

# Rapid Pest Risk Analysis (PRA) for

# Ceratocystis fagacearum

### Summary and conclusions of the Rapid PRA

The pest is the Ascomycete fungus *Ceratocystis fagacearum* that causes the disease 'oak wilt' and is currently only known to be present in the USA, although European oak species are susceptible and can be killed by the disease.

This Rapid PRA shows:

**Risk of entry** is <u>very unlikely</u> on plants for planting, logs/sawn timber or wood packaging material because of the measures that are already in place for *Quercus* imports from North America. However, uncertainties include whether hosts other than *Quercus* can be affected by *C. fagacearum* and whether the pathogen is only present in the USA or is elsewhere, so the probability of movement along various pathways cannot be fully assessed.

**Risk of establishment** under protection in the UK is also considered <u>very unlikely</u>. In contrast, establishment outdoors is considered <u>likely</u> but with medium confidence. The insect vectors species known to spread *C. fagacearum* in the USA are absent from the UK although there are vectors in the UK including the native oak bark beetle *Scolytus intricatus* that are considered suitable for transmission. Therefore, new associations with insect vectors would be required adding to the uncertainty when assessing the likelihood of establishment.

**Economic, environmental and social impacts** in the UK are rated as likely to be <u>large</u> but with medium confidence, as it is not clear how damaging *C. fagacearum* would be without knowing the full potential geographical range of the pathogen and the effectiveness of vectors.

**Endangered area** is assessed as <u>mainly England and Wales</u>, and based on climatic data northern England, Scotland, central-northern Wales and most of Northern Ireland could be outside the range of the pathogen. However, *Ceratocystis fagaceraum* has been found to affect oak trees in a wide range of environments (soil types and climatically) throughout the north-central, mid-western and mid-Atlantic states of the USA and susceptible hosts are widespread throughout the UK.

**Risk management options** of <u>exclusion measures</u>, through existing legislation, are considered the best option for reducing the risk of entry. If established, eradication is unlikely to be successful although control options, based on experience of managing oak wilt in the USA over many decades, could be deployed.

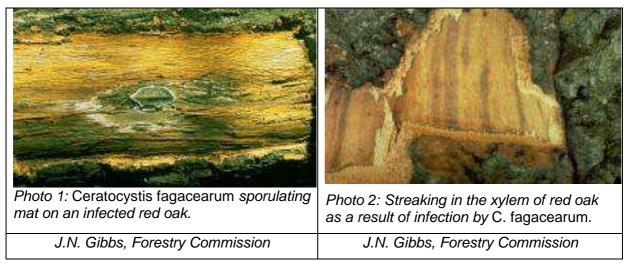
Risk of entry via living plants, logs, sawn wood and also wood packaging material (WPM) are already regulated. All WPM originating from outside the EU has to be ISPM15 compliant requiring debarking and heat treatment to achieve a minimum temperature of 56°C for 30 minutes throughout the wood. The EC Plant Health Directive also has strict requirements to mitigate the risk from any imports of plants, logs and wood of *Quercus* from outside the EU region. This includes removal of bark through peeling or squaring of sawn wood, drying to a moisture content of <20%, kiln drying or fumigation. Where bark removal cannot be achieved because of the end use of the logs then logs must be fumigated or heat treated. All these treatments have been shown to be effective at eliminating viable *C. fagacearum* from infected wood and would also kill any associated insect vectors. If, however, non-*Quercus* 

broadleaf species can act as hosts, most are not currently regulated in the same way as *Quercus* imports and could be at risk of entry.

<b></b>	
Hosts and pathways	<ul> <li>Development or testing of PCR based diagnostic for the detection of <i>Ceratocystis fagacearum</i>.</li> <li>Transparent import figures on quantity of oak imported into the UK from the USA (and other third party countries).</li> <li>Contacts with US scientists for more information about mat production on white oaks, specifically European oaks such as <i>Quercus roburl petraea</i>.</li> </ul>
Factors affecting spread and establishment	<ul> <li>More detailed work on growth temperature relationships for spore germination (conidia and ascospores) and mycelium of <i>C. fagacearum.</i></li> <li>Climate matching of zones where oak wilt is established in the USA with UK to provide fine-scale data on climate suitability.</li> <li>Improved understanding of the degree of root grafting, especially in relationship to oak density in woodland stands in the UK.</li> <li>Updated research on possible insect vector <i>S. intricatus.</i></li> <li>An evaluation of sap (pollen) beetle distribution in Europe, particularly species of <i>Carpophilus</i> which could be possible vectors of <i>C. fagacearum</i> in the UK.</li> </ul>

### Key uncertainties and topics that would benefit from further investigation include:

### Images of the pest



Is there a need for a detailed PRA or for more detailed analysis of particular sections of the PRA? If yes, select the PRA area (UK or EU) and the PRA scheme (UK or EPPO) to be used.

No 🗸

Yes	PRA area:	PRA scheme:	
	UK or EU	UK or EPPO	

# Given the information assembled within the time scale required, is statutory action considered appropriate / justified?

[The text below is a recommendation by the risk analyst which requires approval by PHRG]

Due to the longstanding recognition of the risk posed to trees in the UK and Europe from oak wilt a range of statutory actions are already in place (see section 8 below) to prevent entry of *C. fagacearum* and associated vectors from North America. However, the relatively recent proposals that *C. fagacearum* is more likely to be an introduction into the USA of unknown evolutionary and geographic origins, possibly from Mexico, Central America or northern South America, and associated with trees that extent to hosts other than *Quercus* merit discussion on whether wider statutory action could be considered appropriate.

Yes	No	
Statutory action	Statutory action	

### **STAGE 1: INITIATION**

#### 1. What is the name of the pest?

Teleomorph Ceratocystis fagacearum (Bretz) J Hunt Anamorph Chalara quercina Henry Thielaviopsis quercina Paulin, Harr., McNew Synonym Endoconidiophora fagacearum Bretz

The pathogen is invariably referred to by the teleomorph name Ceratocystis fagacearum.

#### Common name of the pest:

The pest does not have a common name, but the disease that it causes is referred to as 'oak wilt'.

<u>Taxonomic position</u>: Kingdom – Fungi; Phylum – Ascomycota; Class – Sordariomycetes; Order – Microascales; Family – Ceratocystidaceae; - Genus - *Ceratocystis* 

### 2. What initiated this rapid PRA?

The fungus *Ceratocystis fagacearum* was emphasised as a pest of concern to the UK during a review of tree health and plant bio-security action plans (LWEC, 2013). It is also included on the Defra Plant Health Risk Register https://secure.fera.defra.gov.uk/phiw/riskRegister/.

#### 3. What is the PRA area?

The PRA area is the United Kingdom of Great Britain and Northern Ireland.

### STAGE 2: RISK ASSESSMENT

# 4. What is the pest's status in the EC Plant Health Directive (Council Directive 2000/29/EC<sup>1</sup>) and in the lists of EPPO<sup>2</sup>?

*Ceratocystis fagacearum* is listed in the EC Plant Health Directive EU Annex designation as I/A1. It is also on the EPPO A1 list (ie absent from the EPPO region) and this list is reviewed every year.

### 5. What is the pest's current geographical distribution?

The pathogen was initially discovered in the early 1940s in the USA in the Upper Mississippi River Valley (Henry 1944; Henry et al., 1944) and over the next twenty years it was confirmed throughout the north-central, mid-western and mid-Atlantic states (Rexrode and Lincoln, 1965). Current known distribution is recorded in Table 1. It is not present in Canada although the disease occurs in twenty-three US states with some in close proximity to southern Ontario, Canada. The Canadian Food Inspection Agency regularly reviews phytosanitary measures that are in place to prevent the entry of oak wilt disease from the continental United States (CFIA, 2011).

