Native Plant Pathogens in the Wyre Forest: a proposed survey

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Plant pathogens: the enemy?

Plant disease epidemics may have immense ecological or agricultural consequences, often threatening native ecosystems or food security, respectively. Such threats frequently lead to headlines in the popular press, as this in the *Guardian*, August 2019, concerning native trees: "Ash dieback is just the start of killer plagues threatening Britain's trees", by George Monbiot, who continued: "Deadly diseases are marching across Europe unchecked". Scientific journals report similar stories, for example this threat to food security in Nature, 24th September 2019: "CRISPR [a gene-editing tool] might be the banana's only hope [in the Americas] against a deadly fungus [Fusarium wilt, Tropical Race 4]. Researchers are using CRISPR to boost the fruit's defences and prevent the extinction of a major commercial variety [Cavendish]."

Breaches of biosecurity – in the present context, the movement of a plant disease from a region where it is established to another where it does not exist – are not new phenomena, however, although we have recently become more aware of them since their frequency is increasing, mainly as a result of a massive expansion of the international trade in plants and plant products (see Bebber et al 2014). However, as long ago as the 1970s, the import into Britain on infected timbers from Canada of a new strain of Dutch elm disease, *Ophiostoma novo-ulmi,* led to the almost complete eradication of native elm trees from the British countryside (Harwood, et al, 2011). As a result, those born after the late 20th century will never know the English landscape as Constable painted it. And even further back, on 13th September 1845, Dr. John Lindley, editor of the *Gardener's Chronicle,* held up printing to insert the dramatic announcement: "the potato murrain [potato blight; *Phytophthora infestans*] has unequivocally declared itself in Ireland. The crops about Dublin are suddenly perishing … where will Ireland be in the event of a universal potato rot." The Irish potato famine had begun, caused by importation from the Americas, via mainland Europe and England, of infected tubers (Ingram & Robertson, 1999).

Infection by most plant pathogens is closely related to the prevailing precipitation and temperature, thus climate change is an additional factor that should be taken into account in considering breaches of biosecurity. The consequences of the current pace of climate change are difficult to predict, but may well allow some, perhaps many alien pathogens to flourish in regions where a few years ago ambient temperatures and/or rainfall would have inhibited their growth (e.g. Helfer, 2014).

Despite events of the past, however, and although in the UK we are constantly made aware of disease threats to native trees, currently we rarely see epidemics here of plant disease in major agricultural food crops - such as cereals and potatoes - because modern plant breeding has given us a range of disease resistant cultivars and biochemistry a range of efficient fungicides. Even so, plant diseases and pests may lead to c. 30% losses of crop productivity and the threat of an epidemic is ever present. Such an event might occur at any time when there is a dramatic genetic or climatic change, for example, and the delicate balance between a host and a pathogen is upset in a fundamental way. The sudden mutation of a pathogen to overcome a resistant cultivar, especially if accompanied by breaches of biosecurity, may, moreover, enable a new, virulent pathogen to spread rapidly because most modern agricultural crops are widely planted genetic clones. Such was the case recently with strain Ug99 of the wheat rust pathogen *Puccinia graminis* f.sp. *tritici,* which was first noted in Uganda in 1998 and whose descendants are now known to have spread by wind and/or human aid to many other countries in Africa and Asia (Bebber et al, 2014).

Catastrophic events in the past and currently on our own doorstep in the Wyre Forest in the form of, for example, Acute Oak Decline, a syndrome of unknown origin and possibly caused by a complex of bacterial species and the beetle *Agrilus biguttatus*, have conditioned us to see plant pathogens as enemies to be eradicated.

I propose, however, that the diversity of *native* plant pathogens in the Wyre Forest be surveyed, cherished and the threats to it recorded.

