastic-type production of some thick-walled conidia: this gives rise to confusion in the systematics of the species. Another type of conidiophore, with chains of pear-shaped hyaline phyaloconidia, slightly different from the other three types, has also been observed (Marziano, 1988; Panconesi, unpublished data).

In nature, conidia form abundantly on cuts made when infected trees are pruned, or on infected wood that is stacked, when they appear in the form of an ash-coloured, powdery layer. An abundance of thick-walled conidia forms inside the xylem vessels; these conidia can remain sound and viable even for a number of years. I have also observed cylindrical endoconidia several times, but never barrel-shaped endoconidia.

CULTURAL ISOLATES AND SPECIES PLASTICITY. Isolates of *Cfp* can be either homothallic or heterothallic (Webster and Butler, 1967a, 1967b). In Italy the self-fertile strain has been found to be most common (this strain has all three endoconidial forms and the sexual form), but the self-sterile protoperithecial strain is also frequent; this strain possesses the three endoconidial forms and primordia of immature, more or less well-developed perithecia called protoperithecia.

Other strains have also been identified (Panconesi, 1979), both in nature and in culture (sectorial mutations). We list the most common: (*i*) self-sterile, possessing only the three endoconidial forms; (*ii*) perithecium-sterile, possessing the three endoconidial forms plus a perithecium which, though morphologically normal, is sterile because it lacks ascospores; (*iii*) protoperithecium self-fertile, which has the endoconidial forms plus a fertile perithecium that varies in diameter and neck-length. The self-sterile strain can be induced to produce perithecia by hybridisation with cylindrical phyaloconidia from the self-fertile strain. When these strains were inoculated, by culture mycelium through wounds, they all killed 2 cm-diameter suckers of the three most common plane species (*P. orientalis, P. occidentalis, P. acerifolia*). In addition to the differentiation between the strains that occurs in nature and in culture, it is worth noting the great plasticity of the conidiogenous cells in response to given stimuli or environmental conditions; this suggests they have considerable ability to be transformed, probably producing new individuals, and indeed new *formae speciales.*

Nevertheless, an analysis of the Italian population of *Cfp* by amplification of minisatellite DNA has revealed the strong genetic uniformity of the electrophoretic profiles here, while with other *formae speciales* of *C. fimbriata* (those attacking poplar and sweet potato) polymorphism was frequent (Granata *et al.,* 1992; Santini and Capretti, 1998).

THE HOST TREE

THE MOST COMMON HOST SPECIES AND THEIR SUSCEPTIBILITY TO *CFP*. The genus *Platanus* is the only genus of the *Platanaceae,* a small family of the northern hemisphere in the order of the Rosales. It comprises about ten species, the most common of which are: *P. orientalis* L., *P. occidentalis* L., and their natural hybrid *P. acerifolia* Ait. (Willd.). While *P. orientalis* and *P. acerifolia* are extremely susceptible to canker stain and are sooner or later killed by the disease, *P. occidentalis* has shown a degree of resistance to it. The other species of this genus are all from North and

Central America (*P. racemosa* Nutt., *P. wrightii* Wats., *P. mexicana* Moric. *et* Gal., *P. oaxacana* Standl., *P. lindeniana* Mart. *et* Gal., and *P. chiapensis* Standl.), except *P. kerrii* (Gagnep.) which comes from Laos and is considered an ancestral species. There are no reports of canker stain infection in natural populations of these other species, nor of any resistance to the disease they might possess, since they have never been tested by artificial means.

THE GENETIC RIGIDITY OF *P. ACERIFOLIA*. In Italy as in other countries of Central and Northern Europe, the most widespread species is *P. acerifolia*, which derives from a natural crossing between *P. orientalis* and *P. occidentalis* which probably occurred for the first time at Oxford in 1670.

Since *P. acerifolia* is a fertile hybrid, genetic segregation occurs in the second generation, so that characteristics can be maintained by using hybrids from the first generation, or, more commonly, by propagation. Unfortunately the misuse of cuttings has introduced a high degree of genetic uniformity into the species and hence lowered variability, with all the negative consequences ensuing therefrom. City plantings usually consist of a large number of individuals reproduced by seed or vegetatively, each individual resembling either of its parent more or less closely, and this can be clearly seen by examining the bark or leaf morphology of the trees lining our city streets.

The paucity of species that make up the genus *Platanus* and the lack of information about their inter-relatedness is a serious obstacle to exploiting the genetic resources of these species in breeding programmes that envisage interspecific crosses.

THE INFECTIVE PROCESS

INVASION. It has been experimentally determined that *Cfp* is not able by itself to overcome the natural barriers that the host tree possesses. However, even a very small wound is enough to allow the mycelium to invade the plant tissue and start the infective process. The parasite can invade the tree at various levels: on the main or secondary branches subject to frequent pruning; on the trunk, always exposed to wounds from various causes, especially near ground-level; and on roots that emerge above-ground, as occurs with trees growing in compacted and anaerobic soils that are frequently encountered in street and city plantings.

SPREAD OF THE INFECTION. The parasite colonises the wound-exposed tissue immediately on contact. One of the consequences, and an indication, though not conclusive proof, that canker stain infection has occurred is that the wound no longer scars over, or does so only in part. Weather permitting, the reproductive structures of the parasite develop on the infected and colonised wounds: first the asexual form (2-3 days), then the sexual form (6-8 days). After the tissue has become necrotic from *Cfp* infection it is immediately colonised in succession by other fungi such as *Pestalotiopsis* sp., *Fusarium* sp., *Dendrochium* sp., *Sphaeropsis* sp., *Asterosporium* sp., which may make it difficult to identify the agent causing the original infection, and indeed the first visible fruiting bodies to appear on dead cortical tissue are from these later saprophytes, and not from *C. fimbriata*.