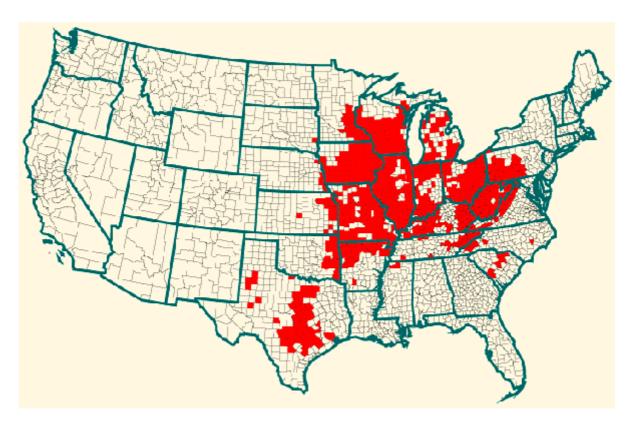
The literature also suggests that there have been three reports of oak wilt in Europe (Bulgaria, Poland and Romania) but analysis of these records by EPPO has concluded that the fungus isolated "*differs from* Ceratocystis fagacearum ... and all so-called European records are simple misidentifications of Ceratocystis-like fungi found in declining oaks." <u>https://gd.eppo.int/taxon/CERAFA/distribution</u> ).

<sup>&</sup>lt;sup>1</sup> http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CONSLEG:2000L0029:20100113:EN:PDF

<sup>&</sup>lt;sup>2</sup> https://www.eppo.int/QUARANTINE/quarantine.htm

Table 1: Distribution of Ceratocystis fagaceraum						
North America:	USA. Found throughout eastern and mid-west states. The pest has been recorded from Alabama, Arkansas, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Missouri, Nebraska, New York, North Carolina, Ohio, Oklahoma, Pennsylvania, South Carolina, South Dakota, Tennessee, Texas, Virginia, West Virginia, Wisconsin					
Central America:	No record					
South America:	No record					
Europe:	No confirmed record					
Africa:	No record					
Asia:	No record					
Oceania:	No record					

A detailed map showing the distribution of oak wilt compiled by the USDA Forest Service in 2005 is shown below (Figure 1).



**Figure 1:** Distribution map of oak wilt in the USA, courtesy of USDA Forest Service http://na.fs.fed.us/fhp/ow/maps/ow\_dist\_fs.shtm

# 6. Is the pest established or transient, or suspected to be established/transient in the UK/PRA Area?

There have been no interceptions and there are no records or other evidence to suggest that *Ceratocystis fagaceraum* has ever been established or transient in the UK. Records of the pest elsewhere in Europe (Bulgaria, Poland, Romania) are now considered spurious (see 5 and https://gd.eppo.int/taxon/CERAFA/distribution).

# 7. What are the pest's natural and experimental host plants; of these, which are of economic and/or environmental importance in the UK?

*Ceratocystis fagacearum* causes a vascular wilt disease which has resulted in the mortality of many thousands of native oak species in the north-central United States (Juzwik et al., 2008). Symptoms vary with oak species, but are caused when *C. fagaceraum* gets into the functional xylem of infected trees. This incites the development of tyloses and gums in affected vascular tissues which subsequently restrict the flow of water and nutrients with the result that the tree wilts and often dies. Further details of symptoms are available on the EPPO<sup>3</sup> website.

http://www.eppo.int/QUARANTINE/fungi/Ceratocystis\_fagacearum/CERAFA\_images.htm?ut m\_source=www.eppo.org&utm\_medium=int\_redirect

Stein et al. (2003) record 50 native *Quercus* species in eastern North America and the Mast Tree Network estimates there are more than 58 native species in total (<u>http://www.mast-producing-trees.org</u>). Oak wilt is reported to affect more than 30 of these species under field conditions (Juzwik, 2000) and Appel (1995) comments that probably all native oaks found in the USA are susceptible to some extent when inoculated with *C. fagacearum*.

Table 2 provides details of oak species that have been recorded as natural hosts of oak wilt in the USA. Red oak species (sub-genus *Erythrobalanus*) are highly susceptible to *C. fagacearum*, and infected trees typically die within 3 months of first symptom expression (Juzwik, 2000). Native American white oaks (sub-genus *Lepidabalanus*) are moderately to highly resistant to *C. fagacearum* and may take several years to die whilst displaying decline symptoms and some trees may even recover from the infection (French and Stienstra,1980). Live oaks (evergreen oak species within *Lepidabalanus*) are considered intermediate between red and white oaks in their response to infection by *C. fagacearum* (Appel et al., 1987).

Trees in other genera of the Fagaceae have been found to be susceptible. For example, American and European chestnuts (*Castanea dentata* and *C. sativa* respectively), species of chinquapin (*Castanopsis sempervirens*) and tanoak (*Lithocarpus*) can all be affected by *C. fagacearum* following artificial inoculation, as have several varieties of apple (*Malus*) (Sinclair and Lyon, 2005; Rexrode and Brown, 1983). Other *Castanea* species including Asian chestnut (*Castanea mollissima*) and bush chinquapin (*C. pumila*) have been found to be natural hosts with reports indicating that affected trees can die within a few weeks of infection (Bretz and Long, 1950). However, Merrill (1975) did not find any evidence that indicated that American chestnut could be a reservoir of the oak wilt fungus in a way that supported disease transfer to the more susceptible oak species.

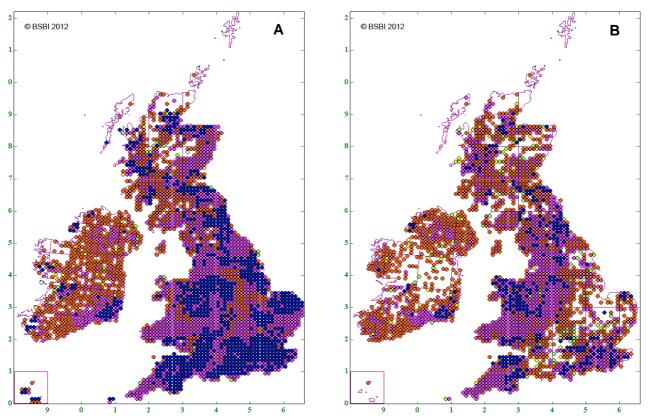
White oak species (sub-genus *Lepidabalanus*) that are native to Europe can also be infected and some have been found to be as susceptible to oak wilt as North American red oaks (Pinon et al., 1987; MacDonald et al., 2001). European oaks *Q. robur* (pedunculate oak), *Q. pubescens* (downy oak) and *Q. petraea* (sessile oak) were tested for susceptibility in an arboreta experiment in South Carolina (Clemson) and West Virginia (Morgantown). In 1996, 12 to 14 year-old trees of these European oak species were inoculated with *C. fagacearum* and disease development compared with native susceptible and resistant controls then assessed in 1996 and subsequent years. The European oaks developed extensive wilt symptoms very similar to those of susceptible red oaks (*Q. rubra*), and most were dead or dying within a year of inoculation (70-100% wilt and dieback). *Quercus petraea* had lower levels of disease in the South Carolina arboretum, but not at the other site, suggesting climatic or site-related influences on disease development.

