Arboriculture Research Note [redacted]

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DIEBACK OF THE FLOWERING CHERRY, *PRUNUS* 'KANZAN',
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Summary

Many *Prunus* 'Kanzan' trees died in England and Wales following the very cold 1981/2 winter and subsequent cold winters. Circumstantial evidence suggests that the bacterium *Pseudomonas syringae pv. syringae* may be the cause. The disease exemplifies the wisdom of using a mixture of species or varieties in ornamental planting schemes.

Introduction

1. In the summer of 1982 the Forestry Commission Pathologists received an exceptionally large number of reports of the death of well established flowering cherry trees in gardens, in parks and along roadsides in England and Wales. As is usual each year, these included some cases of Honey fungus and *Phytophthora* root disease, Silver leaf and Bacterial canker but most proved to be a disease unfamiliar to the pathologists in its symptoms as well as in its severity and widespread nature. Furthermore all occurred on the common, double pink-flowered *Prunus* 'Kanzan'. Since this first outbreak, many further cases have occurred on a wide range of sizes of tree following each particularly cold winter.

The Symptoms

2. Affected trees grow and flower normally at the beginning of the season, then, usually in June, all the leaves on one branch or several branches wilt suddenly and turn brown. In the following few weeks, more and more of the crown may be affected, some whole trees finally dying. Most dead leaves remain firmly attached to the tree, hanging limp, only later gradually falling.

3. The death of leaves results from the death of the inner bark (phloem) and cambium of twigs and branches. (It may be necessary to cut down to the wood, through live outer bark, to detect this. Live tissues are white, dead tissues are brown). The wood underlying dead bark is unstained. No other symptoms have been noted. Often, little or no live bark remains on wilted branches. However, in some cases branches wilt because they have been girdled at their base and when they are first seen to be dying most bark is still alive. This commonly happens where infected bark extends down from a branch to involve the upper portion of the bole and consequently kills the bases of branches which arise from that part.

4. It seems likely that a disease organism initially invades a branch (where precisely is not clear) and then spreads down in the phloem or cambium into the base of neighbouring branches to kill these also.

5. In the following year, some affected trees will die but in others the parts which were still alive at the end of the first year may continue to grow healthily, suggesting that the disease organism has died out. Some of these trees may also produce new shoots from the rootstock - often a wild cherry.

The Cause

6. Many observers have likened the symptoms to fireblight *(Erwinia amylovora)* but the cherries are immune to fireblight. Others have compared it with Verticillium wilt *(Verticillium spp)* in that scattered branches have wilted rapidly and defoliation has been slow. However, the extensive death of bark and lack of wood staining is not typical of *Verticillium* wilt and no *Verticillium* has been isolated from any of the diseased trees.

7. The symptoms themselves and the rapidity with which they develop suggest that this is a bacterial disease; its first occurrence after the exceptionally cold 1981/82 winter and its resurgence after each of the severe winters since then further suggests that the bacterium involved might well be *Pseudomonas syringae* pathovar *syringae.* This is a known cause of bark killing in cherries and other trees, but the chief reason for suspecting this organism is the role freezing temperatures play in its development: they greatly increase the susceptibility of woody plants to infection and encourage the spread of this bacterium through the plant (Lindow, 1983; de Kam 1982). Reports of unusual outbreaks of this bacterial disease on apple trees in England also associated with the 1981/82 winter add weight to this explanation (Anon 1982).

8. This diagnosis remains tentative as even with the help of bacteriologists from other establishments neither this nor any other pathogen has been detected in the diseased tissues.

Control

9. No preventative measures can be suggested and by the time foliar symptoms appear in the summer, the bark will already have been damaged. Some infections die out spontaneously but many do not. To ensure that no further spread takes place it might be possible to cut off affected branches well below any dead bark or brown-stained cambium. However, where the lesion extends into the main trunk, as is so often the case, excision would usually involve an unacceptable amount of mutilation. Healthy *Prunus* 'Kanzan' close to diseased trees do not seem to run an enhanced risk of infection.

10. There is no association between 'the Kanzan disease' and pruning wounds. However, as a precaution against Silver leaf infection, fresh pruning wounds should be protected with either Pancil T (Rohm and Haas) or Binab T (Henry Doubleday Research Association). These products are available for professional use only. To minimise the risk of Silver leaf infection of unprotected wounds, pruning should be carried out in June, July or August.

The outlook

11. Why the disease should have broken out suddenly after the cold winter of 1981/82 in particular is obscure. However, it seems likely that future cold winters will continue to trigger further outbreaks, and it would be unwise, therefore, for any future planting schemes to rely too heavily on Prunus 'Kanzan'.

    Before using a fungicide always read carefully the manufacturers instructions on the label (including any accompanying leaflet) and _apply the chemical for the use at the rate and by the method recommended paying particular attention to aspects of safety.

Acknowledgement

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References

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