When the wound has been colonised, the mycelium of the pathogen advances into the conducting tissues of the underlying sapwood, where it develops both longitudinally and tangentially. A single infection can attain a length of 2-2.5 m in a year, and can kill a tree of 30-40 cm diameter in 2-3 years. When the infection is on a young tree, or on a branch in rapid growth, one of the signs of infection is a slightly sunken necrotic area with its longitudinal axis parallel to the growth axis, in the centre of which can be seen the wound through which *C. fimbriata* originally entered the tree tissue. Flanking this necrotic area there often are some more or less round cortical swellings that later crack along their longitudinal axis and produce ephemeral shoots that are killed by the advancing infection.

As the infection progresses the cortical layer gradually becomes necrotic and turns a clear hazel-brown (in some trees a wine-red colour) until it dries up and cracks, leaving fairly regular-shaped rectangular plaques that eventually separate from the wood underneath, which is a dark sooty colour.

As the parasite advances along the conducting vessels of the sapwood (tracheae and tracheids) it encounters the medullary rays which it crosses inwards into the duramen, and outwards to the cambial tissue, which it kills, causing dark spots visible on the outside of the face, that are often lenticular in shape, called 'risorgenze' (necrotic reappearances coming from the inner sapwood). These spots, which can appear at a considerable distance from the original infection site, are particularly noticeable on trees with smooth bark and are a characteristic symptom of canker stain (Panconesi, 1987). They are very important in that they make it possible to diagnose a canker stain infection even when the tree has been invisibly infected through the roots of a neighbouring tree.

Nevertheless the most obvious symptom of the disease is the sudden wilting of a portion of the crown, the

exact proportion of the wilt depending on where the parasite invaded the tree, which is often an area where the vessels around the entire circumference of the tree are blocked by the reaction process (formation of tyloses, gums, etc.). Crown wilting occurs most often in the spring-summer period when the tree has a high water demand which it is however unable to satisfy because of the necrotic impairment of the conducting vessels. As a result the leaves turn yellow and wilt, then turn red-brown, but they do not immediately fall and can easily be distinguished from surrounding healthy leaves. Alternatively upon the resumption of vegetative growth a branch or an entire tree may fail to sprout, or the buds will suddenly wither and die before they have properly developed, giving the impression that they have been damaged by frost.

When infection is through the trunk of an old tree, it may require some years for the tree to die, during which period there will be increasing microphyllia, crown thinning and yellowing, and often a failure to form the annual rhytidome plaques.

PATHOGEN SPREAD. The pathogen is spread by entering an existing wound or through root anastomoses between neighbouring trees. Whether through accident or neglect, humans are the main agent ensuring the spread of the pathogen, during pruning operations, or in wounding the roots while carrying out construction or other work on the soil around the trees, but especially when treating or removing diseased trees. During sanitary operations considerable amounts of infected sawdust may be produced in which the parasite can survive for long periods (Mutto Accordi, 1988; Panconesi, 1988; Grosclaude *et al.,* 1996). Infected material of this type may also be transported long distances by winds, passing vehicle traffic, water courses or, most often, pruning implements. Of less importance, though not negligible, is the spread of the disease by insects, birds or rodents (Panconesi, 1978). Among the insects the active role played by some coleopterous insects, mainly nitidulids, has been reported by Crone and Bachelder (1961); in Italy a small cerambicid (*Morimus asper* Sulz.) and an as yet unidentified wood-boring beetle have come under suspicion. On the other hand no link has ever been found between the serious outbreaks of the tingid *Corythuca ciliata* Say to which the plane is subject and infection by *Cfp.* Among the rodents the role of *Clethrionomys glaveolus* (Sch.), which is common in plane-plantings of Versilia, has been demonstrated (Panconesi, 1987).

Trees dying or dead from canker stain which are allowed to remain *in situ,* besides providing a store of inoculum for these vectors, allow parasite mycelium to pass to the roots of anastomosed trees (Mutto Accordi, 1986), especially if the trees are very close or if dead or dying trees are left standing for a long time before being removed.

The foregoing will suffice to show the importance of immediately eliminating all possible sources of infection, that is, all dead or infected trees. At the same time, however, it is critically important to avoid wounding the remaining healthy trees in any way, whether aboveground or to the roots. Unfortunately the removal of infected trees is a double-edged sword: though on the one hand it removes the source of inoculum, on the other it produces large amounts of sawdust infected with mycelium and conidia that may spread the disease to other trees.

Analysis of infected tissue of leaving trees (Mutto Accordi, 1989a; Grosclause, 1996) has shown that the parasite rapidly loses its isolability *in vitro* as following commensal saprophytes take over; however, it remains pathogenic for a long period. I have found that it may remain pathogenic for as long as two years on trees that have been cut down as a precautionary measure. Microscopic examination of infected tissue from the collar two years after the tree had died showed mycelium and thick-walled conidia that were still intact. This means that trees that have died from canker stain cannot be freely cut down even after this length of time since the sawdust that would be produced, especially from cutting the inner sapwood tissue, is still highly infectious. This risk has been confirmed by artificial infection with fragments of such tissue.

Even tissue of trees dead for more than two years and that is already colonised by basidiomycetes, whose carpophores are visible, has in rare cases revealed the presence of thick-walled conidia of *Cfp* and given positive results in pathogenicity tests. It is clear that the first fungi that follow *Cfp* make it more difficult to identify this fungus but do not kill it. With time, however, other fungi succeed in colonizing the dead wood: *Chondrostereum purpureum* (Fr.) Pouz*., Fomes fomentarius* (L.) Kickx., *Trametes hirsuta* (Wulf.: Fr.)*, Auricularia auricula-judae* (Bull.: Fr.) Wettst., *Schizophyllum* sp., etc. Unfortunately this only happens at a relatively late stage, when the parasite may already have passed to a neighbouring tree by root anastomosis, or when the degradation of the wood tissue has reached levels that threaten the tree itself. For these reasons it is biologically undesirable, as well as dangerous in practice, to wait for the disease to kill the tree before cutting it down.