<sup>&</sup>lt;sup>3</sup> EPPO is the European and Mediterranean Plant Protection Organization

Table 2: List of Quercus species naturally infected by Ceratocystis fagacearum in the USA*								
Red oaks = sub-genus Erythrobalanus; White oaks = sub-genus Lepidabalanus								
Species	Common name	Species	Common name					
Quercus alba (W)	White oak	Q. nigra (R)	Water oak					
Q.buckleyi (R)	Spanish oak	Q. palustris (R)	Pin oak					
Q. coccinea (R)	Scarlet oak	Q. polymorpha (W)	Mexican white oak					
Q. ellipsoidalis (R)	Northern pin oak	Q. phellos (R)	Willow oak					
Q. falcata (R)	Southern red oak	Q. prinus (W)	Chestnut oak					
Q. fusiformis (W)§	Live oak	Q. rubra (R)	Northern red oak					
Q. ilicifolia (R)	Scrub oak	Q. shumardii (R)	Shumard oak					
Q. imbricaria (R)	Laurel oak	Q. sinuata (W)	Durand oak					
Q. laceyi (W)	Lacey oak	Q. stellata (W)	White post oak					
Q. macrocarpa (W)	Burr oak	Q. texana (R)	Texas red oak					
Q. marilandica (R)	Blackjack oak	Q. velutina (R)	Black oak					
Q. muehlenbergii (W)	Chinkapin oak	Q. virginiana (W)§	Southern live oak					

\* Sources from EPPO database (<u>https://gd.eppo.int/taxon/CERAFA/hosts</u>), CFIA (2012) § Evergreen or 'live oak' species of white oak

Two of the native European oak species, *Q. robur* and *Q. petraea*, that showed high susceptibility to *C. fagacearum* have major economic and environmental importance in the UK. They make up ~9% of total tree cover in woodlands over 2 ha in Britain and are also widespread and common outside woodlands (see Figure 2 and Forestry Commission, 2013). Other susceptible species of oak are present in the UK (eg *Q. coccinea*, *Q. palustris*, *Q. rubra*) but these are mainly grown as ornamentals and so numbers are small and distribution is sparse.



**Figure 2:** Distribution of native oak species based on BSBI maps http://www.bsbimaps.org.uk/atlas/main.php (A), hectad map of *Quercus robur* (pedunculate oak) in Great Britain and Ireland (records 1987-2010); (B), hectad map of *Quercus petraea* (sessile oak) in Great Britain and Ireland (records 1970-2010)

# 8. What pathways provide opportunities for the pest to enter and transfer to a suitable host and what is the likelihood of entering the UK/PRA area?

For most of the history of oak wilt, *C. fagacearum* has been considered a longstanding, native North America pathogen, endemic at low incidence until changes in land use and forest management created favourable conditions for an increased disease incidence. However, more recently evidence has accumulated to suggest that it may be an introduced organism in the USA. Indicators of an exotic pest include (1) the many native species of *Quercus* that are highly susceptible across the disease range and (2) evidence of a genetic bottleneck expressed as limited variation in the fungus, despite the frequent occurrence of sexual reproduction and out-crossing behaviour (Harrington, 2009; Kurdyla et al., 1995).

Juzwik et al. (2008) therefore proposed that *C. fagacearum* is more likely to be an introduction into the USA of unknown evolutionary and geographic origins. They speculate that it originates from Mexico, Central America or northern South America, possibly as an ambrosia beetle symbiont associated with trees of the Fagaceae within this region. If this is so, then pathways for entry could extend beyond the USA and to hosts other than *Quercus*.

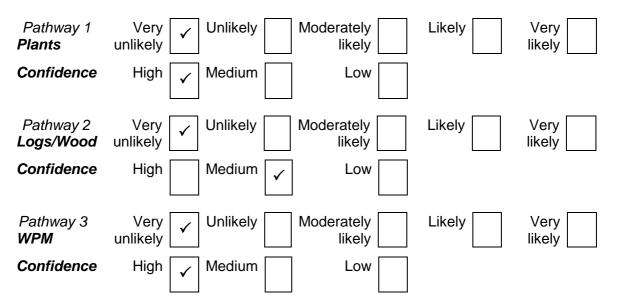
In his analysis of the risk that oak wilt could pose to Europe and the UK, Gibbs (1984) concluded that the risk of the pest reaching Europe from North America was *"very small, even without the present stringent regulations*" although he cautioned that if it did arrive it was *"unlikely to be of minor importance*" (see also Gibbs, 1979).

Pathways for entry that have been identified in previous analysis of risk include living plants, logs, sawn wood and, more latterly, wood packaging material (WPM) from the USA, especially if logs or wood contain associated bark beetle vectors. There is no evidence that seeds or foliage of infected hosts serve as a means of movement for *Ceratocystis fagacearum*, either locally or over long distances (Bretz and Buchanan, 1957; Gibbs, 1984).

It is possible that *C. fagacearum* could enter the UK in the xylem of living plants. However, even in the areas worst affected by oak wilt in the USA, there are no records of small trees in nurseries affected by *C. fagacearum* (Gibbs, 1984; Juzwik et al., 2008) and any plants must originate from an area known to be free of *C. fagacearum*, so an entry pathway from the USA via plants for planting appears unlikely. The EU (EC Plant Health Directive, Annex III: Anon. 2000) also prohibits the import of *Quercus* plants with leaves (other than fruit and seeds) from non-European countries. Overall therefore, this pathway is rated as very unlikely with high confidence.

Logs and sawn timber potentially comprise a higher risk pathway, especially as there is significant trade in oak timber between North America and Europe, although the majority of oak (round and sawn) imported into the UK comes from other EU countries (Moore, 2012). Red oak logs are considered a much more likely pathway for entry of oak wilt than white oak logs (Miller et al., 1985). This is because red oaks are more susceptible to oak wilt, and the bark of infected red oaks can harbour sporulating mats of C. fagacearum as well as the sap beetles (Coleoptera: Nitidulidae) which vector the pathogen (Juzwik et al., 2008; see also paragraph 10). In contrast, American white oak species are less susceptible and rarely form sporulating mats when infected (Cones, 1967). There are strict requirements to mitigate the risk from wood and make entry via this pathway unlikely, including removal of bark through peeling or squaring of sawn wood, drying to a moisture content of <20%, kiln drying or fumigation. Where bark removal cannot be achieved because of the end use of the logs (eg veneering) then logs must be fumigated or heat treated prior to export from the USA. All these treatments have been shown to be effective at eliminating viable C. fagacearum from infected wood (eg Gibbs and French, 1980; Schmidt et al., 1987) and would also kill any associated insect vectors. On this basis, providing the risk is mitigated, entry via this pathway is considered very unlikely, with the caveat that trade pathways for logs and timber may also exist from geographical areas other than North America and include non-Quercus species that are not currently regulated in the same way as Quercus imports so confidence in the rating is assessed as medium.

Likewise WPM derived from oak could also provide a pathway of entry if not compliant with ISPM15<sup>4</sup>. Scolytinae are commonly intercepted on non-compliant WPM (Marini et al. 2011) and *C. fagacearum* could also survive for a period in such material. All WPM originating from outside of the EU should be ISPM15 compliant, which requires debarking and heat treatment to achieve a minimum temperature of 56°C for 30 minutes throughout the entire profile of the wood. <u>Providing</u> this mitigation is in place, entry on WPM is again rated as <u>very unlikely</u> with a high level of confidence as all potential hosts including *Quercus* would be treated.



## 9. If the pest needs a vector, is it present in the UK/PRA area?

For dispersal over longer distances the pathogen is dependent upon the feeding or tunnelling activity of various insect vectors. In the USA these comprise sap beetles of the family Nitidulidae (Jewell, 1956; French and Stienstra, 1980) and oak bark beetles of the genus *Pseudopityophthorus* (EPPO, 2011).

