Diseases of Fruit in Orchards, Hedgerows and Gardens

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1. Introduction

One of us (DI) remembers that in the 1940s and early 50s, when he was living on the very edge of Birmingham, a great pleasure in the early autumn was a trip out to Worcestershire in his grandfather's old Austin to buy, at the orchard gates, bulging brown paper bags of fragrant early Worcester Pearmain Apples and Victoria Plums. What he did not know then, but does now, is that in addition to the annual harvest of edible fruits from orchards and gardens in the Wyre Forest area, the trees and shrubs that produce them and even the fruits themselves, also produce a rich harvest of fascinating plant diseases. We therefore propose that a focus for the Wyre Forest Study Group in 2025 should be: pathogens that cause diseases in orchards, especially abandoned and unsprayed ones, the hedgerows that surround them and private gardens.

Regular readers will already be familiar with the article (by RW) in last year's REVIEW, which dealt with the history of the orchards in Wyre (Winnall, 2023), including those still cared for and productive, many abandoned and a growing number recently restored or replanted. Edible Cherries constitute the principal pathogen hosts in the older orchards, but a diversity of other fruit hosts may be found associated even with these and especially in those still productive or recently restored or replanted. The hedgerows, particularly the older and therefore diverse ones that surround orchards and other enclosed areas of the forest, are also worth examining since Apples, especially crabs, Damsons, Sloes, wild Plums and their relatives often occur in these, as do berries, especially Gooseberries and Raspberries. And of course, gardens and allotments are also excellent places to find fruit host diversity.

During 2025, the Wyre Forest Study Group field meetings will include visits to a number of orchards during which members can gather evidence of pathogens amongst the fruit trees and hedgerows. Photographs and pressed specimens can be sent to RW for identification with help, especially with verifications, from DI. We hope to curate, for next year's REVIEW, a collection of images and reports from the 2025 collecting season. Below we provide a few examples of the sorts of diseases to look out for and photograph during the coming weeks and months.

We should add that we have said nothing about the control of fruit pathogens. Lack of space is the reason, but don't despair, for excellent advice may be found on the Royal Horticultural Society (RHS) website, which is free for all to access. Don't expect suggestions using chemical control, however, since the RHS does not agree with the use of these, for good environmental reasons, a policy with which we totally agree.

2. Blossom Wilt and Fruit Rots

When examined closely in spring, wilting of blossom, often accompanied by rotting of fruit spurs, may sometimes be seen on trees of Apple, Pear, Plum, Cherry and their close relatives such as Sloes, Nectarines, Peaches, Quinces and Apricots, especially in a wet season. These diseases are most often the result of infection by two fungal species in the genus *Monilinia*. One, *M. laxa*, commonly causes blossom wilt on pears and stone fruits, while a specialised form, *M. laxa, forma specialis mali*, can only infect apples.

Another species, *M. fructigena*, occasionally causes blossom wilt, but usually, from midsummer into autumn, is the cause of brown, spreading rots that later form light brown spore pustules, on the fruits of all the hosts listed above (Figure 1). The usual source of infection is damage of some sort, usually inflicted by pecking birds and later, on fully ripe or fallen fruits, by feeding insects, especially wasps (Figure 2). Infected fruits not consumed by birds or insects frequently dry out and are described as being mummified (Figure 3).



Figure 1. Apple fruits showing fruit rot. Pustules carrying spores (conidia) are clearly visible on one of the fruits. David Skydmore



Figure 2. Wasps feeding on an apple which has been pecked by birds. David Skydmore



Figure 3. A mummified pear on the tree. Pustules carrying conidia are abundant. David Skydmore

Soft fruits such as Strawberry, Raspberry, Blackberry, Gooseberry and Grape may suffer from a very different fruit rot at any time during the year, but especially so if grown in a high humidity glasshouse or outdoors during damp days when fruits are ripening. Usually, *Botrytis cinerea* (Ascomycota), the cause of Grey Mould, is the pathogen involved. The symptoms of infection are generally rotting of ripe fruits and other soft tissues, which become covered by a greyish-brown fuzz of mycelium bearing long stalks with branched heads carrying masses of rounded asexual, non-motile spores (conidia). These may easily be seen with a strong lens or low power microscope. A sexual phase has been described but is hardly ever seen on cultivated fruits.