The reason for making this counter intuitive proposal is that the behaviour and significance of native plant pathogens in natural and semi-natural ecosystems – a meadow or forest, for example – is very different from what I have been describing above. Agricultural plant pathologists have long known that disease spreads more slowly in mixed plantings, for example of different cultivars of barley or rice, each with different resistance genes (Wolfe, 1985 & 2000), or even of different species, such as strips of crops interspersed with rows of fruit trees, and so it is with natural and semi-natural ecosystems, but more so. It is a truism that most plants are resistant to, or escape infection by most potential pathogens. This is because most

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ecosystems are complexes of many different plant species exhibiting a variety of life forms, seasonal cycles and genetic variants, the latter especially with regard to disease resistance, and have been co-evolving with their equally diverse pathogen populations, frequently with regard to pathogenic virulence, for millennia. In doing so, they have achieved a balance between limited loss of host productivity as a result of infection and concomitant survival of *both* host and pathogen populations. The diversity of both hosts and pathogens in natural and semi-natural ecosystems is thus the key to the survival of both.

If one looks at almost any ecosystem, like those of the Wyre Forest area, closely, it will be found that plant pathogens, as their hosts, are present in great diversity. Moreover, they are essential components of the ecosystem as a whole, often helping to maintain the delicate balance among the complex of species comprising it and preventing any one species becoming over vigorous (Burdon, J.J., Thrall, P.H. & Ericson, L., 2006; Alexander, 2010; Beever, J.D., Mangan, S.A. & Alexander, H.M., 2015). Such native pathogens may help to control the growth of their host populations in various ways, for example by: killing individual plants, as with many soil-borne pathogens; or by reducing plant growth and seed set, qualitatively or quantitatively, as many leaf pathogens do; or by reducing seed production by direct emasculation or abortion, as with infections of anthers or ovaries. This is, of course a gross over-simplification, but the papers by Burdon, *et al*, Alexander, and Beever *et al*, above, will provide greater detail and depth. Somewhat old papers by Ingram (1999 & 2002) will additionally provide a brief summary of other biological, scientific and commercial reasons why plant diseases in natural and semi natural ecosystems are worthy of study and conservation.

The consequences of the balance between plants and pathogens being disturbed may be seen when a non-native plant species, freed from the constraints of most of its normal pathogen load, is introduced to a new country where it makes rampant growth, soon becoming invasive, as with Himalayan Balsam (*Impatiens glandulifera*) in the UK. However, to further emphasise the point, help in the form of a native Himalayan Rust disease may soon be at hand, for scientists led by the agro-research organization CABI (himalayanbalsam.cabi.org) have found that a previously unidentified Rust, *Puccinia komarovii var. glanduliferae*, that attacks only Himalayan Balsam in its indigenous range in the Himalayan foothills, may provide an effective biological control for this pernicious alien in the UK landscape.

If, then, the diversity of native plant pathogens in the Wyre Forest is to be cherished and recorded, conducting an initial survey – a census of the pathogens present and their conservation status - might be a good way to begin. It must be recognized, however, that to do this thoroughly would require the work of a dedicated team over a very long period of time. It is essential, therefore, that careful plans are laid to do the job in stages, with each stage representing an achievable goal. This in itself is not an easy task, for the taxonomic diversity of plant pathogens is extremely wide and includes large numbers of Viruses, which have no cellular structure, and an immense range of cellular species spread across four Kingdoms: Monera (seven major genera of bacteria include plant pathogens); Protozoa (often water borne, plasmodial organisms and chytrids), Chromista (fungus-like, hypha[3]-forming organisms, with cellulose as the main structural component of the hyphal walls and a largely diploid life-cycle); Fungi, again hyphal, with chitin as the major structural component of the hyphal wall and a predominantly haploid life-cycle; and Plantae, flowering plants partially or completely pathogenic on other plants, as with mistletoes. Members of these groups, moreover, cause disease in plant groups as diverse as angiosperms, gymnosperms, pteridophytes, mosses, liverworts, and algae.

Where to start then?

I suggest, first, that the list of groups for initial survey is confined to pathogenic *micro-organisms* since parasitic flowering plants will already have been surveyed along with their hosts. Of the rest, I suggest that Viruses and members of the Kingdoms Monera and Protozoa are also eliminated for the present as either requiring complex laboratory facilities for study or not being easily identified initially by eye. Further focusing of the list, however, requires a consideration of the diversity of the symptoms caused by the pathogenic members of the two remaining groups – Chromista and Fungi - which is dictated by their 'trophic strategies' (how they acquire nourishment), as follows.