At least 12 species of sap beetles have been associated with oak wilt mats in some states in the USA and are considered the primary vectors in north and central states (Cease and Juzwik, 2001; Juzwik, 2001). However, the main nitidulid species transmitting the pathogen from diseased to healthy oaks are Colopterus truncatus and Carpophilus sayi (Juzwik et al., 2004). These sap beetles are commonly attracted to the sporulating mats that are produced by C. fagacearum between the bark and wood of oak wilt-killed trees. The formation of the mats lifts and cracks open the overlying bark making them accessible to the nititulids and the fruity odour of aromatic volatiles given off by the mats also actively attracts these insects to feed (Lin and Phalen, 1992). In the USA, the mats are usually produced between April and late June on oaks (predominantly red oaks) infected during the previous summer (Gibbs and French, 1980). As the nitidulids feed on the sporulating mats, the sticky conidia and ascospores adhere to the insects and then are carried to healthy trees and deposited in wounds when the insects alight (Davies, 1992). If the pathogen is to be successfully transferred to healthy trees, there must be fresh wounds with exposed xylem which are receptive to infection. Such wounds are usually produced by human activity or weather related injuries, but wounds older than three days are not susceptible to infection (Gibbs and French, 1980). Visitation of fresh wounds by C. fagacearum-contaminated nitidulids to feed on oozing sap then results in oak wilt infection although transfer efficiencies are likely to be variable, depending on contamination levels of the vectors, tree species and wound age (Hayslett et al., 2009. Dorsey and Leach (1956) found that mean weekly temperatures of 10°C were optimal for nitidulid activity.

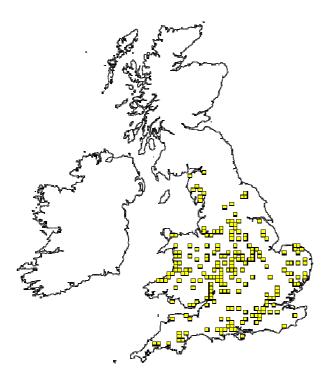
<sup>&</sup>lt;sup>4</sup> International Standards for Phytosanitary Measures ISPM 15 http://www.ispm15.com/ISPM15\_Revised\_2009.pdf

Although nitidulid beetles are necessary for overland transfer of C. fagacearum in some parts of the USA, oak bark beetles can also act as vectors where sporulating mats are rarely produced by infected oaks (Gibbs et al., 1984; Hayslett et al., 2008; 2009). For example, oak bark beetles, Pseudopityophthorus minutissimus and P. pruinosus, are considered significant vectors of the oak wilt fungus in Missouri and Ohio whereas in Minnesota the extent of their role in the spread of C. fagacearum is unclear (Ambourne et al., 2005). These bark beetles make gallery systems in diseased oaks, almost exclusively utilising small diameter branches (up to 10 cm diameter). This can bring them into contact with the pathogen in the bark and sapwood such that when the new generation of beetles emerges. as many as 30% can carry C. fagacearum on their bodies and transfer it to the wounds that they make as they feed on healthy trees (EPPO, 2011). Commonly, feeding wounds are in the twig crotches, leaf axils, bud axils and immature acorn axils of both red and white oaks. Fresh feeding wounds can be found from early spring onwards and they can act as infection courts when inoculated with the oak wilt fungus. The percentage of young adult beetles carrying the fungus is very variable; typically it ranges between 0.4 and 2.5% because of the poor survival of C. fagacearum in small diameter branches where the beetles often breed (Ambourne et al., 2005).

Another insect species, the two-lined chestnut borer – *Agrilus bilineatus*, and other animals such as birds and squirrels are considered potential carriers of the oak wilt fungus, but their role has not been established in the USA (Himelick et al., 1953; Gibbs and French, 1980; Juzwik et al., 2008). For similar vectors in the UK their potential would also be dependent on the formation of sporulating mats on infected trees and these are much more frequent on red oaks than white oaks.

Neither nitidulid beetles of the genus *Colopterus* or scolytid beetles of the genus *Pseudopityophthorus* are represented in the UK. Beetles of the genus *Carpophilus* (known as pollen beetles) are present in the UK and represented by 11 species (Duff, 2012) so potentially vectors of the pathogen are present. However, more importantly nothing is known about the potential for infected European oaks to produce sporulating mats of *C. fagacearum* after infection (Gibbs, 1978); with American white oaks (also in the sub-genus *Lepidabalanus*) mats are rarely produced (Engelhard, 1955; Davies, 1992). These sporulating mats are essential for disease spread by nitidulid beetles as they are attracted to the mats to feed and breed, thereby picking up spores of the pathogen and subsequently transferring them to healthy trees (Appel et al., 1986; Davies, 1992).

It has been suggested that the UK native oak bark beetle *Scolytus intricatus* has many of the attributes that would make it an effective vector of *C. fagacearum* in the UK (Gibbs et al., 1984) and it could operate in much the same way that *Scolytus* elm bark beetles vector the Dutch elm disease fungus (Webber and Brasier, 1984). Indeed, *Scolytus intricatus* has the potential to be a more effective vector than *Pseudopityophthorus* spp because its more aggressive behaviour allows it to rapidly invade the bark of compromised oak trees, and as it favours thicker branches (10-40 cm diameter) it would be more likely to breed in parts of the tree where *C. fagacearum* is likely to be present in the xylem and able to remain viable for longer (Gibbs et al., 1984; Yates, 1981). *Scolytus intricatus* also feeds on twigs, particularly in the crotches between the preceding and current seasons' growth, and in leaf axils and bud axils on current growth, in much the same way as *Pseudopityophthorus*, which would aid entry and subsequent infection by *C. fagacearum* (Yates, 1981). Records of the distribution of *S. intricatus* suggest that it is not common but relatively widespread in England and Wales, although there are no records of the insect in Scotland or Northern Ireland (see Figure 3).



**Figure 3:** Distribution of *Scolytus intricatus* (oak bark beetle) in the UK, based on findings at a resolution of 10 x 10 km squares provided courtesy of the National Biodiversity Network database

https://data.nbn.org.uk/Taxa/NBNSYS0 000025525/Grid\_Map

**10.** How likely is the pest to establish outdoors or under protection in the UK/PRA area? (The likelihood rating should be based on the area of potential establishment, e.g. where hosts are present and the climate is suitable, within the UK/PRA area)

**Under protection:** Establishment under protection in the UK is very unlikely because *C. fagacearum* does not affect crops grown under protection.

**Outdoors:** In the wider environment, temperature and moisture are known to have some influence on the extent of the distribution and spread of C. fagacearum in the USA. The optimum temperature for growth of the pathogen in culture is reported as 22-26°C (Lewis, 1985) and 24-28°C (Henry, 1944), with spore germination at 25°C (Bretz, 1952). Temperatures greater than 32°C inhibit the growth of *C. fagacearum* both in affected trees as well as in culture (Houston et al., 1965) and it can only survive for a few hours when tested in culture at temperatures above 37 °C (Lewis, 1985). This temperature limit is thought likely to have prevented disease spread into the warmer southern states, with reduced moisture in addition to heat, limiting the development of disease and survival of the pathogen in trees in states such as South Carolina (Appel, 1995; Tainter and Ham, 1983). However, oak wilt also has a serious impact in central Texas where summer temperatures exceed 32°C. The pathogen has been shown to survive in dry wood of oaks in Texas and apparently is able to withstand temperatures of  $> 32^{\circ}$ C inside tree trunks and root systems (Appel, 1995). Temperatures suitable for growth and survival of C. fagacearum are maintained in the root collar and lower bole of infected during the hottest periods of summer in Texas (Lewis, 1985).