Lovers of wine will know that this fungus also causes 'Noble Rot' of white wine grapes, which occurs when infection of ripe fruits happens during warm wet weather followed by a warm, dry period during which the grapes become 'raisined'. The dessert wines made from such grapes, when served chilled and slowly sipped, have an intense aromatic sweetness that is hard to resist!

3. Fruit Tree Rots

Honey Fungus Rot. In addition to pathogens that cause rotting of fruits, there are others that destroy whole fruit trees and shrubs. The easiest to recognise in the field is caused by Honey Fungus, not a single species but a diversity of *Armillaria* species in the Basidiomycota. The name derives from the fruiting bodies, honey coloured, often glossy toadstools, that appear in autumn at the base of infected trees and shrubs (Figure 4). Many species may become infected, but among the fruit producing genera *Malus* (apples and rarely pears) and *Prunus* (plums and cherries) are the usual victims. The *Armillaria* species grow as mycelium in the soil, often spreading from an infected tree to a healthy one by producing long, bootlace like structures called rhizomorphs, composed of bundles of hyphae with a blackish exterior, that can grow through the soil for considerable distances (Figure 5). They then transport nutrients from the infected tree to provide the energy required to infect the healthy one. First the roots of the infected trees are killed, and the fungus then grows between the bark and wood of the trunk, producing sheets of white mycelium and further rhizomorphs, eventually destroying the whole tree.



Figure 4. Honey Fungus *Armillaria sp.* fruiting bodies. Rosemary Winnall



Figure 5. Rhizomorphs of Honey Fungus *Armillaria sp.* Rosemary Winnall

Three other examples of diseases capable of destroying fruit trees are as follows (illustrations of the symptoms may be found on the RHS website).

Phytophthora root rot, caused by various *Phytophthora* species (Oomycota), may infect the roots of Currants, Blackberries, Apples, Plums and Cherries, but not Pears and Grapes. Infections occur most readily when the weather is wet since the asexual spores of the pathogens are motile and swim in water; thick-walled sexual oospores allow the pathogens to persist in the soil even after a host has been killed and removed.

Fireblight, originally imported accidentally from the USA during the 1950s, is caused by the bacterium *Erwinia amylovora*. Apples and Pears may become infected from late spring through to autumn. Infected foliage and flowers appear

to have been scorched by fire and bacterial slime usually oozes from the infection sites when the weather is wet. Cankers may also form, and the infected woody tissues take on a foxy-red colouration.

Silver leaf, caused by *Chondrostereum purpureum* (Agaricales, Basidiomycota), infects Plums, Damsons, Cherries, Apricots and occasionally Apples. One of us (RW) had to cut down their lovely Victoria Plum tree as a result of this devastating disease. The air-borne basidiospores, produced on bracket-like basidiocarps (Figures 6a and 6b), which have whitish, somewhat fluffy upper surfaces and purple to brown lower surfaces, become active in September and remain so until May the following year. The result is infection of the wood through wounds, especially those resulting from pruning, which turns dark, followed by a dramatic change in the foliage which develops a silvery sheen. Ultimately infected branches die and produce bracket-shaped basidiocarps on the dead wood. Since Plum and Cherry trees are especially prone to attack, it is recommended that these are not pruned until June.

Interestingly, the characteristic silvery leaves on infected trees are not caused by the pathogen itself, but by a toxin it produces. This is carried up to the crown in the sap from the infected wood and causes the tissues of the leaves to separate, forming airspaces, which give the leaves their silvery appearance.



