Ash Disease: the present state of knowledge or ignorance

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Ash Disease has been scandalously neglected in Britain, where tree pathologists are a Critically Endangered taxon. But there have been many investigations on the Continent. Not much has been published, but I have ascertained some of the essential data from a skeleton report of a conference in Oslo in 2010:[1]
archives.eppo.org/MEETINGS/2010_conferences/**chalara**_oslo.htm.

The causal fungus

Like many plant-pathogenic fungi, the Ash Disease fungus exists in two alternating phases which look like very different fungi and have separate names. It was discovered and published in 2006 as a fungus, new to science, given the name *Chalara fraxinea*. Three years later this was interpreted as a stage in the life-cycle of a cup-fungus, *Hymenoscyphus albidus*, which had been known since 1851 as an obscure and harmless inhabitant of fallen ash leaves. For example my German fungus flora has:

Helotium robergei . . . (= *H. albidum*) [obscure fungi tend to have many names] . . . In blackened spots on rotting ash-leaves.[2]

In 2010 it was discovered that *Chalara fraxinea* is really the pathogenic phase of another, closely related, previously unknown cup-fungus to which the name *Hymenoscyphus pseudoalbidus* was given. This is identical to look at to *H. albidus*, but genetically different; the two can be distinguished only by their DNA.[3]

The life-cycle appears to be as follows. *Chalara fraxinea*, living on the leaves and shoots of ash trees, turns into *Hymenoscyphus pseudoalbidus* when the leaves fall. This, like any other cup-fungus, puffs out ascospores into the air. Some of the ascospores drift up into the canopy, alight on new shoots of ash and — somehow — get established and start an infection. This gives rise to a new generation of *Chalara.* Most of the new infection appears to occur in late summer.

How it gets about

The *Hymenoscyphus* phase of the fungus is only 3 mm across; it grows only on the rhachis (the midrib, which falls separately from the leaflets) of fallen leaves. It evidently occurs in great abundance under infected ash-trees and can produce vast numbers of spores. An average leaf-rhachis is reported as bearing 20 cup-fungi, each producing 1500 ascospores an hour for about two weeks: several million spores from each ash-leaf. Spores seem to be put out mainly in late summer, especially around dusk. They measure typically around 17 x 4 μ, so they should efficiently float in the air like specks of dust. They appear to be the main natural means of transmission over long distances.

The *Chalara* phase also produces spores (called conidia) which are said to cause new infections within the tree. They seem not to be blown over long distances, although it occurs to me that they could be transmitted on the feet of migrating birds.

Effects on the tree

Once started, an infection grows slowly along the twig. Most infections stop at a branch-to-twig point, but some extend into branches and the trunk of the tree, eventually killing it. Infections continue from year to year, extending mostly in spring, with a smaller phase of activity in midwinter.

Chalara makes a fungal poison called viridiol, which is toxic to ash.

When *Chalara* kills a twig, the tree responds by making replacement shoots, which are infected in turn, giving rise to bunched foliage like the small witches'-brooms that arise on many other trees from various causes. I have seen these in Estonia. Bunched foliage can arise from other causes: in Czechia, for example, from the aphid *Prociphilus bumeliæ*[4] (which seems not to occur in Britain).

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[1] I am indebted to Louise Bacon for the reference.

[2] M Moser (1963) *Kleine Kryptogamenflora Band IIa: Ascomyceten* Fischer, Stuttgart

[3] T Kowalski 2006 *Chalara fraxinea* sp. nov. associated with dieback of ash (*Fraxinus excelsior*) in Poland *Forest Pathology* **36:** 264—70

T Kowalski & O Heldenrieder 2009 The teleomorph of *Chalara fraxinea*, the causal agent of ash dieback *Forest Pathology* **39:** 304—8

V Queloz *et al.* 2010 Cryptic speciation in *Hymenoscyphus albidus. Forest Pathology* **41:** 133—42

[4] This too sounds like an introduced species: *Bumelia* is a genus of North American shrubs.