When an infected oak dies, *C. fagacearum* develops sporulating mats in the bark which produce conidia and ascospores when mature (French and Stienstra, 1980). The mats are produced frequently on red oaks but are rarely seen on live oaks or white oaks (Engelhard, 1955; Davies, 1992). They are important for disease spread as they attract insects and these then feed and even breed in the mats, thereby picking up spores of the pathogen and subsequently transferring them to healthy trees (Appel et al., 1986; Davies, 1992). These mats are not produced under dry climatic conditions (Sinclair and Lyon, 2005) and this is also believed to have suppressed the southerly spread of the disease in the USA (Appel, 1995: White, 2001).

### Host and suitable climate in the UK:

Suitable host tree species (*Q. robur* and *Q. petraea*), already shown to be highly susceptible to oak wilt in the USA (Pinon et al., 1987; MacDonald et al., 2001), grow throughout much of

the UK (Figures 1a, b). With an optimum temperature for growth of 22-26°C, a minimum of 7-8°C, poor growth at 16°C and a maximum of 32°C (Barnett and True1955; Houston and Kuntz, 1960), the pathogen is likely to be able to establish in the UK providing an insect vector is available to aid spread over longer distances. Climatic conditions in the south of Britain may be the most favourable for the pathogen, but Gibbs and French (1980) also note that high summer temperatures can significantly reduce the survival of the pathogen in branches and smaller diameter sections of infected oaks. A comparison of mean monthly weather data for St Paul - Minnesota, Dallas – Texas, and London – England are shown below in Table 3. There are marked differences in temperature ranges between the three regions (and even within region), but temperatures in England during summer appear likely to sustain growth of the pathogen, including the production of sporulating fungus mats which normally form in spring in Texas and Minnesota.

**Table 3:** Comparisons of mean monthly min and max temperatures and precipitation for Minnesota\*, Texas\* and England

Location	Jan	Feb	Mar	Apr	May	June	July	Aug	Sep	Oct	Nov	Dec
Texas High °C	16.4	18.4	22.3	26.6	30.3	33.4	35.3	36.1	32.5	27.7	21.9	17.1
Texas Low <sup>o</sup> C	5.3	7.1	10.7	14.8	19.3	22.4	23.6	23.7	20.8	15.9	10.3	5.7
Texas rain (mm)	56	51	70	53	11	110	48	60	76	99	75	61
Minnesota High <sup>o</sup> C	-3.4	-0.6	6.0	14.7	21.7	26.7	29.2	27.8	22.7	15.2	5.7	-1.8
Minnesota Low <sup>o</sup> C	-13.8	-11.3	-4.3	3.1	10.0	15.1	17.8	16.6	11.6	4.9	-2.7	-10.8
Minnesota rain	20	17	39	73	94	107	112	121	83	74	46	28
England High <sup>o</sup> C	6	7	10	13	17	20	22	21	19	14	10	7
England Low <sup>o</sup> C	2	2	3	5	8	11	13	13	11	8	5	3
England rain (mm)	53	40	37	38	46	56	56	59	50	57	64	48

\* See http://www.usclimatedata.com/climate/united-states/us

Assuming that pathogen establishment will be unaffected by winter temperatures in the UK since it survives in much colder winters in the USA, the extent to which temperatures exceed 7°C (minimum temperature for growth) at the northern limits of its distribution in the USA can be compared with the UK. Mapping the number of annual degree days above 7°C for the USA and the Europe using figures available between 1991-60 indicates that the annual degree days above 7°C at the northern edge of the oak wilt range in Michigan are between 1,000 and 1,500 and the same range occurs in some of Britain but not northern England, Scotland, central-northern Wales and most of Northern Ireland (Figure 3a,b; R. Baker, unpublished). This suggests that the climate would be suitable for the pathogen to establish in the UK.

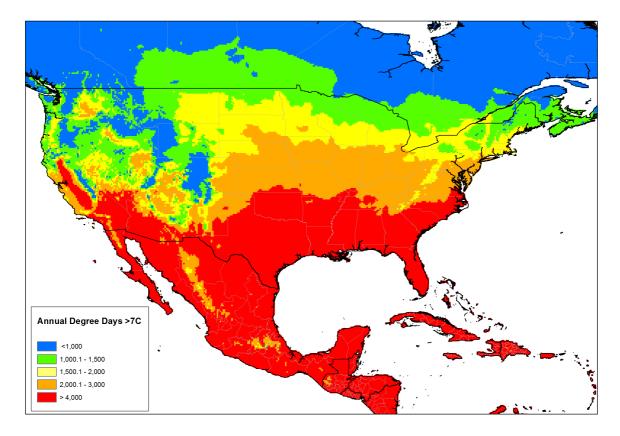
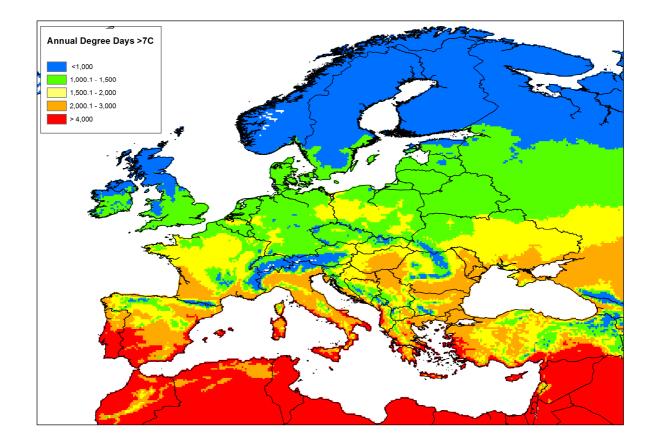
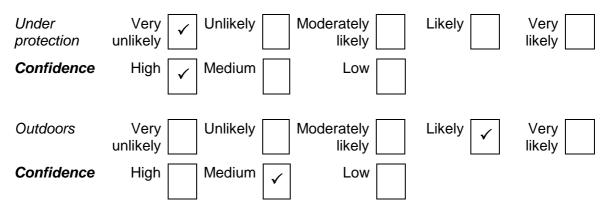


Figure 3a: Map showing the number of annual degree days above 7°C for the USA.



**Figure 3b:** Map showing the number of annual degree days above 7°C for Europe including the UK.

On this basis, the likelihood of establishment in at least some parts of the UK is rated as <u>likely</u> with medium confidence. However, many of the insect vectors known to spread the pathogen in the USA are absent from the UK, so that new associations with other insect vectors (see paragraph 9) would be required, adding to uncertainty when assessing the likelihood of establishment.



### 11. How quickly could the pest spread in the UK/PRA area?

Local spread of *C. fagacearum* in the USA occurs from tree to tree through root connections or root grafts, resulting in expanding infection centres. This mode of spread is thought to be responsible for a far greater proportion of disease spread (up to 90% in some areas) compared with spread by insect vectors (Gibbs and French, 1980; Cooke, 2001). However, transfer through roots may only be typical of certain soils and habitats, where large, homogeneous, highly interconnected stands of oak exist, allowing rapid spread sometimes over considerable distances (Appel, 2001).