Trophic strategies of Fungus-like and Fungal plant pathogens

Note: the categories listed represent a continuum, with no hard and fast divisions between them.

Based on accounts by Ingram & Robertson (1999) and Ingram (2002)

1. Holobiotrophs

Normally grow only in living host tissues. Some grow only between host cell walls, gaining nutrients either from

[3] *Hypha* (plural *ae*; from the Greek *hyphe* = a web): the microscopic tube-like filamenta that are the basic body form of most Fungi and fungus-like organisms.

Fig. 1. A holobiotroph, *Kuehneola uredinis* (Pale Bramble Rust), on *Rubus fruticosus* agg. Note the numerous aecia, producing rusty-orange aeciospores[4], on the upper surfaces of the otherwise intact green leaves.
Image by Rosemary Winnall

the intercellular fluid or by a combination of this and via intracellular (within cells) hyphae or short feeding branches (haustoria), which penetrate the cell walls and invaginate the plasmalemma to form an intimate association with the protoplast. Holobiotrophs are usually host species specific and most are obligate parasites, unable to grow on artificial media in the laboratory except under highly specialized conditions.

Examples of symptoms include: general stunting of the host as carbohydrates are diverted to the infection site; premature ageing (senescence); rusty coloured spore pustules erupting from living leaves and stems, often with green islands of chlorophyll retention around them as the rest of the leaf senesces (Rusts; Uredinales [syn. Pucciniales], Basidiomycota; Figs. 1 & 2); black spore pustules erupting from living leaf and stem surfaces or flower parts (Smuts; Ustilaginales, Basidiomycota; Figs. 3 and 11); spores erupting over the living leaf surface from hyphae growing within the tissues (Downy Mildews; Peronosporaceae, Oomycota in the Chromista; Fig. 4); the living leaf surface covered with a fine weft of hyphae producing spores all over its surface (powdery mildews; Erisyphales, Ascomycota; Figs. 5 and 10); tumours, galls (e.g. Fig. 6), distortion of infected tissues, witches'

Fig. 2. A holobiotroph, *Uromyces muscari* (Bluebell Rust), on a senescing leaf of *Hyacinthoides non-scripta*. Note the pustules of rusty-brown teliospores[5] (syn. teleutospores), surrounded by green islands of chlorophyll retention.
Image by D.S. Ingram

[4] From the Greek *aikia* = injury + *sporos* = a seed

[5] From the Greek *telos* = end + *sporos* = a seed

Fig. 3. A holobiotroph, *Ustilago violacea* (Anther Smut of Red Campion), on *Silene dioica*. Note the pustules of black ustilospores (syn. teliospores) on the anthers of the otherwise intact flowers.
Image by Debbie White, formerly of the Royal Botanic Garden Edinburgh

brooms of proliferating twigs (various pathogen groups); a 'hypersensitive' response (necrotic flecking) around the point of infection of a plant carrying a gene for resistance to a pathogen (usually a Rust, Powdery Mildew or Downy Mildew), the infecting mycelium being restricted to and often destroyed in the dead cells.

2. Hemibiotrophs

These grow for the beginning of their pathogenic cycle in the living tissues of host plants, on which they produce dispersal spores, later causing rots in tissues they have killed and digested with toxins and cell wall degrading and other enzymes. In most cases they then produce the sexual, resting spore stages of the life cycle on the dead tissues. Most may be grown in culture in the laboratory, but in nature, unless already established within host tissues, are poor competitors with other micro-organisms and must survive as resting spores or structures.

Symptoms caused by species with a long biotrophic phase may include scabby host deposits of corky or pigmented cells around or over the infection sites (e.g. Apple Scab; *Venturia inequaelis*, Ascomycota); or cankers composed of infected, slowly dying tissues and sporulating tissues (various

Fig. 4. A holobiotroph, *Bremia lactucae* (Lettuce Downy Mildew), on a leaf of *Lactuca sativa*. Note the extensive sporulation of the pathogen on the undersurface of the young, green leaf.
Image by D.S. Ingram

Fig. 5. A holobiotroph, *Erysiphe alphitoides* (syn. *Microsphaera alphitoides*; Oak Powdery Mildew), on young leaves of *Quercus robur*. Note the dense weft of spore-bearing surface mycelium on the otherwise green leaf tissues. Image by D.S. Ingram

pathogen groups).