Chalara does not always (or even usually?) kill the tree. Finnish and Czech scholars show pictures of infected ashes getting better as well as worse from year to year, but remark 'overall development negative'. It is not true that 90% of the ash trees in Denmark are dead: I was there this year and saw plenty of ashes: I am told that there are still many ashes left alive in Poland. However, a research article has already been inspired by the ecological effects of ash decline in Lithuania.[5]

Relation to Hymenoscyphus albidus

It is said that *H. albidus* has the same life-cycle, including a *Chalara*-type phase, but makes no viridiol and does not harm the tree: it may be a normal endophyte (a fungus that lives symbiotically within healthy tissue).

H. pseudoalbidus appears to be genetically variable through hybridization with *H. albidus.*

Spread of the disease

Chalara seems to have been first noticed in or around Latvia *c.*1990. This would be consistent either with an introduction from somewhere outside Europe, or a new mutation or hybridization in *Hymenoscyphus. Chalara* is supposed to spread at 20—30 miles a year, but since the disease is poorly defined and confusable with other conditions such claims are unreliable. It is likely that it has been present but unobtrusive for many years.[6] Re-examination of herbarium specimens attributed to *H. albidus* has shown that *H. pseudoalbidus* was present in Switzerland already in the 1960s.[7]

Many reported outbreaks, in various countries, are linked to the nursery trade, both within and between countries. Ash seedlings grown in nursery conditions are evidently very susceptible to infection, and can convey it long distances or to islands that it might not otherwise reach. However, occurrences like that in Ashwellthorpe Lower Wood, not obviously linked to any nursery, show that this is not the only explanation. Ascospores blowing from the Continent are a possibility — they are small enough to get that far. However it seems unlikely that a single spore, after a wind journey of hundreds of miles, would have enough *inoculum potential* (as Dennis Garrett, the great plant pathologist, taught me in the 1960s) to start an infection, though maybe a mass of conidia on the foot of a bird might do so on rare occasions.

Hymenoscyphus is reported as having natural enemies, including fungus gnats and the ubiquitous parasitic fungus *Paecilomyces marquandii.*

What is to be done?

On present information, not much. *Chalara* is here, and no human action will make it go away. Now that most of the leaves have fallen, it is almost impossible to detect further infections, so the full extent of the disease will not be known until late spring.

There seems to be no point in sanitation felling. The practical difficulties with such a common tree — even finding out who owns the trees around an outbreak — are formidable. As Keith Kirby, ecologist, said:

> [Sanitation felling] is only likely to be effective at the early stages of an outbreak or in tightly defined areas. After that the disease may be too widespread for eliminating local sources to make much difference.

That stage has already been passed. Even if the initial outbreaks could yet be identified and suppressed, there will be other outbreaks in later years, leading to more fellings, until no ash trees were left.

Exhorting people to wash boots, children, and dogs after visiting a wood is a counsel of despair — unless they also wash the deer; and even if they could, it is unlikely to have any measurable effect on a disease that is probably airborne. 'We have to do something; this is something; let us do it!'

Not moving ash plants will suppress an obvious and plausible route of transmission. Not moving ash timber or underwood is less plausible, for the fruit-bodies are associated with living or dead leaves rather than bark.

Any such measures presuppose that it will be possible to establish, and maintain into the indefinite future, a situation where Ash Disease is present in some parts of the country but kept out of others. This seems fanciful, seeing how *Chalara* has marched unopposed across Europe. The best that

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[5] Roberge J-M *et al.* 2011 Edge creation and tree dieback influence the patch-tracking metapopulation dynamics of a red-listed epiphytic bryophyte [the moss *Neckera pennata*] *Journal of Applied Ecology* **48:** 650—8

[6] In the late 1970s, following the great droughts of 1975—6, there was a widespread bunched-foliage dieback of ash-trees in the English Midlands, resembling what I saw in 2012 with Ash Disease in Estonia. Although spectacular, it was never investigated, and most of the trees recovered. Could Ash Disease already have been involved?

[7] Queloz *et al.* (see note 3)

could be hoped for would be to exclude *Chalara* from Ireland (supposing it is not already there) or from remote islands.

Fungicide might be of some use, but hardly beyond the scale of protecting individual special trees.

'I told you so'

As I have said many times before,[8] globalization of tree diseases and pests is the greatest threat to the world's trees and forests: greater than climate change or even than proliferation of deer. Examples:

Elm Disease, the probable cause of the Elm Decline at the beginning of the Neolithic, some 6000 years ago, in Europe but not America.