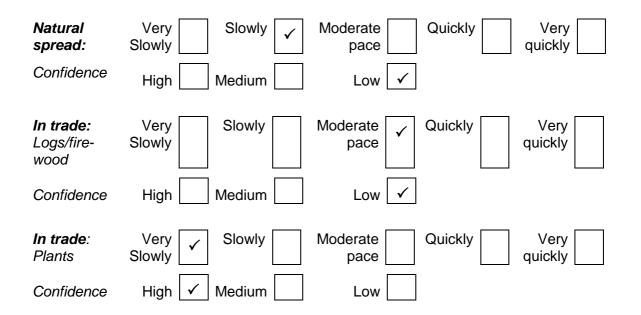
Above ground and over longer distances, the speed of spread is dependent on the availability and effectiveness of suitable insect vectors. It will also depend on whether the fungus mats which aid spore dispersal with certain vectors are capable of forming on *Q. robur* and *Q. petraea*, and can be produced under typical UK weather conditions. In the USA the fungus mats are produced on red oaks that are infected in late summer, and mats then form on these trees usually in spring the following year but occasionally in the autumn (Appel et al., 1987, Wilson, 2005). Survival and attractiveness of the mats to casual insect vectors may also be short-lived, typically they are viable for only 2-3 weeks and deteriorate rapidly with the onset of high summer temperatures in the USA (Sinclair and Lyon, 2005). Indeed, fungal mats are not produced at all in dry conditions and this has probably suppressed the spread of oak wilt in the southwest towards Texas (Appel, 1995). Reduced moisture, in addition to heat, is also thought to limit the development of disease and survival of the pathogen in trees in the south eastern state of South Carolina (Tainter and Ham, 1983), so much so that rapid drying by girdling at the base of the trees has been employed as a control method to limit transmission (True and Gillespie, 1961; Gillespie and True, 1963).

Under UK conditions, the behaviour of *S. intricatus* could be suitable for the transmission of *C. fagacearum*. It has also been suggested that the wounds produced by *S. intricatus* during maturation feeding in the twig-crotches of oak would favour transfer of *C. fagacearum* into the vascular system of healthy oaks in much the same way that occurs with *Pseudopityophthorus* in the USA (Yates, 1981). However, the seasonality of maturation feeding could be an important factor influencing the transmission of oak wilt. The susceptibility of North American oaks to *C. fagacearum* is greatest during springwood formation in spring and early summer. In contrast, feeding by *S. intricatus* reaches a peak in Britain during late-wood formation which is later in the year. Therefore, disease spread could be impeded because the phases of maximal insect feeding and optimal tree susceptibility may not coincide closely.

Transportation of firewood from diseased red oaks has been shown to result in further distribution of the fungus since fungal mats in the bark can attract nitidulid beetles which subsequently carry fungal spores to new locations (Davies, 1992; Wilson, 2001). Diseased red oak firewood is believed to have been the source of the oak wilt infection in three west Texas counties, removed from the main infected area in Texas by 150 or more miles (Davies, 1992). *Ceratocystis fagacearum* can be isolated from sawn lumber up to 24 weeks after sawing (Gibbs and French, 1980).

In relation to natural spread, transfer through root grafts or common root systems is well documented (eg Gibbs and French, 1980; Cooke, 2001), but typically only results in expansion of localised disease foci. Whitford et al. (2007) suggest that in Wisconsin oak savanna when the average distance between mature oak trees is less than 30 feet (approx 9 m) this assures continuous root to root transmission of the disease. In the UK, transfer through root grafts will therefore be dependent on the interconnectedness of oak root systems and the density of oak in woodlands. Spread via possible native vectors such as *S. intricatus* could support spread to new areas, and studies on the dispersal of this insect suggest it can fly distances of at least 0.35 km after emergence from breeding material (Gibbs et al., 1984). However, rate of spread by *S. intricatus* would also be influenced by climate and population levels of this native bark beetle. On this basis, natural spread is rated as likely to occur <u>slowly</u>, but with a low level of confidence in the rating because of the lack of data to support the evaluation about insect vectors or the extent of root grafts.

The absence of any records of nursery trees affected by *C. fagacearum* (see paragraph 8) even in the areas worst affected by oak wilt in the USA, suggests that spread in the plant trade is unlikely and if it occurs at all it is likely to be <u>very slow</u> (high confidence). With logs, particularly those used for fuel (ie firewood), if both pathogen and vector scolytid beetles are present then spread could occur and is assessed as likely to occur at <u>moderate pace</u> because of the potential to move over long distances. However, there is a low level of confidence associated with the assessment because of the lack of data to suggest how effective European native scolytids would be as vectors, or whether mycelial mats would be produced on infected European oaks. Where firewood has acted as a pathway for disease spread in the USA, this is associated with the formation of fungus mats which has allowed dispersal via nitidulid beetles; only *Carpophilus* beetles are present in the UK and not the full range of nitidulid beetles that vector the pathogen in the USA although other casual insect vectors might take on this role.



# 12. What is the pest's economic, environmental and social impact within its existing distribution?

In the USA, *C. fagacearum* kills thousands of oak trees annually within the known disease range (Juzwik et al., 2008). However, severity of oak wilt varies considerably and in many parts of the infested areas of the United States there is minimal impact on local forest-based economies.

Although oak wilt is widely distributed in Midwestern, Middle and South Atlantic states (Figure 1), disease severity when measured by (i) numbers of oaks killed per hectare, (ii) area affected by oak wilt or (iii) number of disease foci per hectare, can differ greatly between the regions and within states (Juzwik, 2009). In 2007 Juzwik (2009) commented that although the disease was found in 52 out of 55 counties in West Virginia, the incidence was sporadic and severity low, whereas in Minnesota oak wilt was present in far fewer counties (25 out of 65) but disease severity was very high in many locations. Between 1991-2001, oak wilt was estimated to affect 15,359 acres in Minnesota with a projected two-fold increase in infection rates expected by 2008 (MNDNR, 2000). In Texas, oak wilt was estimated to affect a minimum of 6,500 acres in 2007 (Texas Forest Service, 2007).

In urban areas where susceptible oaks are abundant, the impact on property or other social values has also been significant. In central Texas, for instance, oak wilt has caused considerable decline in urban and rural property values through landscape degradation, shade loss and a resulting decline in property values. In 1990, the Texas Forest Service estimated that over 10,000 trees worth millions of dollars were lost in the City of Austin alone (Davies, 1992). However, an economic analysis of over 20 years of work by the Texas Cooperative Oak Wilt Suppression Project has documented a spend of \$9.2 million of federal, state, city, and private funds but this expenditure has yielded an average benefit:cost ratio of 6:1 and saved Texas communities an estimated \$55 million in tree removal, replanting, and fungicide costs (Billings, 2008).

Based on this information the overall impact of oak wilt is assessed as large in its existing distribution in the USA (and very large in some locals), although detailed quantitative data on the economic, environmental and social impacts caused by this pathogen have been difficult to find but there are some examples. Haight et al (2011) estimated that in Anoka County, Minnesota (an area with 5.92 million oak trees and 885 active oak wilt pockets covering 5.47 km2 in 2007) between 2007-2016 some 76,000–266,000 trees would be infected, with discounted removal costs of \$18–60 million depending on the rates of oak wilt pocket establishment and expansion. The authors also noted that these removal costs did not take into account other impacts such as loss of landscape vale and increased hazards.

Whilst acknowledging the very variable intensity of the impacts over the oak wilt disease range, if the time span of the epidemic in the USA (first described in the 1940s), the geographical extent and the millions of trees affected or killed by the pathogen are taken into account, the impacts are assessed as <u>very large</u> with a high level of confidence in the rating.