Symptoms caused by species with a shorter biotrophic phase, perhaps of a few days, may include a period of sporulation over the leaf surface from an internal mycelium, followed by blackening and collapse of the infected tissues with the formation of sexual resting spores or structures as the tissues decay and rot (e.g. Potato Blight; *Phytophthora infestans*, Oomycota; Fig. 7).

Symptoms caused by species with a very short biotrophic phase (various pathogen groups) of only a few hours, appear to all but the specialist as the result of infection by a necrotroph (see below).

3. Necrotrophs

Normally grow in host tissues they have killed and digested with toxins and cell wall degrading and other enzymes. They usually form dispersal spores on the dying and dead tissues, later forming resting spores or structures as nutrients become exhausted. They often have a wide host range and in the absence of a host, may infect and compete with other necrotrophs on plant tissues these have killed. They may usually be cultured easily in the laboratory on simple synthetic media.

Fig. 6. A holobiotroph, *Puccinia smyrnii* (Alexanders Rust), on *Smyrnium olusatrum*. Note the aecia, producing rusty-orange aeciospores, on galls induced by the pathogen on the developing fruits of the host.
Image by Rosemary Winnall

Fig. 7. A leaf lesion of a hemibiotroph, *Phytophthora infestans* (Potato Blight). Note the older, necrotic zone in the centre of the lesion, surrounded by a haze of emerging, spore-bearing hyphae on the recently colonized, green edge of the lesion.
Image courtesy of the Scottish Crop Research Institute, Dundee (now part of the James Hutton Institute)

Examples of symptoms include: 'damping-off' of seedlings (many Ascomycota and Peronosporales); vascular wilts (many Ascomycota and Peronosporales); twig blights, target spots (Fig. 8), rots of fruits or tubers; cankers of dead tissue on woody structures; or rotting of woody roots and stems, as with tree rots (many Basidiomycota, Ascomycota and Oomycota).

4. Saprotrophs

These are non-pathogens important in breaking down and recycling already dead tissues and are outwith the present study.

In a large, complex area of habitats such as the Wyre Forest, the only way to proceed in the face of such a diversity of trophic strategies and symptoms is to focus yet further, ideally on groups of pathogens that may easily be identified initially with the naked eye and then usually further identified with straightforward magnifying equipment.

Therefore, necrotrophs and hemibiotrophs with a short biotrophic phase may, I suggest, be eliminated because: they are relatively non-specific and therefore

Fig. 8. A necrotroph, *Coniothyrium hellebori*, causing Target Spot disease on *Helleborus niger*, Christmas Rose.
Image by Debbie White

not easily identifiable through association with a particular host; and the diseased tissues cannot easily be stored and often include not only the causal pathogen, but also secondary invaders, with the true cause of the disease only being identifiable by culture on selective media.

Among the holbiotrophs, and hemibiotrophs with a medium to long biotrophic phase, there are four clearly defined groups that include large numbers of species and hosts and satisfy our other criteria. These are: the Downy Mildews (Chromista, Peronosporaceae), the Powdery Mildews (Fungi, Ascomycota, Erisyphales), the Rusts (Fungi, Basidiomycota, Uridinales [syn. Pucciniales]) and the Smuts (Fungi, Basidiomycota, Ustilaginales). Of these, the best studied in the UK are the Rusts and Smuts, and this, I suggest, is where to begin.

Homing-in on Rusts and Smuts

Rusts and Smuts are normally easily spotted by eye, easily stored by pressing and drying infected host material, easily identified initially with a hand lens, host specific, with usually only a small number of species or subspecies occurring on any one host species, and finally, relatively easy to examine with a simple transmission light microscope. Both groups are reasonably well documented and both have recently been surveyed in Wales and their Red Data List status determined by the Welsh Rust Group. The excellent publications arising from this work – perfect examples of the challenges to be faced in the Wyre Forest – are available free of charge, on-line, as follows.