PHYSIOLOGICAL ASPECTS

MECHANISMS OF PATHOGEN AGGRESSION. Modification of the pit primary cell walls and marked alteration of the middle lamella indicate that *Cfp* may release hydrolytic enzymes, as previously suggested (D'Ambra *et al.,* 1977; Mutto *et al.,* 1978).

It has recently been shown that *Cfp* produces a succession of phytotoxic metabolites that are probably involved in the symptom manifestation of canker stain (Ake *et al.,* 1992; Burki, 1996). Of particular interest is fimbriatan, a water-repellent protein (Ake *et al.,* 1992) with a molecular weight of about 15,000 Da, which strongly inhibits cellular growth of plane tree calli *in vitro.*

Since the effect of culture filtrates of *Cfp* on plane tree calli resembles that of the first stages of canker stain on young seedlings (Ake *et al.,* 1992), and fimbriatan mimics the effect of the *Cfp* culture filtrate**,** it is hypothesised that fimbriatan has a role in canker stain pathogenesis, at least in the very early stages. This role must be specifically related to the inhibition of mitotic activity of the calli during the process of scarring (hence the failure of canker stain infections to scar over properly). The water-repellent nature of fimbriatan has further prompted the suggestion that the protein has a role in the dissemination and preservation of spores.

Recently from *Cfp* culture filtrate a new proteic metabolite, named ceratoplatanine, with a molecular weight of 12,380 Da and rich in cisteine has been purified and sequenced (Santini, 1998). This metabolite induces hypersensitive necrosis on tobacco leaves and fluorescence on plane-tree leaves, and is structurally and functionally similar to cerato-ulmin, a hydrophobine that has an important role in Dutch elm disease.

More recent research indicates that ceratoplatanine also occurs on the outer wall of cylindrical ameroconidia (A. Scala, personal communication, 1998).

HOST TREE DEFENCE MECHANISMS. It has already been stated that *C. fimbriata* is a wound parasite as it is unable to penetrate the outer defences of the host tree on its own.

The post-infectional defence mechanisms of the plane tree examined so far are either chemical, with the production of phytoalexins, or physical, with the compartmentalisation of infected areas (El Modafar *et al.,* 1993; Clerivet and El Modafar, 1994; El Modafar *et al.,* 1995). The accumulation of flavans (El Modafar *et al.,* 1996) and the production of jasmonic acid and the two hydroxycoumarins, umbelliferone and scopoletin, are induced by inoculating seedlings and cell cultures of *P. acerifolia* with *Cfp* (Alami *et al.,* 1997). Biological assays have shown that these phenol compounds have fungicidal properties and can therefore be considered phytoalexins. Unfortunately they accumulate too slowly to inhibit conidia germination entirely, and so the parasite is able to survive and expand into the area adjacent to the infection site. (El Modafar *et al.,* 1993).

During infection, inside the internal conducting vessels of the sapwood there are important changes that lead to the formation of tyloses and the production of a gel containing flavans**.** Moreover, in the vessels around the points of inoculation there are concentrated fluorescent blue substances that are thought to be coumarins. At a later stage of infection the pits of the vessels are closed and ultimately both walls and pits are covered with a granular coating (Clerivet and El Modafar, 1994).

The inoculation of leaves of canker-stain resistant *P. occidentalis* and canker-stain susceptible *P. acerifolia* with *Cfp* conidia causes foliar necrosis in both species, as well as the synthesis and accumulation of the hydroxycoumarins umbelliferone and scopoletin mentioned before. The accumulation of phytoalexin compounds occurs immediately with the resistant species but more slowly in susceptible genotypes (El Modafar *et al.,* 1995).

CONTROL

CHEMICAL CONTROL. Since externally applied treatments have not met with appreciable success, alternative approaches have been tried in which the fungicide is brought in direct contact with the parasite mycelium, which grows by preference in the conducting vessels of the alburnum. For this it was necessary to construct a device that would be able to inject the fungicide under pressure into the vascular system of the tree and to solve the difficult problem of finding a fungicide that would be effective against *Cfp* yet not toxic to the tree. Some fairly effective substances were identified after a search (Carbendazim, Thiabendazole and Imazalil sulphate) and a good delivery technique was also developed (pressure injection). Unfortunately, even repeated testing failed to produce the desired results: the most that could be achieved was a temporary halt to the infective process, and here there was sometimes considerable success. The pathogen was not eliminated, however: after some years of preventive treatment it was again able to overcome the compartmentalisation barriers created by the protected tree and kill it.

Recently a new approach was attempted (Panconesi, unpublished data); an unstable sulphur-based substance (Potassium thiosulfate) was injected into a tree

and at the same time an acidic buffer solution was applied. The intention was to obtain a precipitate of colloidal sulphur within the tissues and around the canker in the hope that thus the sulphur would kill the mycelium of *C. fimbriata,* as it had done in *in vitro* experiments. However, this attempt also proved unsuccessful.

BIOLOGICAL CONTROL. Attempted biological control measures (pressure injection), whether with bacterial suspensions of *Bacillus subtilis* or with spores of *Trichoderma harzianum* (these last had shown a strong antagonistic effect *in vitro* against *C. fimbriata*) have failed to give the desired results (Turchetti and Panconesi, 1982; Mutto Accordi, 1989b).