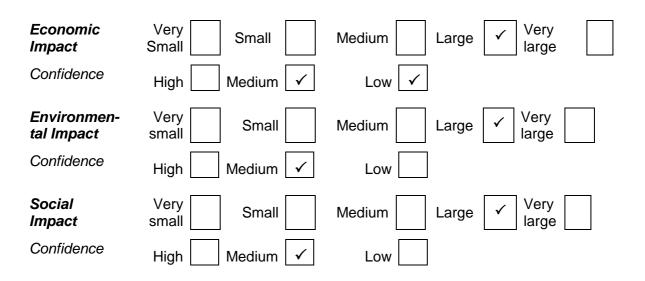


# 13. What is the pest's potential to cause economic, environmental and social impacts in the UK/PRA area?

Valuation methods applied to oak, estimate that as a proportion of total woodlands in Great Britain oak makes up around 7.5% (ie 230,000 ha as a proportion of a total of 3 million ha) and therefore the social and environmental value of oak is estimated at around £135 million per year. Combined with the commercial value of oak, estimated at just under £40m per year, the total yearly value is calculated at around £175 million per year (Defra, 2014). Oak woods are potentially some of the most diverse in Britain and oak trees in particular support an enormous variety of invertebrate life (423 species) (Southwood, 1996). Oaks are also an extremely long-lived species, with some recorded specimens being as much as 1000 years old. Such ancient trees have enormous biodiversity value because of the large number of microhabitats which they provide for species such as lichens (up to 324 species), as well as a great variety of invertebrates, birds and mammals (Alexander et al., 2006).

Therefore, oak is of major significance in the landscape in Britain, highly prized as a timber tree with large environmental and social values. Current evidence also suggests that the climate will be suitable for *C. fagacearum* in at least some parts of the UK.

On this basis, *C. fagacearum* has the potential to have <u>large economic, environmental and</u> <u>social impacts</u>, and these effects are likely to be amplified because of other recent threats to oak. Operating in conjunction with the occurrence of defoliating insects including the more recently introduced oak processionary moth (*Thaumetopoea processionea*) (Defra, 2014), and Acute Oak Decline (Denman et al., 2014) the cumulative effects of *C. fagacearum* are likely to be very damaging to oak. However, without knowing the full potential geographical range of the pathogen and potential vectors and as data on impacts are largely qualitative, these ratings have medium confidence.



### 14. What is the pest's potential as a vector of plant pathogens?

*Ceratocystis fagacearum* is a plant pathogen with no capacity to act as a vector of other pathogens.

### 15. What is the area endangered by the pest?

*Ceratocystis fagacearum* could potentially become established throughout much of the range of its known hosts in Britain (principally *Quercus robur* and *Q. petraea*) which are found in woodlands, hedgerows and roadsides, parklands and gardens (National Forest Inventory <u>http://www.forestry.gov.uk/forestry/infd-8eyjwf</u> and Figure 2). Mapping the number of annual degree days above 7°C (minimum temperature for growth of the pest) for the USA and the Europe using figures available between 1991-60 (see section 10) suggests that some of Britain but not northern England, Scotland, central-northern Wales and most of Northern Ireland would be most endangered.

### **STAGE 3: PEST RISK MANAGEMENT**

## 16. What are the risk management options for the UK/PRA area?

#### Exclusion

The risk of entry via living plants, logs, sawn wood and also wood packaging material (WPM) are already under management.

All WPM originating from outside the EU has to be ISPM15 compliant. This requires debarking and heat treatment to achieve a minimum temperature of 56°C for 30 minutes throughout the entire profile of the wood.

In addition, the EC Plant Health Directive, Annex III demands strict requirements to mitigate the risk from any imports of plants, logs and wood of *Quercus* from outside the EU region. The requirements include removal of bark through peeling or squaring of sawn wood, drying to a moisture content of <20%, kiln drying or fumigation. Where bark removal cannot be achieved because of the end use of the logs (eg veneering) then logs must be fumigated or heat treated prior to export. All these treatments have been shown to be effective at eliminating viable *C. fagacearum* from infected wood and would also kill any associated insect vectors.

If, however, non-*Quercus* broadleaf species can act as hosts, most are not currently regulated in the same way as *Quercus* imports. To be epidemiologically significant hosts they would have to be a reservoir of the oak wilt fungus in a way that supported disease transfer to the more susceptible oak species.

#### Eradication, containment and controls

Exclusion, through legislation, is the best risk management option. The nature of the pathogen and host brings the possibility that oak wilt might become established but symptoms, at least initially, could be mis-attributed to other disorders already present in the UK such as chronic or acute oak decline (Denman et al., 2014). This could make early detection difficult or impossible and on this basis, eradication of *C. fagacearum* if it establishes is unlikely to be successful.

In the USA, a wide range of control and management practices are used against oak wilt. For prevention and eradication these include:

- public education on proper timing of pruning and treating wounds on oak trees to prevent fresh infections via nitidulid beetles,
- elimination of potential fungal inoculum by destroying diseased red oaks, proper handling of firewood and planting diverse resistant tree species.

For containment and direct control, procedures include:

- detection, field evaluation and survey of expanding oak wilt centres,
- installation of trenches, at least 1.2 m (4 feet) deep, to prevent continual tree-to-tree spread of the fungus through interconnected live oak root systems,
- root-flare injections with the fungicide propiconazole to prevent the development of severe disease symptoms (repeated every 2 years) in affected trees.

Further details and an evaluation of disease management methods effective against *Ceratocystis fagacearum* are described in Billings and Appel (2008), Koch et al. (2010) and Cook (2012).

### 17. References

Alexander K, Butler J, Green T. 2006. The value of different tree and shrub species to wildlife. British Wildlife October 2006, 18-26.

Ambourn AK, Juzwik J, Moon RD, 2005. Seasonal dispersal of the oak wilt fungus by *Colopterus truncatus* and Carpophilus sayi in Minnesota. Plant Disease 89,1067-1076.

Appel DN, Anderson, Lewis R. 1986. Occurrence of nitidulid beetles (Coleoptera: Nitidulae) in Texas oak wilt centres. Journal of Economic Entomology 79,1276-1279.

Appel DN, Peters R, Lewis R. 1987. Tree susceptibility, inoculum availability, and potential vectors in a Texas oak wilt centre. Journal of Arboriculture 13,169-173.

Appel DN, 1995. The oak wilt enigma: perspectives from the Texas epidemic. Annual Review of Phytopathology 33,103-118.

Barnett HL, True RP. 1955. The oak wilt fungus *Endoconidiophora fagaceraum*. Transactions of the New York Academy of Sciences Series II, 17 (7), 552-559.

Billings RF. 2008. The Texas Cooperative Oak Wilt Suppression Project: lessons learned in the first 20 years.

Bretz TW, 1952. The ascigerous stage of the oak wilt fungus. Phytopathology 42, 435-437.

Bretz TW, Long WG. 1950. Oak wilt fungus isolated from Chinese chestnuts. Plant Disease Reporter 34, 291.

Bretz TW, Buchanan WD. 1957. Oak wilt not found in acorns from diseased tree. Plant Disease Reported 41: 546.

Bruhn JN, Pickens JB, Stanfield DB, 1991. Probit analysis of oak wilt transmission through root grafts in red oak stands. Forest Science 37, 28-44. Canadian Food Inspection Agency, 2008. Ottawa, Canada: Government of Canada. http://www.inspection.gc.ca/ Accessed April 22, 2009.

Cease KR, Juzwik J. 2001. Predominant nitidulid species (Coleoptera: Nitidulidae) associated with spring oak wilt mats in Minnesota. Canadian Journal of Forest Research 31, 635-643.