Figure 6a. The fungus *Chondrostereum purpureum* causes Silver Leaf. Rosemary Winnall



Figure 6b. The brackets are the source of basidiospores that will initiate new infections. Rosemary Winnall

4. Scab of Apple and Pear

Perhaps the most familiar disease of fruit for most folk is Apple Scab and for those with a taste for the intensely flavoured, juicy texture of pears, Pear Scab. The disease may also infect related hosts, including *Sorbus* and *Cotoneaster*. The causal fungi, two species of *Venturia* (Ascomycota), *V. ineaequalis* on Apples and *V. pirina* on Pears, overwinter on dead infected leaves. In spring, the hyphae undergo sexual reproduction and the sexual fruiting bodies (ascocarps), appear, embedded just below the surface layer of cells. These are spherical with a neck and pore to the surface and their elongated, sac-like asci produce ascospores, each comprising two unequal cells, in the spring. These are ejected through the pores and those that land on healthy emerging leaves and young twigs of the hosts may infect them, producing a mycelium between the cuticle and the epidermal cell layer. The resulting symptoms are similar in both cases: defined areas of olive-green to brownish velvety spots that release airborne, single-celled asexual spores (conidia) which go on to infect further leaves of the same or adjacent trees and later in the season, the developing fruits. Infected leaves are often shed early, while the surface of infected twigs may crack, providing an entry point for other disease such as those responsible for cankers development.

On infected fruits brown or black scabby areas develop just below the cuticle, causing scab-like spots or blotches (Figure 7). If the attack is not heavy, the eating quality of the fruits is not affected significantly, although for commercial growers the unsightly appearance of the scabbed fruits makes them unsaleable. In the case of severe infections, the lesions may restrict the enlargement of the fruits, often leading to distortion and sometimes cracking, the latter providing an entry point for fruit rot pathogens (Figure 8).



Figure 7. Apple Scab *Venturia ineaequalis* on Apple. David Skydmore



Figure 8. Apple Scab *Venturia ineaequalis* on Worcester Pearmain. Rosemary Winnall

5. CANKERS, DISTORTED GROWTHS AND TUMOURS ON FRUITS AND FRUIT TREES

Apple canker. Apple and Pear canker, caused by *Neonectria ditissima* (Ascomycota), is a fungal disease that enters through wounds and cracks in the bark, pruning wounds or leaf scars in mid spring. Symptoms of infection are sunken, roundish to irregular patches of dead bark on twigs and branches. The disease grows and develops throughout the year and the scars sometimes completely girdle and kill small, infected twigs or slender branches. In extreme cases, cankers may develop on larger branches or trunks. These are often perennial, with the bark in the centre sometimes being destroyed completely, leaving the inner wood exposed. In such cases the bark sometimes tries to regrow, forming a raised edge around the infection site, but eventually the pathogen gains the upper hand, causing death of whole branches or young trunks.

The new infection-cycle begins each year with mycelium and red sexual fruiting bodies (ascocarps) of *N. ditissima* which have overwintered on existing cankers. On moist days in spring, the mycelium on young cankers produces multi-cellular, non-sexual, non-motile conidiospores on the ends of vertical branched hyphae. These are dispersed by splashing rainwater and initiate new cankers through leaf scars, damaged bark or young fruits, a process which continues throughout the summer.

The ascocarps, which are found on much older cankers, contain sac-like asci filled with two-celled ascospores. Also in spring, these may be forcibly ejected through the long neck of the ascocarp and then dispersed by the wind over long distances, or they may be extruded in a sticky mass and then splash dispersed for short distances by rainwater. In both cases infection through leaf scars, damaged bark or fruits is the result.

Pocket Plum. This disease results from an infection in summer of the young fruits of Plums, Damsons and related *Prunus* species by the fungus *Taphrina pruni* (Ascomycota). The consequence, from midsummer onwards, is soft, usually distorted and elongated hollow fruits in which the stones have failed to develop (Figures 9 and 10). Eventually the infected fruits become covered with white fungal hyphae, shrivel and fall from the tree.

On stems carrying infected fruits, it is not unusual to see thickening, deformed growth and occasionally the development of 'witches'' brooms comprised of clusters of slender living and dead twigs.