Although the necrotrophic action of *Gonatobotryum fuscum* Saec. (*Deuteromycotina Hyphomycetes*) and *Hirschioporus pargamenus* (Fr.) Bond. and Sing. (*Basidiomycotina Hymenomycetes*) against *Cfp* is known, there are no reports on the use of these mycoparasites to control *Cfp*.

GENETIC IMPROVEMENT. The epidemic spread of canker stain is in part favoured by the high susceptibility and the genetic uniformity of *P. acerifolia*, this last probably due to the spread of a limited number of original individuals multiplied primarily by vegetative propagation. Because of this susceptibility, as well as the lack of genetic variability and the small number of species in the genus *Platanus,* it is to be expected that the genetic improvement of *P. acerifolia* for resistance to canker stain will be a difficult undertaking.

The literature on this subject is not very extensive apart from Coggeshall *et al.* (1981) and McCracken (unpublished data) in the USA and El Modafar *et al.* (1995) in France. As regards variability in the pathogenicity of *C. fimbriata,* a vital element in any genetic improvement scheme, it has been shown that all strains isolated in Italy kill suckers of the plane tree 4-5 cm in diameter from the three most commonly cultivated species, *P. orientalis, P. acerifolia* and *P. occidentalis,* although this last species has shown itself more resistant than the others (Panconesi, unpublished data).

McCracken selectively bred some individuals of *P. occidentalis* resistant to *Cfp.* Unfortunately trees of this species have serious difficulty in adapting to the European environment and are also extremely susceptible to attack from *G. platani*; for these reasons it is practically impossible to use this species as such. Attempts are being made in France to restore the original hybrid using as parents *P. orientalis* and the resistant clones selectively obtained by McCracken (Vigouroux, 1992). But this work is proceeding very slowly; a problem that has not been solved is that relating to inoculum dosage, yet this problem is crucial when it is decided to carry out mass screening tests on young seedlings from controlled crosses.

The rate of production of the two phenolic phytoalexins, scopoletin and umbelliferone (correlated with tree resistance in that they trigger compartmentalisation, which halts the infection process), likewise has not yet been translated into practice in a way that will make mass screening possible (fast foliar reaction tests). The study of these fast tests is vital to speed up the individual selection process which, with traditional approaches, would take many years.

Stopinska (1994) found that leaves of the poplar clones resistant to *C. fimbriata* contained more auxins (IAA) and cytokinins, especially zeatin, and fewer growth inhibitors (ABA) than the leaves of susceptible poplars. A similar test could also be undertaken to identify *Cfp* resistant plane trees.

PREVENTION ON THE PLANTINGS. So far prevention is the only type of control which has given positive results, but it is often inexpertly or carelessly applied and some aspects of it need to be further refined. This therefore seems a good place to restate and comment on some fundamental points.

Identification of diseased trees. A knowledge of symptoms to identify the disease at an early stage is the first step on which the entire control effort must be based. This requires preparing and carrying out periodic inspections of plane tree plantings. Since such inspections are very onerous the bodies normally in charge of plant protection should be assisted by personnel of the towns concerned and this personnel must be qualified and kept up to date. It might also be a good idea to produce and distribute a technical and practical illustrative booklet to make it easier for tree-growers to identify and combat this serious disease.

Immediate felling of diseased and adjacent trees. Diseased trees can basically be divided into two types: trees that have been infected from above, *i.e.* through the crown, and trees that have been infected from below, *i.e.* through the roots.

If the infection comes from above and the mycelium has not yet reached the roots it is possible to fell the tree immediately at the base, separating the healthy stump from the diseased crown without risk of scattering infected sawdust, except perhaps the sawdust that is produced when the infected parts of the tree are cut up into smaller pieces later. With crown infection the healthy tree stump could conceivably remain where it is

since it does not pose a threat to neighbouring trees, even if the roots are joined by anastomosis. Nevertheless it is sound practice always to remove the stump since it can still become infected later as other infected trees are cut down, and in order to avoid root rot, which is a serious hazard.

If the infection has invaded the roots of a tree, both the infected tree and surrounding trees should be felled and uprooted immediately. Merely felling the surrounding healthy, or presumed-healthy trees without uprooting them is not sufficient since the parasite can survive for long periods on the roots of infected but not uprooted trees, enabling them still to infect the roots of other trees that were considered far enough away from the infected tree not to require felling.

A theoretical alternative to the uprooting of trees would be to kill them with the herbicide Glyphosate injected under pressure to block the mycelium development; however, this method has not been successful in practice. The most seriously infected trees absorb Glyphosate with great difficulty, and are therefore often killed only slowly, and moreover a certain amount of root activity and vitality remains that allows the parasite to spread to neighbouring trees. Failure with this herbicide has led to the search for a chemical that will kill both the tree and the fungus. Tests with sodium hypochlorite and a Glyphosate-sodium hypochlorite mixture have not proved effective since these substances did not reach all parts of the root system tissue, so that these could still be passed by the vegetative mycelium of *Cfp,* albeit with greater difficulty. It should be borne in mind that herbicides have deleterious effects on the growth of adjacent anastomosed trees as well as the tree being treated. These effects include the partial or total dieback of the crown. This was particularly noticeable when, to stop an infection from spreading, glyphosate was used to kill the healthy or presumed healthy trees growing immediately adjacent to an infected tree.

At present the infection can be halted only by the physical separation of diseased and healthy trees, despite the technical difficulties and costs that may be entailed.