Woods, R.G., Stringer, R.N., Evans, D.A. & Chater, A.O. (2015) Rust Fungus Red Data List and Census Catalogue for Wales. A.O. Chater, Aberystwyth (www.aber.ac.uk/waxcap/downloads/).

and

Woods, R.G., Chater, A.O., Smith, P.A., Stringer, R.N. & Evans, D.A. & (2018). Smut and Allied Fungi of Wales, A Guide, Red Data List and Census Catalogue. A.O. Chater, Aberystwyth (www.aber.ac.uk/waxcap/downloads/).

I am duty-bound, but embarrassed to report, that the Welsh Rust Group recommend that "Ingram and Robertson [1999] provide … [two] of the most readable introduction[s] to the [Rusts and Smuts] in Chapter[s] 9 (*A Treacherous, Mutable Tribe – The Rusts*) and 10 (*The Dark and Secretive Smuts and Bunts*) in their [New Naturalist] book *Plant Disease*".[6] I might add that further chapters in this book provide introductory accounts to all the other groups of pathogens mentioned in the present paper. Beware, however, for the scientific names of many pathogens have changed since this book was written.

Other, excellent introductory material on the Rusts is provided by Larry J. Littlefield in *Biology of the Plant Rusts – An Introduction*, Iowa State university Press, Ames, Iowa (1981).

Although there is no up to date, well-illustrated identification guide to British Rust Fungi in print, the monumental book by Ellis & Ellis, *Microfungi on Land Plants: An Identification Handbook, 2nd edn.* (2017) is an invaluable guide to all plant pathogenic fungi in Britain, including Rusts and Smuts. The fungal descriptions and many of the illustrations are grouped by host genus, so if the host can be identified, the user is well on the way to identifying the pathogen.

The most comprehensive monograph on UK Rusts is *British Rust Fungi*, by Malcolm Wilson & Douglas M. Henderson, published by Cambridge University Press in 1996. Fortunately, a reprint of this old but invaluable guide is currently available from the publishers. Unfortunately, however, although the book is illustrated with line drawings of many rust spore stages, the identification key is not easy to use because it is based on the description of one spore stage only - the teliospore – which is frequently not present on diseased plants.

I have found an easy to acquire and valuable rust identification booklet to be Douglas M. Henderson's *The Rust Fungi of the British Isles – a Guide to Identification by Their Plant Hosts*, published by The British Mycological Society (2004), now downloadable from: www.aber.ac.uk/waxcap/links/. Woods et al (2015) recommend that it be used in conjunction with Malcolm Storey's "excellent" images at: www.bioimages.org.uk.

Finally on the Rusts, Woods et al (2015) suggest a modern volume of which I have no experience, namely A. Termorshuizen & C.A.Swertz's *Roesten van Nederland*, Termorshuizen (2011). This is available with descriptions of species in English as well as Dutch. Woods (personal communication) has recently suggested that Klenke, F. & Scholler, M. (2015), *Pflanzenparasitische Kleinpilze*, Springer Spectrum, Berlin is in his opinion now the best Rust book available. It is written in German, but can be purchased as an electronic version and perhaps run through a translation programme.

Unfortunately, there is no recent guide to the Smut fungi in print. However, *Ustilaginales of the British Isles*, by J. Mordue & G.C. Ainsworth, CABI (1984) is currently available from the publisher, although the

[6] Now out of print, although print-on-demand copies are available from the publishers. Second-hand paperback copies may, however, be available from second-hand book dealers.