The infection cycle of *T. pruni* has not been studied in detail, but it is suggested that in late summer, asexual spores (conidia) formed by surface hyphae on infected fruits are carried by the wind and catch in healthy bark fissures and between bud scales. Once there, they grow over winter but do not infect until spring. The fungus then invades the plant tissues, causing them to become swollen and deformed in the first season. In subsequent years, blossom and developing fruits are invaded, resulting in characteristic Pocket Plums.



Figure 9. Pocket Plum *Taphrina pruni*, carrying conidia, on Blackthorn *Prunus spinosa*. Rosemary Winnall



Figure 10. Pocket Plum *Taphrina pruni*, carrying conidia, on Blackthorn *Prunus spinosa*. Rosemary Winnall

Peach Leaf Curl. A related *Taphrina* species, *T. deformans*, sometimes infects Peaches, Almonds, Nectarines and occasionally Apricots in spring, causing the disease Peach Leaf Curl in which the young leaves and sometimes fruits grow in a thickened, blister-like distorted way, often deep red in colour (Figure 11). Usually, the tissues are covered with a whitish bloom of asexual spore-(conidia)-bearing hyphae and before long fall prematurely. A second flush of new leaves may then form, but do not become infected.

As with Pocket Plum, the details of the life cycle have not been fully studied, but the causal fungus seems to overwinter in buds in a yeast-like form and then, in spring, as the buds begin to break, the tissues are penetrated, but not killed, and grow in the characteristic deformed way described above. Asexual spores from the surfaces of infected leaves then, if the weather is wet, become lodged in uninfected buds to begin the cycle again.



Figure 11. Peach Leaf Curl *Taphrina deformans* on Almond. Rosemary Winnall

Tumours and Galls frequently form on fruit trees, especially apples. Discovering the causes can be frustrating for the non-specialist since, in many cases, the cause is not known with certainty. In two cases it is, however, Burr Knot and Crown Gall which are described below.

Burr Knots are most often seen on Apple trees (Figure 12), although Quince may sometimes be affected too. The visible symptoms are rounded or knobbly outgrowths, sometimes covered with fissured bark or even short stubby adventitious roots, on trunks or branches. The cause is not a pathogen but seems to be a combination of a genetic predisposition of the cultivar or rootstock affected, combined with adverse environmental conditions such as shade or high humidity. The resulting tumours are perennial and enlarge whenever the tree is in growth. In the worst cases the infected tree may be stunted, or when the tumours become very large and heavy, branch breakage or other structural damage may occur.



Figure 12. Burr Knot on Apple. Rosemary Winnall

Crown Gall usually occurs on the roots or lower stems of Apples, Blackberries, Cherries, Currants, Gooseberries, Grapevines, Peaches, Pears, Plums and Quince. In these cases, the causes are soil-inhabiting, pathogenic strains of a bacterium, *Rhizobium radiobacter* (syn. *Agrobacterium tumefaciens*), which enter the tree or shrub through wounds on roots or stems (including graft unions), stimulating the over-production of growth substances ('hormones'). The result is that the tissues become 'transformed' and grow in an uncontrolled way to form tumours that continue to grow even after the bacterium itself has died out.

The mechanism for such transformation is that a small piece of non-chromosomal bacterial DNA (called a plasmid) passes from the pathogen and attaches itself to the DNA of the plant cells, causing them to commence tumour (gall)-formation and also to produce 'opines', derivatives of amino acids such as arginine, lysine and histidine, which only the bacterium is able to break down enzymatically to form the carbohydrates and nitrogen essential for its own growth.

Intense study of this remarkable three entity disease combination - pathogen, plasmid and host - led to the development of the controversial biotechnological technological procedures for introducing isolated pieces of DNA from other sources into plant cells during genetic manipulation (GM).