It is unfortunate that the recent Italian decree law (D.L.) of 17/04/1998, replacing D.L. No. 412 of 3/09/1987, which makes it a legal obligation to combat canker stain of plane trees in Italy, is seriously flawed. The new law states that all infected trees must be felled, but for the question of eradication the reader is referred to the explanatory notes, where the eradication or cutting of infected roots is only 'recommended', and the eradication of neighbouring trees is not even mentioned. In this way the entire sanitation effort is again rendered ineffective**.**

Pruning. In areas where canker stain is known to occur pruning of any kind is to be avoided unless it is absolutely necessary. In that case the tree/trees should be pruned in the coldest and driest months of the year, and pruning wounds should be kept to a minimum and carefully disinfected afterwards, preferably with a mixture of Vinavil (polyacetyl vinylic glue) and 1% Benomyl.

Collecting and destroying infected material. The collection of sawdust and all other residues while felling or cutting up infected trees should be undertaken with the utmost care. All vehicle traffic should be suspended during felling operations and large plastic sheets should be spread under the trees to be treated. Cuts at higher levels, especially on infected trees, should be avoided or kept to a minimum. Whenever possible trees should be felled in one piece, or an infected part lopped off in one piece, or with as few cuts as possible, and the trees should be cut down to size on the ground. Sawdust and smaller infected plant residues can be gathered and burnt. The soil involved in the operation as well as all remains of roots and stumps that cannot be burnt must be taken to a disposal site and buried.

Cuts at higher levels should be carried out with a chainsaw furnished with a device whereby the sawdust is trapped and not dispersed into the atmosphere.

Disinfection of the felling site. Since some of the sawdust produced during felling may escape despite all care taken to trap it, after the felling is completed it will be necessary to disinfect the plastic sheeting, and the entire area that may be affected, with a 2% Benomyl or a 2% sodium hypochlorite solution.

Disinfection of work implements. All tools used to cut infected material (axes, saws, chainsaw blades, knives etc.) must be disinfected by immersion for some minutes in a solution consisting of 50% denatured ethyl alcohol and 50% of an 8% sodium hypochlorite solution.

Use of wood obtained. The disposal of wood residues can be a difficult problem that must be dealt with in advance. The main and other branches can be used in industry for the manufacture of woodchip board. The high temperatures (90-100°C) during the manufacturing process ensure that the parasite is killed. The tree trunks can be processed in sawmills with a closed circuit that recycle the sawdust generated by burning it to create the moist heat necessary for manufacturing timber materials (Ferrario *et al.,* 1996).

Town budget estimates. Towns must set up a special fund to deal with sanitary operations that have a certain degree of urgency. We have often had occasion to note that some epidemic diseases, such as canker stain or harmful insects like *Cynara cupressi* on *Cupressus* spp., were able to spread alarmingly in city plantings because of the long delay between the diagnosis of a problem and effective steps being taken to deal with it. These delays were due to a slow-moving bureaucracy or to the difficulty of finding the requisite funds quickly in the town budget.

Cooperation. Public and private bodies and research organisations, both local and international, should cooperate to the best of their ability to combat canker stain as soon as it is detected anywhere. If this is not done there is a risk of causing serious economic losses and damage to the urban environment, which too often is already under strain.

REPLANTING

It has been pointed out above that all stumps from trees that die for whatever reason should always be removed to avoid root rot, which poses a much greater hazard to public safety than canker stain itself. Unfortunately, mainly for technical and economic reasons, this practice is almost never observed.

When the stumps of healthy or rotted trees are uprooted, under current agronomic conditions they can be replaced with other plane trees. When the uprooted stumps are those of trees that have died from canker stain**,** replanting with other plane trees using traditional methods is inadvisable at least for a number of years, since remaining infected wood residues in humid soil are an optimum substrate for spore production and can easily infect wounded roots of newly outplanted saplings. To replace plane trees in historic locations (as on the city walls of Lucca) a new system of individual replacements has been tested and is giving good results. The system consists of digging large holes (2-2.5 m wide, 1-1.5 m deep) which are drained and filled with farmland soil. At the centre of each hole a sapling is planted**.** It is important that the wounds naturally present on the roots do not come in contact with infected soil until they have scarred over.

CONCLUSIONS

Of the various topics discussed in this review, one of the most interesting to me is the plasticity shown by the pathogen. This is due not only to the presence of many *formae speciales*, or to the appearance of natural or cultural mutants (different strains), but also to its tendency to modify some of its physiological characteristics (chemical composition of the cell walls) or its form of reproduction, whether sexual (protoperithecia and sterile perithecia) or asexual (pear-shaped hyaline phyaloconidia and holoblastic thick-walled brown conidia). All these characteristics suggest that *C. fimbriata* is probaly genetically more plastic than other species. Perhaps this *forma specialis* is at the point of speciation from other f.sp. of *C. fimbriata*.

By contrast the host, *P. acerifolia*, one of the most common trees chosen for urban plantings in European cities, is a highly susceptible hybrid tree showing genetic segregation in the second generation. To maintain its genetic stability the tree must be reproduced vegetatively, resulting in a reduction in genotypic variability. Therefore, our city streets are lined with a 'population' of very susceptible and genetically homogeneous individuals on which the pathogen cannot exert the selective pressure that normally leads to development of resistant individuals, as can be seen in all populations endowed with genetic variability.

If to the plasticity of the pathogen, and the many and varied ways in which it spreads, we add the genetic rigidity of the host tree and the ineffectiveness of the control methods tested out so far, we are left with a rather alarming situation, as the epidemiological data at our disposal make clear. This situation concerns not only the countries in which the disease has already been detected but also, and particularly, those countries of northern and north-central Europe where plane trees are intensively planted in areas that are under heavy anthropogenic stress. It must be pointed out that unfortunately, despite the obvious destructive nature of canker stain and the economic losses it already produces, our knowledge of the disease is still far from complete and that no research efforts are being undertaken at an international level to study the problem and deal with it.