Vine pests and diseases: downy mildew, powdery mildew, and the root aphid *Phylloxera*, brought from America to Europe in the 19th century. They nearly killed wine-making. Wine was saved by new technology, but growing is has permanently become a complex business requiring grafting and chemicals.

Oak Mildew: imported from America in the 1900s, now universal in Europe; apparently a trivial disease, but may have permanently changed the behaviour of oak, which no longer grows readily from seed in existing woods as it used to.

Chestnut Disease: the fungus *Endothia parasitica*, probably from Asia, that appeared in the 1920s and devastated the sweet-chestnut of southern Europe and the related species in North America. The European chestnuts have mainly recovered, thanks to the intervention of a fungal virus, but in America, where Longfellow sang 'Under the spreading chestnut-tree The village smithy stands', the chestnut is now rarer than the smithy.

Elm Disease, brought from Europe to North America in the 1920s, which by 1980 had subtracted most elms from the NE United States.

Phytophthora cinnamomi, a common tropical root parasite, which got into SW Australia in the 1930s and has been destroying most of the unique flora of that long-isolated land — one of the great ecological tragedies of the 20th century.

Alder Disease, a *Phytophthora* of unknown origin, first noticed in England in the 1990s, which continues to spread and ravage alders.

Marchalina hellenica, a woolly-aphid-like insect which lives on pines in the east Mediterranean. It is encouraged by Greek beekeepers because it is a source of honey. (Most Greek honey comes not from flowers but from the back ends of insects that feed on conifers.) It may be native to Greece, but beekeepers used to introduce it to new regions and for a time were subsidized to do so by the government. (At one stage they rejoiced that someone had persuaded *Marchalina* to attack firs as well as pines.) Too late it was discovered that the insects kill the tree: they are devastating the pines in the arid mountains of SW Crete.

Emerald Ash Borer, an insect brought from east Asia to Canada. In 2004 the United States was spending many millions of dollars trying to keep it out of Ohio, where previous globalizations had left ash as the commonest remaining tree. I predicted that Uncle Sam would fail, and he has since failed.

Red-Band Needle Blight, a fungus that attacks many pines. In 2010 the Forestry Commission stopped planting Corsican Pine because of its ravages. It is now a worldwide problem on *Pinus radiata*, one of the most widely planted forestry trees and the source of a large proportion of the world's cellulose supplies.

I could easily quote a dozen more, especially other soil-inhabiting *Phytophthoras*. Introduced diseases and pests continue to subtract tree after tree at random from the world's ecosystems and plantations. (Each time, millions of tons of CO_2 are released into the atmosphere.) So far Britain has been let off lightly, but if this goes on for another century, how much will be left?

Lessons for the future

Globalization of plant diseases expanded in parallel with globalization of trade. Part of the reason is probably the swiftness of intercontinental transport. When live plants were taken in Wardian cases (miniature greenhouses) by sailing ship round Cape Horn, it was likely that any parasites would either die or kill their hosts on the voyage. With steamships, parasites began to survive ocean crossings (as they did in the 19th century), and with air transport many survived.

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[8] For example:
O Rackham 2006 *Woodlands* Harper Collins, London pp542—4
O Rackham 2008 Ancient woodlands: modern threats *New Phytologist* **180:** 571—86

More than this is the expanding volume of trade in plants and soil, much of it of the coals-to-Newcastle kind. Here the movement for planting 'native' trees must bear some responsibility. Tree-planting has become institutionalized, entering a world of grants and tenders and contracts and subcontracts and work to be finished on time and money to be spent before the end of the financial year: an environment geared to the anthropology of bureaucrats and at variance with the 'real' world of trees and parasites. A subcontractor, required to produce so many oak seedlings and finding that the oaks did not bear acorns this season, will go to another country where acorns are more reliable and labour is cheaper.

Volume is what matters. Travellers, bringing back little Christmas trees in their luggage, though in theory persecuted by the Customs, are unlikely to do much harm. But a commercial supplier, importing a million container-grown hawthorns from Hungary (as though there were no hawthorns in Britain!), inevitably imports a thousand tons of Hungarian soil and whatever is in it. However thoroughly the Customs, or a responsible nurseryman, inspect the consignment, they cannot be expected to detect a microscopic pathogen when they cannot know in advance what to look for.