6. Mildews on leaves and fruits

Powdery Mildew. The fungi that cause powdery mildew diseases (Erisyphales, Ascomycota), cover the surfaces of the stems, leaves (Figure 13), petals and fruits of their hosts with a fine weft of pathogen hyphae that produce chains of asexual, non-motile spores (conidia). These are dispersed by wind to other hosts of the same species where they initiate new infections (species and strains of

the fungus are highly specific in their host-range). The infecting fungi are obligate biotrophs that must gain their nutrients by penetrating the epidermal cell walls of the living hosts with short feeding branches (haustoria). These become surrounded by the host cell membrane (plasmalemma) to create an interface for the transfer of sugars and other nutrients from one to the other. The result is usually stunting of the growth of the host, often accompanied by distortion of infected soft branches and leaves.

Later, pinhead-sized, black spherical structures may sometimes be seen among the surface hyphae on leaves or other infected structures. These are the closed fruiting bodies (ascocarps) and are often covered with microscopic hyphal appendages which may be straight, curled, forked or branched, according to species. The ascocarps are capable of remaining dormant for long periods, often over-winter. In due course there develop within the ascocarps, sac-like membranous asci, the number depending on the species. Within these the sexual spores develop, usually eight per ascus and when conditions are suitable for infection - warm and humid, but not wet - the ascocarps split open and the asci eject their ascospores and establish new infections.

Asexual infections may also overwinter, usually in infected buds. In spring, when conditions are suitable - warm and moist, not wet, as for ascospores - these may grow and produce asexual spores (conidia) which may be dispersed by wind to infect further appropriate hosts.

Being surface dwellers, powdery mildews are generally adapted to cope with a limited water supply, enabling them not only to survive on the host surface, but also to out-compete other surface-dwelling fungi and other organisms. As a result, the disease is usually most obvious in dry weather and most serious in the drier parts of the United Kingdom.

A very wide range of fruit hosts may be infected, some being listed here with the pathogen specific to that host being shown in parentheses: Apple and Pear (*Podosphaera leuchotricha*) (Figure 14), Cherry (*Podosphaera clandestia*), Plum (*Podosphaera tridactyla*), Strawberry (*Sphaerotheca macularis*), Black Currant and Gooseberry (*Podosphaera mors-uvae*) and Grape (*Uncinula necator*; syn *Erysiphe necator*) (Figure 15).



Figure 13. Powdery Mildew producing chains of air-borne conidia, on Courgette. Rosemary Winnall



Figure 14. Powdery mildew *Podosphaera leuchotricha* on Pears. Rosemary Winnall



Figure 15. *Uncinula necator* (syn. *Erysiphe necator*) producing chains of air-borne conidia, on Grapes. Rosemary Winnall

Downy Mildew

Downy Mildew infections are not seen on most of the fruits commonly grown in the UK, except for Grape Vine (*Vitis vinifera*). The cause of Downy Mildew on this species is *Plasmopara viticola* (a fungus-like organism belonging to the Oomycota), which often infects vines grown out of doors, especially if the weather is wet. The most obvious symptom of infection is the appearance of yellowish areas on the upper surfaces of infected leaves, often confined by the veins, together with the appearance of whitish 'mould' on the corresponding lower surfaces. The pathogen is an obligate biotroph, so may only grow on the living tissues of its host.

Plasmopara viticola survives (but does not grow) overwinter as thick-walled sexually produced

resting spores, oospores, in the tissues of leaves which have fallen and died during the previous autumn. These germinate to cause new infections in spring, the hyphae of which produce zoosporangia on branched, aerial structures. These, in turn, are dispersed by water or wind to release zoospores (swimming spores with two flagellae), on the same or adjacent vines, and these initiate further infections on tissues where free water is present. Later in the summer sexual reproduction occurs, resulting in thick-walled oospores which again survive in dead leaves until the next year. The oospores are long-lived and may lie dormant in the soil for many years if no suitable host is available when they are first formed.