It should nevertheless be added in conclusion that studies and experiments in Italy over the last ten years have shown that if prevention is carried out systematically and rigorously in a context of concerted overall action, it is possible gradually to reduce the incidence of canker stain in affected areas, and to inhibit its spread to as yet unaffected areas. Meanwhile genetic improvement programmes may well succeed in producing plane tree individuals resistant to *Cfp* and also adapted to European environmental conditions.

AKNOWLEDGEMENTS

The Author wishes to thank Mrs. Irene Dellavalle for her helpful assistance.

REFERENCES

Ake S., Darbon H., Grillet L., Lambert C., 1992. Fimbriatan, a protein from *Ceratocystis fimbriata*. *Phytochemistry* (Oxford) **31**: 1199-1202.

Alami I., Jouy N., Clérivet A., 1997. Jasmonic acid in elicitor-induced phytoalexin production in plane tree (*Platanus acerifolia* Willd.) cell-suspension cultures. *Proceedings of the 10th Congress of the Mediterranean Phytopathological Union, Montpellier* 1997, 351-354.

Andrus C.F., Harter L.L., 1933. Morphology of reproduction in *Ceratostomella fimbriata*. *Journal of Agricultural Research* **12**: 1059-1078.

Anselmi N., Cardin L., Nicolotti G., 1994. Plane decline in European and Mediterranean countries: associated pests and their interactions. *EPPO Bulletin* **24**: 159-171.

Bakshi B.K., 1950. Fungi associated with ambrosia beetles in Great Britain. *Transactions of the British Mycological Society* **33**: 111-120.

Biocca M., Annesi T., 1996. Il platano a Roma: aspetti fitosanitari e paesaggistici. *Acer* **3**: 22-28.

Burki N., 1996. Isolement et identification de métabolites secondaires de *Ceratocystis fimbriata* f.sp. *platani* agent pathogène du platane. Université de Neuchâtel, Suisse.

Cadahia D., 1983. Nuevos problemas fitosanitarios. *Boletín Servicio de defensa contra Plagas e Inspección Fitopatologica* **9**: 275-285.

Clérivet A., El Modafar C., 1994. Vascular modifications in *Platanus acerifolia* seedlings inoculated with *Ceratocystis fimbriata* f.sp. *platani*. *European Journal of Forest Pathology* **24**: 1-10.

Coggeshall M.V., Land S.F. Jr., Ammons V.C., 1981. Genetic variations in resistance of young American sycamore. *Plant Disease* **65**: 140-142.

Cole G.T., Samson R.A., 1979. Patterns of development in conidial fungi. Pitman, London.

Crandal B.S., 1935. *Endoconidiophora fimbriata* on sycamore. *Plant Disease Reporter* **90** (Supplement): 98 (Abstract).

Cravero S., Gianetti G., Scapin I., 1992. La situazione del cancro colorato del platano in Piemonte. *Acer* **1**: 15-17.

Cristinzio M., Marziano F., Vernau R., 1973. La moria del platano in Campania. *Rivista di Patologia Vegetale* **9** (Supplement IV): 189-214.

Crone L.J., Bachelder S., 1961. Insect transmission of canker stain fungus. *Ceratocystis fimbriata* f. *platani*. *Phytopathology* **51**: 576 (Abstract).

D'Ambra V., Ferrata M., Baldan B., 1977. Growth and behaviour of *Ceratocystis fimbriata* in plane-tree tissues. A scanning electron microscope study. *European Journal of Forest Pathology* **7**: 351-357.

Davidson R.W., 1935. Fungi causing stain in logs and lumber in the United States including five new species. *Journal of Agricultural Research* **50**: 789-807.

De Hoog G.S., 1974. The genera *Blastobotrys*, *Sporothrix, Calcarisporium* and *Calcarisporiella* gen. nov. *Studies in Mycology* **7**: 1-84.

De Hoog G.S., Scheffer R.J., 1984. *Ceratocystis* versus *Ophiostoma*: a reappraisal. *Mycologia* **76**: 292-299.

Domsch K.H., Gams W., Anderson T.H., 1980. Compendium of soil fungi. Vol. I, Academic Press, London.

Dowding P., 1984. The evolution of insect-fungus relationships in the primary invasion of forest timber. In: Anderson J.M., Royver A.D.M., Walton D.W.H. (eds.). Invertebrate-microbial interactions, pp. 133-153. Cambridge University Press, New York.

Elliot J.A., 1923. The ascigerous stage of the sweet potato black-rot fungus. *Phytopathology* **13**: 56 (Abstract).

Elliot J.A., 1925. A cytological study of *Ceratostomella fimbriata* (E. & H.) Elliot. *Phytopathology* **16**: 417-422.

El Modafar C., 1993. Aspects histologiques et biochimiques des interactions hôte-parasite (*Platanus* spp., *Ceratocystis fimbriata* f.sp. *platani*). Reactions associées à la défense de l'hôte. Thèse Doctorat. Université Montpellier 2.

El Modafar C., Clérivet A., Fleuriet A., Macheix J.J., 1993. Inoculation on *Platanus acerifolia* with *Ceratocystis fimbriata* f.sp. *platani* induces scopoletin and umbelliferone accumulation. *Phytochemistry* **34**: 1271-1276.

El Modafar C., Clérivet A., Macheix J.J., 1996. Flavan accumulation in stem of *Platanus acerifolia* seedlings inoculated with *Ceratocystis fimbriata* f.sp. *platani*, the canker stain disease agent. *Canadian Journal of Botany* **74**: 1982-1987.

El Modafar C., Clérivet A., Vigouroux A., Macheix J.J., 1995. Accumulation of phytoalexins in leaves of plane-tree (*Platanus* spp.) expressing susceptibility or resistance to *Ceratocystis fimbriata* f.sp. *platani*. *European Journal of Forest Pathology* **101**: 503-509.

