Abstract

Stem bleeding on horse chestnut (*Aesculus hippocastanum*) in the UK was first reported in the 1970s, when the cause was found to be a fungal pathogen known as *Phytophthora* (Brasier and Strouts, 1976). The same disorder had also been recognised in the USA much earlier in the 1930’s (Caroselli, 1953). Symptoms visible on affected trees include bleeding areas on their stems and sometimes on their scaffold branches. However, over the past four or five years the number of reports of horse chestnut trees with 'bleeding cankers' has increased markedly. Closer investigation of the bleeding cankers on horse chestnut has revealed that *Phytophthora* is no longer the primary causal agent. Instead there is accumulating evidence that a different pathogen, probably a bacterium, is responsible for the increase in these symptoms on horse chestnut.

Keywords: horse chestnut, *Phytophthora*, bleeding canker, bacterium, *Pseudomonas*

1. Incidence of the disease

Until recently *Phytophthora* bleeding cankers on horse chestnut (*Aesculus hippocastanum*) were considered to be uncommon and were only seen in the south of England (Strouts and Winter, 2000). However, over the past four or five years the number of reports of horse chestnut trees with 'bleeding cankers' has increased markedly (Figure 1). The increased incidence of stem bleeding on horse chestnut is not just limited to the UK; the Netherlands, France and Germany are also experiencing a similar upsurge. In 2003 the Disease Diagnostic Advisory Service (DDAS) of Forest Research received more than 40 reports of stem bleeding in horse chestnut and so far 120 reports have been collated for 2005. Affected trees have been recorded as far north as Lancashire and Glasgow.

![Figure 1. Bleeding canker cases reported to Forest Research 2000 to 2005](Image)
2. **What trees are affected?**

Trees of all ages have been affected by the recent disease upsurge. Young trees with a stem diameter of only 10cm (4 inches) have been found with advanced symptoms. However, the impact on the environment can be particularly profound when large, mature trees are infected and disfigured by the disease. If the disease is severe and the areas of bark which are killed are extensive, large trees can undoubtedly be killed. However, younger trees (10-30 years old) are at greater risk and can succumb to the disease in just a few years (3-5) as the smaller diameter of their trunks means that they can be girdled more quickly.

3. **Extent of the problem**

The problem of bleeding canker on horse chestnuts is now very widespread: thousands of trees are affected. Although no widespread systematic survey has been undertaken, Forest Research scientists estimate some 35,000 to 50,000 trees are affected and probably a few thousands have already been felled as a result of the disease.

![Figure 2. Geographical location of cases of Horse Chestnut Bleeding Canker reported to Forest Research Disease Diagnosis Advisory Service](image-url)
In a detailed survey of around 230 horse chestnuts in Hampshire, about half were found to be suffering from bleeding canker (Straw and Green, unpublished data). A higher proportion of trees in towns and rural areas displayed symptoms compared with woodland trees, while slightly more red horse chestnuts (A. x carnea) were affected compared with white flowered trees (A. hippocastanum). An example of the rare hybrid chestnut, A. + dallimorei, has also been found to be susceptible to bleeding canker.

4. Reasons for increased incidence?

The reasons for the increase in number of affected trees are currently under investigation by scientists at Forest Research. There has been speculation that the recent spate of mild winters and wet springs have favoured spread and infection by the Phytophthora species known to cause bleeding cankers on horse chestnut. However, although climate may indeed be playing a part in the incidence of this disorder, it now seems likely a causal agent other than Phytophthora is responsible for these recent cases of stem bleeding and tree death. Culturing from the margins of the dying bark tissue of affected trees in the south of England usually yields several potential pathogens. In The Netherlands, where recent surveys have revealed that one in three of all horse chestnuts are now affected to a greater or lesser extent by the same disorder, research has also been underway to discover the cause.

5. Symptoms

Trees which have been affected for some years may show crown symptoms (Figure 3).

Figure 3. Yellowing and reduction of foliage in part of the crown

However, the early symptoms tend to be limited to bleeding lesions; scattered drops of rusty-red, yellow-brown or almost black, gummy liquid ooze from small or large patches of dying bark on the stems or branches of infected horse chestnuts (Figures 4 and 5).
Bleeding patches may be associated with the base of the tree at the soil surface or may start higher up the trunk at about one metre, and then extend upwards. Early in the year (spring) the exudate from bleeding patches is a dark colour but transparent. However, as the weather becomes warmer, bleeding from infected tissues becomes more copious and runs some way down the tree. At this time it is often a conspicuous rusty-colour and no longer transparent but cloudy or opaque. Under dry conditions during the summer, this exudate dries to leave a dark, brittle crust near the point of exit in the bark (Figure 6).