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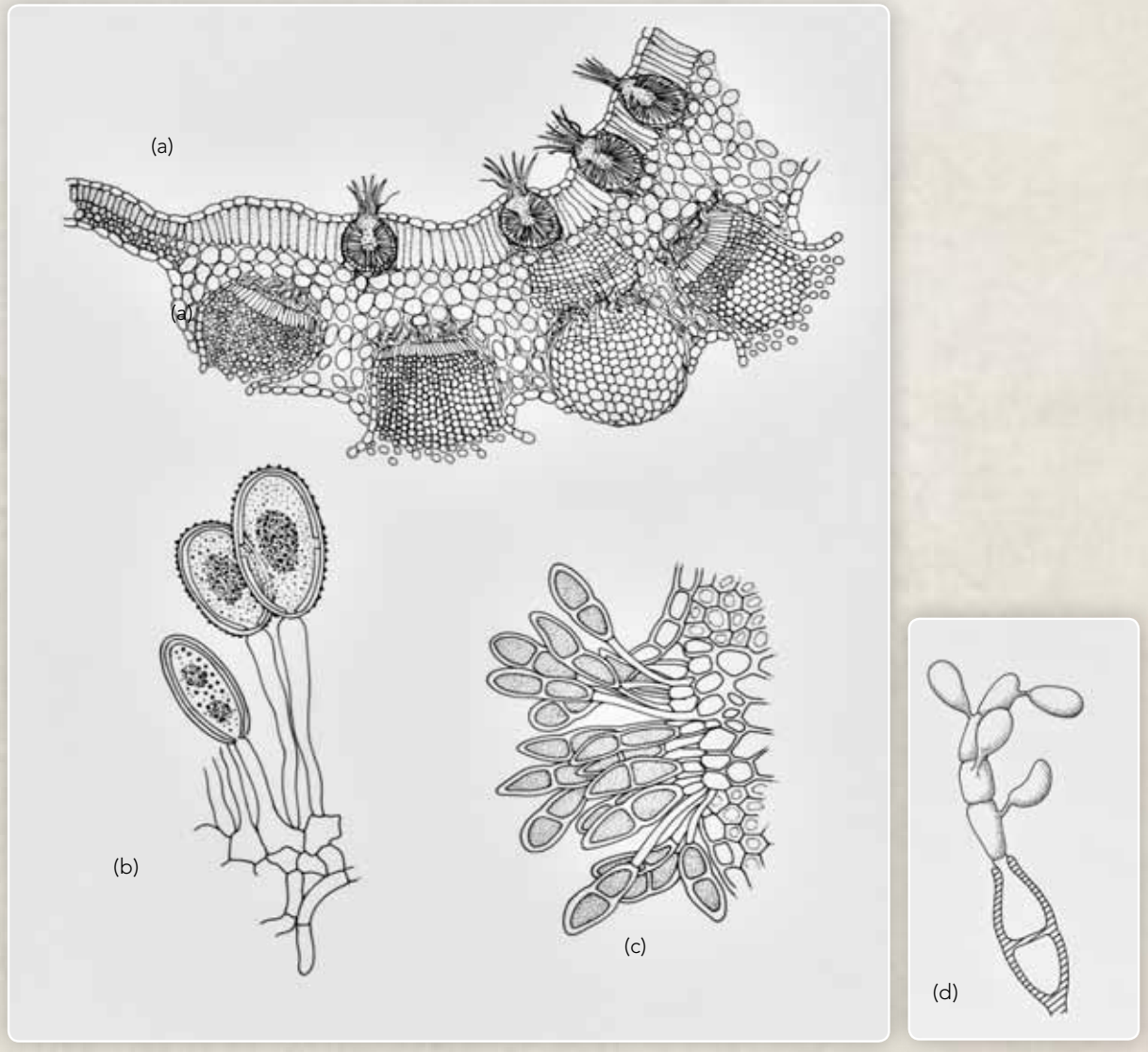

Fig. 9. The life cycle of *Puccinia graminis*, Black Stem Rust of grass species (and cultivated cereals):

(a) The cycle begins when uninucleate, haploid basidiospores from a grass host (see [d] below) infect the first alternate host, *Berberis vulgaris* and form intercellular hyphae with one haploid nucleus per cell (i.e. they are monokaryotic); these hyphae then form flask-shaped pycnia[7] (depicted here as they usually occur, embedded in the upper surface of the leaf); this is followed by formation of receptive hyphae and pycniospores, the latter, in a slimy secretion, being carried by insects to other *B. vulgaris* leaves; if these leaves are also producing pycnia with receptive hyphae, the pycniospores may fuse with them to form hyphae with two haploid nuclei per cell (i.e. they are dikaryotic); the dikaryotic hyphae grow within the tissues and finally produce rusty-orange aecia, containing chains of dikaryotic, rusty-orange aeciospores (depicted here as they usually occur, on the lower surface of the leaf).