Fernandez de Aña Magan F.J., Gil M.C., 1977. Estudio de las causas productoras de daños en la masa arbórea de la "Devesa" de Gerona. I.N.I.A. Departemento de Producción Forestal, Lourizan, Pontevedra, España.

Ferrari J.P., Pichenot M., 1974. *Ceratocystis fimbriata* responsable d'une grave maladie du platane en France: la tache chancreuse. *Comptes Rendus hebdomadaires des séances de l'Académie des Sciences de Paris. Série D.* **278**: 2787-2789.

Ferrari J.P., Pichenot M., 1976. The canker stain disease of plane-tree in Marseilles and in the south of France. *European Journal of Forest Pathology* **6**: 18-25.

Ferrario P., De Filippo G., Perucca M., 1996. Risanamento di legname di platano affetto da cancro colorato. *L'Informatore agrario* **39**: 74-75.

Gellini R., Grossoni P., 1978. Aspetti botanico-forestali del genere *Platanus*. *Informatore Fitopatologico* **28**: 45-52.

Gervasini E., 1996. Situazione del cancro colorato in Lombardia. *Atti del Seminario "Il cancro colorato del platano", Luino (Varese)* 1996, 23-24.

Gessler C., Mauri G., 1987. Le malattie e i parassiti del platano, situazione nel Ticino. *Botanica Helvetica* **2**: 349-355.

Gianetti G., 1996. Situazione epidemiologica del cancro colorato del platano in Piemonte. *Atti del Seminario "Il cancro colorato del platano", Luino (Varese)* 1996, 29-31.

Granata G., Guastella G., 1986. Grave moria di platani lungo i corsi d'acqua del territorio Ibleo. *Sviluppo Agricolo* **20**: 5-8.

Granata G., Parisi A., Cacciola S.O., 1992. Electrophoretic protein profiles of strains of *Ceratocystis fimbriata* f.sp. *platani*. *European Journal of Forest Pathology* **22**: 58-62.

Grosclaude C., Olivier R., Romiti C., 1996. Chancre coloré du platane. Comment l'agent responsable peut survivre dans le sol. *Phytoma la Défense des Végétaux* **479**: 41-42.

Harrington T.C., 1981. Cycloheximide sensitivity as a taxonomic character in *Ceratocystis*. *Mycologia* **73**: 1123-1129.

Harrington T.C., 1987. New combination in *Ophiostoma* of *Ceratocystis* species with *Leptographium* anamorphs. *Mycotaxon* **28**: 39-43.

Hunt J., 1956. Taxonomy of the genus *Ceratocystis*. *Lloydia* **19**: 1-58.

Jackson L.W.R., Sleeth B., 1935. A new disease affecting *Platanus orientalis* in the eastern United States. *Phytopathology* **25**: 22.

Jewell T.R., 1974. A qualitative study of cellulose distribution in *Ceratocystis*. *Mycologia* **66**: 139-146.

Levi Pires M.G.R., 1998. Cancro azulado do plátano. Una grave doença em ambiente urbano. Eresmus thesis. Universidade Técnica de Lisboa, Portugal.

Marchetti L., Vai N., 1996. Diffusione del cancro in Emilia Romagna. *Atti del Seminario "Il cancro colorato del platano", Luino (Varese)* 1996, 27-28.

Marvin E.F., 1939. *Ceratostomella* (*Endoconidiophora*) on plane-trees. *Plant Disease Reporter* **23**: 154-156.

Marziano F., 1988. L'agente del cancro colorato del platano: notizie micologiche. *Acer* **4**: 26-29.

Matasci Stanga M., 1993. *Ceratocystis fimbriata* Ell. *et* Halsted f. sp. *platani* Walter: evoluzione e gestione dell'epidemia in Canton Ticino. Tesi. Scuola Politecnica Federale di Zurigo.

McCracken F.I., Burkhardt E.C., 1977. Destruction of sycamore by canker stain in the midsouth. *Plant Disease Reporter* **61**: 984-986.

Melin E., Nannfeldt J.A., 1934. Researches into the blueing of ground wood pulp. *Svenska Skogsvardforen. Tidskr.* **32**: 397-616.

Morgan-Jones G., 1967. *Ceratocystis fimbriata*. CMI, Descriptions of Pathogenic Fungi and Bacteria No. 141.

Mutto Accordi S., 1986. Diffusione di *Ceratocystis fimbriata* attraverso le anatamosi radicali. *Informatore Fitopatologico* **36**: 53-58.

Mutto Accordi S., 1987. Diffusione del cancro colorato del platano nel territorio delle Tre Venezie. *Le Foreste* **1**: 29-33.

Mutto Accordi S., 1988. Aspetti epidemiologici del cancro colorato del platano: *Acer* **4**: 36-37.

Mutto Accordi S., 1989a. Sopravvivenza nel terreno di *Ceratocystis fimbriata* f.sp. *platani*. *Informatore Fitopatologico* **39**: 57-62.

Mutto Accordi S., 1989b. Prove di lotta contro il cancro colorato del platano con l'impiego di *Trichoderma harzianum*. *Informatore Fitopatologico* **39**: 49-51.

Mutto S., Ferrata M., D'Ambra V., 1978. Ricerche al microscopio elettronico sul legno di platano invaso da *Ceratocystis fimbriata* f. *platani*. *Phytopathologische Zeitschrift* **91**: 39-51.

Nag Ray T.R., Kendrik W.B., 1975. A monograph of *Chalara* and allied genera. Wilfrid Laurier University Press, Waterloo.

Panconesi A., 1972. I nostri platani sono in pericolo. *Informatore Fitopatologico* **22**: 10-13.