Renewed bleeding may be seen later in the year, often in autumn. This suggests that pathogen activity is greatest under moist, mild conditions of spring and autumn.
After some months the centre of the bleeding bark patch may become cracked. In time, fruit bodies of wood-rotting fungi often appear on the surface of the dead bark, protruding out of the bark cracks (Figure 7).

![Figure 7. Bark cracks on trunk](image)

Sometimes, part of the crown will fail to flush, and later in the year the remaining foliage withers and dies. In trees with chronic dieback caused by the disease, the leaves may also be smaller, and seem thinner and more flaccid than the foliage of healthy trees. Over several years, and particularly if a tree has multiple bleeding cankers, the areas of dead phloem and cambium underneath the bleeding areas may coalesce and extend until they encircle the entire trunk or branch. When this happens crown symptoms become visible, typically consisting of yellowing of foliage, premature leaf drop and eventually, crown death.

The inner bark (phloem) under the bleeding patches is usually necrotic or dead, with an orange-brown colour which is often clearly mottled or zoned (Figures 8 and 9).
Figures 8 & 9. Mottled and zoned lesions in inner bark

Underneath the wood may be stained blue-black (Figure 10).

Figure 10. Black staining of wood under bark lesion

Sometimes white fungal mycelium (Figure 11) can be seen under the dying bark but this is usually indicative of Armillaria (honey fungus) or other decay fungi which invade the moribund tissue, and not the original cause of bark death. In these instances it is rarely possible to isolate the agent that originally caused the bleeding canker.
Sometimes *Armillaria* does attack healthy trees and causes stem bleeding, as it invades via the tree root system. However, in such cases the stem bleeding is likely to be confined to the root collar and lower stem.

### 6. Causal agent

Prior to the current upsurge in the incidence of bleeding canker, affected trees were invariably found to be suffering from attack by *Phytophthora*. However, although this continues to be the case in 5-10% of all trees that are examined, the majority of the horse chestnuts currently suffering from bleeding canker in the UK are not infected by *Phytophthora*. DNA analysis of the diseased tissue taken from a small number of trees has also failed to detect any *Phytophthora*.

Instead, the most frequently isolated agent appears to be a species of gram-negative fluorescent bacterium, most probably *Pseudomonas syringae pv aesculi*. Tests are now underway to confirm if this organism, (either singly or in combination with some of the fungi that have also been isolated from the bleeding cankers on horse chestnut), could be the cause of the current epidemic. Proving that a particular organism is responsible for causing a disease by inoculating it into healthy plants to confirm that it causes the disease and its associated symptoms, and then re-isolating the same organism from the infected tissue is known as satisfying Koch’s Postulates.

### 7. Summary

Confirming exactly what agents are causing horse chestnut bleeding canker is critical to any recommendations about effective control measures. Before embarking on any control measures on sites where there is a significant number of horse chestnuts, a survey to assess the number of affected trees is recommended. There is no chemical treatment currently available to cure or arrest the development of bleeding canker:
If the lesions become so extensive that the entire trunk is girdled, the tree will inevitably die and have to be removed and disposed of appropriately. If major branches are infected and show dieback they should be removed, because recently dead branches of horse chestnut may be susceptible to sudden fracture and drop as the wood dries out. Advice on the best time of year to prune is given in Arboriculture Note 117 (Lonsdale, 1993). However, many trees with trunk infections retain healthy-looking crowns and may not pose an immediate safety risk. Some may even survive for many years as disease progression can be very slow or even cease, and show signs of recovery as vigorous callus development occurs at the margins of wounds created when bark has been killed by the disease.

Our understanding about this disease is limited at present and this makes it difficult to make precise recommendations about the management of the disease and affected trees. In these circumstances the following course of action is recommended until better information is available. Current work is underway to confirm that the bacterium, *Pseudomonas syringae* pv *aescluli*, isolated from the cankers is the cause of the recent outbreak.

8. Acknowledgements

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9. References


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