(b) The aeciospores are dispersed by moving air and infect leaves of the second alternate host, a grass species, producing dikaryotic intercellular hyphae in the tissues and then rusty-brown coloured pustules containing dikaryotic, stalked, rusty-brown uredospores[8] (syn. urediniospores), depicted here emerging from the leaf surface (note the dark areas of spore cytoplasm do not represent nuclei). The uredospores may either re-infect other parts of the same grass plant, or be dispersed by wind to infect other plants of the same species.

(c) The dikaryotic hyphae resulting from the infection of the grass host next produce almost black pustules containing two-celled, dikaryotic, thick walled, blackish teliospores (syn. teleutospores), depicted here emerging, as they often do, from the surface of the grass stem.

(d) The teliospores next enter a resting period, often over winter or during a dry spell, followed by germination to form one dikaryotic basidium[9] per cell; as the basidium emerges, nuclear fusion occurs, immediately followed by meiosis and the formation of four uninucleate (i.e. monokaryotic), haploid basidiospores (as depicted here); finally, the basidiospores are dispersed and infect leaves of *Berberis vulgaris*, the first alternate host, to begin the cycle again (see [a] above).

For further information see Ingram & Robertson (1999), pp. 176-200; drawings by the late Mary Bates (after Anton De Bary, 1887).

[7] From the Greek *pyknos* =close-packed.
[8] From the Greek *urere* =to burn + *sporos* = a seed.
[9] From the Greek *monon* = alone, or *dis* = double + *karyon* = a nut.

even earlier *The British Smut Fungi*, by G.C. Ainsworth & K. Sampson, Kew (1950), is currently out of print and second-hand copies difficult to find. However, Malcolm Storey's website (see above) includes many excellent images of named Smuts.

Life cycles of the Rusts & Smuts

The Rusts are so called because of the rich colours of their spore stages, ranging from ochre and orange to almost black, reminiscent of the colours of oxidised iron (Figs. 1, 2 & 6). They comprise an attractive and very widespread and large group of highly specialized holobiotrophic pathogens with a great capacity for genetic change. Their hyphae are divided into cells, each with either one or two haploid, complementary nuclei (monokaryons or dikaryons; - see Footnote 9 and Fig. 9). Also, like many invertebrate parasites of vertebrates, they have complex life cycles with a number of different spore stages produced in sequence, often on two alternate hosts (Fig. 9). The hyphae grow between the living cells of the hosts and normally produce haustoria. Most are obligate parasites, although a few species may be grown with difficulty in culture. Many rusts, Black Stem Rust of Wheat (*Puccinia graminis*) in particular, are major pathogens of cereal crops worldwide.

Three different types of Rust life-cycles are known: short cycle and autoecious[10], with a reduced number of spore stages (e.g. *Uromyces muscari* (Duby) Niessl on *Hyacinthoides non-scripta* (L.) Chouard ex Rothm.); long cycle and heteroecious[11] (e.g. *Puccinia graminis* (Pers.), which has several different spore stages distributed between two separate host species; and long cycle and autoecious (*Puccinia violae* (Schumach.) DC. on *Viola* species). The spore stages of one of these, *Puccinia graminis*, are illustrated in Fig. 9.