Panconesi A., 1973. Il rinvenimento della *Ceratocystis fimbriata* (Ell. and Halst.) Davidson f. *platani* su platano. *Rivista di Patologia Vegetale* **9** (Supplement IV): 167-188.

Panconesi A., 1979. Il cancro del platano: aspetti biologici e possibilità di lotta. *Atti Convegno "Produzione e Patologia delle Piante arboree forestali e ornamentali", Milano* 1979, 15-26.

Panconesi A., 1986. Il cancro colorato del platano. *Acer* **2**: 31-34.

Panconesi A., 1987. Il cancro del platano: alcune considerazioni sulle conoscenze acquisite in questi ultimi anni. *ETSAF, Quaderni di qualificazione dell'assistenza tecnica: "La Difesa del Platano". Vol. 15, Marina di Massa* 1987, 8-32.

Panconesi A., 1988. Attuali conoscenze sui mezzi di lotta contro la *Ceratocystis fimbriata. Acer* **4**: 40-45.

Panconesi A., Nembi V., 1978. La *Ceratocystis fimbriata* del platano: aspetti biologici e possibilità di lotta. *Informatore Fitopatologico* **28**: 17-27.

Ruperez A., Muñoz C., 1980. Nuevas causas de desaparición del platano. *Boletín Servicio de defensa contra Plagas e Inspección Fitopatologica* **6**: 106-107.

Sammarco G., Torta L., 1997. Indagine sui funghi associati al deperimento dei platani nel Comune di Palermo. *Informatore Fitopatologico* **47**: 42-46.

Santini A., 1998. Purificazione ed attività biologica della cerato-platanina, una nuova idrofobina fungina prodotta da *Ceratocystis fimbriata* f.sp. *platani*, e caratterizzazione della popolazione italiana dell'ospite. Tesi di Dottorato di Ricerca in Patologia Forestale, X Ciclo.

Santini A., Capretti P., 1998. Analysis of *Ceratocystis fimbriata*

f.sp. *platani*. Italian population by amplification of mini-satellite DNA. *Proceedings of the International Congress of Plant Pathology 98, Edinburgh* 1998, 2.2.10.

Simonian S.A., Mamikonyan T.O., 1982. Disease of plane-tree. *Zashchita-Rastenii* **8**: 23-24.

Smith M.J., Patrik C.M., Rosinski M.A., 1967. A comparison of cellulose production in the genus *Ceratocystis*. *Mycologia* **59**: 965-969.

Spencer J.F.T., Gorin P.A.J., 1971. Systematics of the genera *Ceratocystis* and *Graphium*. Proton magnetic resonance spectra of the mannose-containing polysacharides as an aid in classification. *Mycologia* **63**: 387-402.

Stopinska J., 1994. Plant growth regulators in poplar clones differing in resistance to the fungus *Ceratocystis fimbriata* Ell. *et* Halst. *Acta Societatis Botanicorum Poloniae* **63**: 53-59.

Sydow H., Sydow P., 1919. Mykologische Mitteilungen. *Sydowia* **1**: 33-47.

Turchetti T., Panconesi A., 1982. Osservazioni preliminari sull'antagonismo di alcune specie di *Bacillus* verso *Ceratocystis fimbriata* (Ell. & Halst.) Davidson f. *platani* Walter. *Rivista di Patologia Vegetale* **18**: 71-76.

Upadhyay H.P., 1981. A monograph of *Ceratocystis* and *Ceratocystiopsis*. The University of Georgia Press, Athens.

Upadhyay H.P., Kendrik W.B., 1975. Prodromus for revision of *Ceratocystis* (*Microascales*, *Ascomycetes*) and its conidial states. *Mycologia* **67**: 798-805.

Vigouroux A., 1986. Les maladies du platane avec référence particulière au chancre coloré; situation actuelle en France. *EPPO Bulletin* **16**: 527-532.

Vigouroux A., 1992. Preliminary results for obtaining a plane-tree resistant to canker-stain and adapted to European conditions. *Proceedings of the International Symposium on Selection and Breeding of Woody Ornamentals, Angers*, 1992, no. 320, 91-96.

Von Arx J.A., 1974. The genera of fungi sporulating in pure culture. 2nd Ed. J. Kramer, Vaduz.

Von Höhnel F., 1918. Mykologische Fragmente. CXCV. Über die Gattung *Parodiopsis* Maublanc. *Annales Mycologici* **16**: 40-41.

Walter J.M., 1946. Canker stain of plane-trees. United States Department of Agriculture, Circular 742.

Walter J.M., Rex E.G., Schreiber R., 1952. The rate of progress and destructiveness of canker stain of plane-trees. *Phytopathology* **42**: 236-239.

Webster R.K., Butler E.E., 1967a. A morphological and biological concept of the species *Ceratocystis fimbriata*. *Canadian Journal of Botany* **45**: 1457-1468.

Webster R.K., Butler E.E., 1967b. The origin of self-sterile, cross-fertile strains and culture sterility in *Ceratocystis fimbriata*. *Mycologia* **59**: 212-221.

Wejman A.C.M., De Hoog G.S., 1975. On the subdivision of the genus *Ceratocystis*. *Antoine van Leeuwenhoek* **41**: 353-360.

Wingfield M.J., 1993. Problems in delineating the genus *Ceratocystiopsis*. In: Wingfield M.J., Seifert K.A., Webber J.F. (eds.). *Ceratocystis* and *Ophiostoma*: taxonomy, ecology and pathogenicity, pp. 21-25. APS Press, St. Paul, Minnesota.

Wulf A., 1995. Gefährdung der Platane durch zunehmende Ausbreitung des Kebserregers *Ceratocystis fimbriata*. *Gesunde Pflanzen* **47**: 12-15.