An interesting aside is that Vine Downy Mildew was responsible for development of an early fungicide in the late nineteenth century. The story goes that French Vinyard owners sprayed their vines at the edges of plantations with a mixture of water, copper sulphate and quicklime to deter passers-by from stealing the grapes. An astute professor of botany noticed that not only did the treated vines retain their grapes but also remained free of Downy Mildew. With some refinement a copper sulphate-lime-water fungicide with the name Bordeaux Mixture was developed; not exactly good for the environment, or passers-by either, but effective in preventing infection by Vine Downy Mildew and its relatives such as Potato Blight (*Phytophthora infestans*)! Modern formulations are still in use today, but only under strictly controlled conditions.

7. Rusts

Rust fungi (Basidiomycota, Pucciniales; syn. Uredinales) are among the most common causes of diseases on garden plants. Many fruits may be affected, including Pear, Plum, Bramble and Raspberry. Rusts usually become apparent on these hosts from mid-summer onwards, especially in wet summers. As with Powdery Mildews, Rust fungi are highly specialised regarding host range, with different species or even strains only able to infect specific host species or cultivars. The most common symptoms are rusty spore-producing (orange, yellow, brown or black, depending on spore stage) pustules on the leaves and stems, in some instances accompanied by abnormal tissue growth.

Rust fungi are obligate biotrophs, which means they may only obtain the nutrients essential for growth from the living tissues of their hosts. The hyphae, which grow between the cells of the host, form short, usually club-shaped feeding branches (haustoria) which penetrate living cells where they become surrounded by the cell membrane (plasmalemma) and obtain water and the nutrients they require for continued growth from these and intercellular fluids. Haustoria may also secrete chemicals to modify the cells' metabolism to facilitate nutrient exchange.

The cells of Rust hyphae are, for a significant part of their life cycles, dikaryons. This means that each cell of their hyphae contains two haploid nuclei, each with a single set of chromosomes that carry the genetic information. Since each nucleus in a dikaryotic Rust cell is derived from a different parent, the cell is functionally diploid (the state of the single nucleus in the body of a plant or animal which contains two sets of chromosomes, one from each parent).

The life cycles of Rust fungi are very complex, usually involving several spore stages and frequently two different host plant species. Whatever the Rust species and whether the life cycle involves one or two host species, there are always two stages when some of the cells become monokaryotic, a state in which each cell contains only a single, haploid nucleus. These stages are concerned with sexual reproduction and genetic mixing. Thus, at one stage fusions of pairs of monokaryotic hyphal cells from different parents create new dikaryons. At another stage, fusion of the two haploid nuclei within these new dikaryotic cells, followed by a period of diploidy and then meiosis (a reduction division with genetic mixing) creates new monokaryons.

To see how such fascinatingly complex nuclear and genetic acrobatics work in practice, please read or re-read the paper entitled 'Life Cycles of the Rusts', by Rosemary Winnall, Carol Taylor & David S. Ingram, *Wyre Forest Study Group* REVIEW 2022, from which the above two paragraphs are reproduced near verbatim. Here, for reasons of space and practicality, we must concentrate in the following paragraphs only on the symptoms of Rust infections as seen in the field.

Pear Rust (*Gymnosporangium sabinae*; This spectacular Rust, which is common throughout mainland Europe, used to be seen only rarely in the United Kingdom. In recent years, however, for reasons that are not yet understood, it now occurs widely in the UK, including the Wyre Forest area. It is a long-life cycle Rust with two hosts, the many spore stages being distributed between them.

The life cycle begins on Pear Trees in early summer, where basidiospores produced on teliospores from

the alternate host, a Juniper species, infect the Pear leaves causing bright orange to reddish spots on the upper surfaces (Figures 16, 17, and 18). As the season advances, pycniospores are produced on the upper surfaces of the leaves, resulting in cross fertilization and formation of aecia on the outgrowths on the lower surfaces. The same sequence may also occur on fruits. The aecia next produce aeciospores that in late summer infect the alternate host, one of several Juniper species. Perennial stem swellings form on the Juniper host and the fungus overwinters in these. In spring, bright, orange coloured, jelly-like telial horns form on the stem swellings and these produce two celled teliospores (Figure 19). These, in turn, produce uninucleate, haploid basidiospores which reinfect a pear host and so the cycle begins again.