The Smuts (Ustilaginales; see Figs. 3 & 8), named after their almost black, powdery resting spores or ustilospores (equivalent to the teliospores of Rusts and also called teliospores, chlamydospores or brand spores), which in a mass resemble the black carbon 'smuts' produced by steam engines. The Smuts are dramatic, numerous and like the Rusts, holobiotrophic members of the Basidiomycota, but differ in that most have a systemic phase, when they are not apparent to the naked eye. The hyphae are dikaryotic and grow between and through the living cells of the host, without haustoria. Also, the smuts have simpler life cycles than the Rusts, with only two or three spore stages – ustilospores, sporidia and sometimes, secondary sporidia - on a single host. They are relatively easy to grow in culture and have been widely used in genetic research. Some cereal Smuts are referred to as Bunts, a reference to the fat, pouter pigeon-like appearance of infected grains. The Smuts and Bunts were once common in agriculture, their sporulating lesions arising from the systemic mycelium, usually in the flower heads of cereals in place of grain. However, control is now so effective that they rarely cause a problem in the UK. Fortunately, however, Smuts are plentiful on wild plants.

In wild Smuts, as in agriculturally important species, the ustilospores form in pustules on infected plants. These usually develop from systemic or partially systemic hyphae and are produced in large numbers from (depending on species): stems or leaves (e.g. *Urocystis eranthidis* (Pass.) Ainsw. & Sampson on *Eranthis hyemalis* (L.) Salisb.); or flower parts (e.g. *Microbotryum lychnidis-dioicae* (Liro) G. Demi & Oberw. on *Silene dioica* (L.) Clairv. (Fig. 3) and *Ustilago avenae* on *Arrhenatherum elatius* (L.) P.Beauv. ex J. Presl & C.Presl); or very occasionally, roots. A new infection cycle is then usually initiated either by ustilospores present on seeds or in the soil germinating to infect emerging host seedlings, or ustilospores from the soil or infected plant material being blown by wind and infecting actively growing aerial parts of potential hosts, such as young shoots or flower parts. The details of Smut reproduction and infection, involving fusion of haploid nuclei, meiosis, restoration of the dikaryon and penetration of host tissues, vary greatly from species to species (see Webster & Weber, 2007, Woods et al, 2018 and Ingram & Robertson, 1999), but usually involve the ustilospores (teliospores) first producing sporidia, and sometimes secondary sporidia or budding, yeast-like growth, before penetration occurs. The resulting intercellular mycelium usually becomes either dormant for the winter, or fully systemic in the growing plant, before producing, the following season, new pustules whose ustilospores will initiate the cycle again.

Making a plan for Wyre

This will depend on how many people wish to be involved in the project and so is best left until after the AGM on 20th March 2020. Rosemary Winnall has very generously agreed to act as local convenor. Since I live in Scotland, however, I shall only be able to visit from time to time, but hope usually to be available by post or email to answer queries or to try to identify difficult specimens.

In recording species it will be important always to carry a hand lens in the field (ideally with a simple transmission microscope as back-up at home or in a

[10] From the Greek *autos* = self + *oikos* = home; a rust which completes its life-cycle on a single host species.
[11] From the Greek *heteros* = other + *oikos* = home; a rust that must infect two host species in order to complete its life cycle.

group facility). Each host/fungus species combination to be recorded will need to be photographed (with the date noted) and a specimen picked (if legal to do so) and pressed, dried and labelled for future reference. If the host species can be identified accurately, its name should also be noted. If it cannot, a photograph showing as much as the plant as possible – ideally including the flowers – will need to be made.

Once the survey of Rusts and Smuts is underway, I suggest that the next group to be considered is the Powdery Mildews (Erysiphales), for which the Welsh Rust Group have just published their latest Guide: Chater, A.O. & Woods, R.G. (2019) *The Powdery Mildews (Erysiphales) of Wales: An Identification Guide and Census Catalogue*. A.O. Chater, Aberystwyth (www.aber.ac.uk/waxcap/downloads/).

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Rusts, Smuts and Powdery Mildews

See text.

Fig. 10. A holobiotroph, *Uncinula nectator* (Grape Powdery Mildew) on fruits of *Vitis vinifera*. Note the dense weft of spore-bearing surface hyphae on the otherwise intact fruits. Image by Rosemary Winnall

Fig. 11. Not all Smut spores are dark in colour. Here white, non-sexual 'bud spores' produced by budding hyphae of *Urocystis primulae* emerge from the infected anthers of Primrose *(Primula vulgaris)*. Image by Debbie White