At a conference in Ireland in 2004, I remarked that the Irish had had enough experience of imported plant disease — the calamitous potato famine in 1845, caused by a fungus that fell off an American ship — to last them a thousand years. The man from the Ministry got up and bleated that nothing could be done because this would restrict trade and the World Trade Organization would not allow it. The WTO, it seems, won't let the stable door be locked until plant pathologists have certified that the horse has gone.

Globalization of pathogens has been described as a 'fundamental flaw' in international trade. I remarked years ago that any of the world's plant diseases is at liberty to enter Britain provided it does so via some other European Union country. (*Chalara* has done just that, it seems.) By the time the problem has been detected and the bureaucracy has clanked into action, it is too late. Once a tree disease has become established in a country, it is almost unknown for it to be controlled, let alone exterminated.[9] As one of the participants at the Oslo conference said: 'No eradication of an invasive plant pathogen has been recorded in Europe'.

As John Gibbs, the great tree pathologist, pointed out, it is useless reacting to known plant diseases: that battle has already been fought and mostly lost. What is needed is to forestall plant diseases that have not yet reached this country or that are still unknown.

The first step is to discourage the long-distance movement of plants and soil: not only between countries, but within a country (as is done in the United States and Australia). The Norwegians report that one outbreak of *Chalara* followed a consignment of nursery plants sent from south to middle Norway. Banning routine movement would no doubt make tree-planting more expensive, but that is not necessarily to the bad. At present, cheap trees are planted close together and too often neglected and left to die. A better result might come from planting expensive trees wider apart and taking care of them.

The public has drifted into the habit of treating plants as mere articles of commerce, like cars or soap, that can be made and brought from anywhere in the world with no consequences. Planting trees has become a default option: 'Ye may be aye stickin' in a tree; it'll be growin' while ye're sleepin'. This casual mind-set needs to be changed. The pros and cons of every tree-planting scheme need to be formally discussed, including assessing the risk that planting trees will threaten existing trees.

Update to 21 November 2012

Ash Disease — or rather DNA that is indistinguishable from that of pathogenic *Chalara* — has been found in more than 100 sites, mainly in eastern England, but with outliers as far as Wales, Scotland, and once in Ireland.

Many sites have definite symptoms of the disease in recently-planted ash trees got from nurseries. This confirms that the nursery trade is one of the agencies disseminating the disease. Other sites involve symptoms on young coppice growth.

Records of disease in big ash trees appear to be rare. In many instances, however, ash trees have been sampled at random, without evidence of disease or of fruit-bodies, and yet *Chalara* DNA has been found. That is to say, false positives — fungal DNA without disease — seem to be common, including in woods that are remote from nursery ash trees.

[9] There is one exception, or rather half an exception. Sweet-chestnut disease is no longer much of a problem in southern Europe: not because anyone did anything, but because God raised up a fungal virus that rendered the fungus incapable of killing the tree. All efforts to get the virus going in America have failed.

It looks as if *Chalara,* or something indistinguishable from it on DNA, is already common and has been present for many years — as is indicated by the Swiss herbarium findings. Among possible explanations are these:

(a) DNA technology is not yet certain of distinguishing pathogenic from non-pathogenic *Chalara.*

(b) Most ash trees are resistant, perhaps carrying *Chalara* as a harmless endophyte, and only exhibit symptoms when something — transplanting, coppicing etc. — triggers it to produce viridiol.

There is little prospect of resolving these matters before spring 2013. It will be at least some years before the effects on British ash trees will be known. Most information about effects in Europe has passed through several hands before reaching us, each time with an opportunity to exaggerate.

Anyone interested should look for one or more of the following:

1. Discoloured lesions around buds on coppice shoots or young stems.
2. Little witches'-brooms (masses of crowded twigs), especially on big trees. These are the effect of twigs repeatedly dying back and being replaced by other twigs. They can be caused by other factors, including deer if they are near the ground.
3. Fungus fruit-bodies. These are little cups on stalks (like a golfer's tee), 3 mm or less in diameter, white and thus conspicuous, on the rhachis of fallen and rotten ash leaves (that is the midrib of the leaf, which falls separately from the leaflets). When immature they appear as tiny whitish spikes. They are indistinguishable (except on DNA) from those of non-pathogenic *Hymenoscyphus.*