Figure 16. *Gymnosporangium sabinae* on Pear (pycniospores are produced on such lesions as the season advances). Rosemary Winnall



Figure 17. *Gymnosporangium sabinae* on Pear (the outgrowths on the lower surface of the upper leaf are where the aeciospores are produced). Rosemary Winnall



Figure18. *Gymnosporangium sabinae* on Pear (the outgrowths on the surface of the fruit are where the aeciospores are produced). Rosemary Winnall



Figure 19. Spent *Gymnosporangium sabinae* telial horns from which teliospores were once produced, on Juniper. Rosemary Winnall

Other fruit Rusts

We illustrate below three more common fruit Rusts.

Plum and Damson Rust (*Tranzschelia discolor*). The images are of Damson (*Prunus domestica* subsp. *insisita*) leaves (Figures 20a and 20b). A long-life cycle Rust, the alternate hosts being *Anemone coranaria* or *A. x fulgens*.



Figure 20a. Rosemary Winnall



Figure 20b. Rosemary Winnall

Figures 20a and 20b. Damson leaves with spore pustules of the Rust *Tranzschelia discolor*: a, lower surface with yellow urediniospore lesions and dark brown teliospores lesions; b, upper surface with yellow spots corresponding to the lesions. Rosemary Winnall

Bramble Rust (*Phragmidium violaceum*).

This Rust has a long life cycle, but without an alternate host species, all spore stages being produced on a single host, Bramble *Rubus fruticosus* aggregate (Figure 21). The aecia and aeciospores form on leaves and stems in reddish-brown lesions with a violet margin; the urediniospores, which follow, are yellow-orange; and the teliospores are chestnut-brown (Figure 22).





Figure 21. *Phragmidium violaceum* on Bramble (*Rubus fruticosus* aggregate): a & b, the upper surface of a leaf with aecia, from which aeciospores are produced, shown at different magnifications. Rosemary Winnall





Figure 22. *Phragmidium violaceum* (Bramble Rust): a & b, Teliospore lesions shown at different magnifications on the lower surfaces of host leaves; c. elongate, four-celled teliospores viewed with a low-power microscope. Rosemary Winnall

Bramble Rust (*Kuehneola uredinis*). This Rust completes its life cycle on a single host, Bramble (*Rubus fruticosus* aggregate Figures 23a and 23b). Spermagonia and aecia producing aeciospores occur on the upper surfaces of the leaves, while urediniospores are formed later on the lower surfaces.







Figure 23. *Kuehneola uredinis*: a, Aecial lesions on the upper surface of a host leaf; b, low-power microscopic image of aecial lesions producing aeciospores. Rosemary Winnalll

Raspberry Rust (*Phragmidium rubi-idaei*). This Rust completes all stages of its life cycle on Raspberry (*Rubus idaeus*; Figure 24). Yellow pycnia and aecia, the latter forming aeciospores, develop on the upper surfaces of the leaves in early summer. These are followed, as the season progresses, by orange urediniospores which form on the lower surfaces of the leaves, and these spread the disease widely to other canes. Finally, towards the end of the season, dark brown-black teliospores form on the lower surfaces of the leaves. The fungus probably overwinters as teliospores on dead leaves.



8. Conclusions (Rosemary Winnall)

In this paper we have illustrated some of the diseases of fruit trees and soft fruits known to occur in Wyre Forest orchards and gardens. The challenge for this coming season is to locate these and others, to record them photographically and wherever possible, press and label specimens, so that these can be verified and added to our dried collection that we hold from previous years. Please note that some symptoms may be caused by other pathogens such as viruses or insects but these should still be recorded for future reference.

We thank David Skydmore and Cherry Greenway who have kindly allowed us to use some of their images. I would personally like to thank David Ingram for sharing his knowledge and enthusiasm for plant pathogens over many years, for kindly verifying species from specimens and spore photographs, and for encouraging us to continue our studies in and around Wyre.

9. Further Reading

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