CFIA 2011. D-99-03: Phytosanitary measures to prevent the entry of Oak Wilt Disease (*Ceratocystis fagacearum* (Bretz) Hunt) from the Continental United States. http://www.inspection.gc.ca/plants/plant-protection/directives/forestry/d-99-03/eng/1323852753311/1323852875523 [Accessed 28 July 2015]

CFIA 2012. *Ceratocystis fagacearum* (oak wilt) – fact sheet. <u>http://www.inspection.gc.ca/plants/plant-protection/diseases/oak-wilt/fact-sheet/eng/1325629194844/1325632464641</u> [Accessed 28 July 2015]

Cones WL, 1967. Oak wilt mats on white oak in West Virginia. Plant Disease Reporter 51,430-431.

Cook B. 2012. Oak wilt in Michigan's forest resource. Extension Bulletin E-3169. http://michigansaf.org/ForestInfo/Health/E3169-OakWilt.pdf [Accessed 28 July 2015]

Cook SJ, 2001. Current practices and suppression methods for managing oak wilt disease. In: Ash, CL, ed. Shade Tree Wilt Diseases. St. Paul, Minnesota, USA, 93-100.

Davies CS. 1992. Environmental Management of Oak Wilt Disease in Central Texas. Environmental Management 16, 323 - 333.

Defra, 2014. Tree Health Management Plan

https://www.gov.uk/government/uploads/system/uploads/attachment\_data/file/307299/pb141 67-tree-health-management-plan.pdf

Duff AG. (editor) 2012. Checklist of Beetles of the British Isles, 2012 edition.

Dorsey CK, Leach JG.1956. The bionomics of certain insects associated with the oak wilt with particular reference to the Nitidulidae. Journal of Economical Entomology 48, 219-230.

EPPO, 2011. Datasheet on quarantine pests. *Ceratocystis fagacearum* and its vectors (revised).

http://www.eppo.int/QUARANTINE/fungi/Ceratocystis\_fagacearum/CERAFA\_ds.pdf [Accessed 7 February 2015].

EPPO, 2014. PQR database. Paris, France: European and Mediterranean Plant Protection Organization. <u>http://www.eppo.int/DATABASES/pqr/pqr.htm</u>

Engelhard AW. 1955. Occurrence of the oak wilt fungous mats an pads on members of the red and white oak groups in Iowa. Plant Disease Reporter 39, 254-255.

Farr DF, Bills GF, Chamuris GP, Rossman AY. 1989. Fungi on plants and plant products in the United States. APS Press, St. Paul, Minnesota, USA. 1252 pp.

Feather S. 2007. Oak wilt: protecting oak trees. <u>http://www.treeboss.net/oak-wilt.htm</u> [Accessed 5 February 2015].

Forestry Commission. 2013. Forestry Statistics.

http://www.forestry.gov.uk/pdf/ForestryStatistics2013.pdf/\$FILE/ForestryStatistics2013.pdf [Accessed 5 February 2015].

Forestry Commission. 2014. Importing wood, wood products and bark. Plant Health Guide. 32 pp. Forestry Commission: Edinburgh. http://www.forestry.gov.uk/pdf/FCPH001.pdf/\$file/FCPH001.pdf [Accessed 5 February 2015].

<u>nttp://www.torestry.gov.uk/pat/FCPH001.pat/\$file/FCPH001.pat</u> [Accessed 5 February 2015].

French D, Stienstra WC. 1980. Oak Wilt. Extension Folder 310 - Revised 1980. Agricultural Extension Service, University of Minnesota. 6 pp.

Gibbs JN, 1979. Oak wilt. Arboricultural Journal 3, 351-356.

Gibbs JN. 1979. Measures to prevent oak wilt from reaching Europe. In: Plant Health (Eds Ebbels DL & King JE), pages 103-112.. Blackwell, OxfordGibbs JN. 1984. Oak wilt. Forestry Commission Forest Record 126. 7 pp. HMSO.

Gibbs JN, French DW, 1980. The transmission of oak wilt. USDA Forest Service Research Paper NC-185. St. Paul, MN: North Central Forest Experiment Station. 17 pp.

Gibbs JN, Liese W, Pinon J. 1984. Oak wilt for Europe. Outlook on Agriculture 13, 203-207.

Gillespie WH, True RP. 1963. Impact of the deep-girdle treatment and associated effects upon the production of fungus mats by naturally infected oak wilt trees. Plant Disease Reporter. 47(8): 748–52.

Haight RG, Homans FR, Horie T, Mehta SV, Smith DJ, Venette RC. 2011 Assessing the cost of an invasive forest pathogen: A case study with oak wilt. Environmental Management 57, 506-417. <u>http://www.nrs.fs.fed.us/pubs/jrnl/2011/nrs\_2011\_haight\_001.pdf</u>

Harrington TC. 2009. The genus *Ceratocystis*. Where does the oak wilt fungus fit? *Proceedings of the 2nd National Oak Wilt Symposium*,(eds Appel, D.N. and R.F. Billings). Austin, TX

Hayslett M, Juzwik J, Moltzan B, 2008. Three *Colopterus* beetle species carry the oak wilt fungus to fresh wounds on red oak in Missouri. Plant Disease 92, 270-275.

Hayslett M, Juzwik J, Moltzan B, Appel D. Camilli K. 2009. Insect vectors of the oak wilt fungus in Missouri and Texas. *Proceedings of the 2nd National Oak Wilt Symposium*,(eds Appel, D.N. and R.F. Billings). Austin, TX.

http://www.texasoakwilt.org/NOWS/conference\_assets/conferencepapers/HayslettJuzwikMol tzanAppelandCamilli.pdf [Accessed 7 February 2015].

Henry BW. 1944. Chalara quercina n. sp., the cause of oak wilt. Phytopathology 34,631-635.

Henry, BW, Moses CS, Richards CA, Riker AJ, 1944. Oak wilt: Its significance, symptoms and cause. Phytopathology 34, 636-647.

Himelick EB, Curl RD, Zuckerman BM. 1953. Rodent feeding on mucelial pads of the oak wilt fungus. Plant Disease Reporter 38, 588-590.

Houston DR, Drake CR, Kuntz JE, 1965. Effects of environment on oak wilt development. Phytopathology 55,1114-1121.

Houston DR, Kuntz JE, 1960. Effects of temperature and moisture on oak wilt development. University of Wisconsin Forestry Research Notes No. 67, 1-3.

Jewell FF, 1956. Insect transmission of oak wilt. Phytopathology 46,244-257.

Juzwik, J. 2000. An oak wilt primer. International Oaks 11, 14-20.

Juzwik, J. 2001. Overland transmission of *Ceratocystis fagaceraum*: extending our understanding. In: Shade Tree Wilt Diseases, ed CL Ash), pp83-92. APS Press, St Paul, Minnesota.

Juzwik J, Skalbeck TC, Neuman MF. 2004. Sap Beetle Species (Coleoptera: Nitidulidae) Visiting Fresh Wounds on Healthy Oaks During Spring in Minnesota. Forest Science 50, 757-764.

Juzwik J, Harrington TC, MacDonald WL, Appel DN, 2008. The origin of *Ceratocystis fagacearum*, the oak wilt fungus. Annual Review of Phytopathology 46,13-26.

Koch KA, Quiram GL, Venette RC. 2010. A review of oak wilt management: Asummary of treatment options and their efficacy. Urban Forestry and Urban Greening 9,1–8.

Kurdyla TM, Guthrie PA, McDonald BA, Appel DN, 1995. RFLPs in mitochondrial and nuclear DNA indicate low levels of genetic diversity in the oak wilt pathogen *Ceratocystis fagacearum*. Current Genetics 27,373-8.

Lewis R. 1985. Temperature tolerance and survival of *Ceratocystis fagaceraum* in Texas. Plant Disease 68(5), 443-444.

Liese W, Ruetze M. 1987. On the risk of introducing oak wilt on white oak logs from North America. Arboriculture Journal 11, 237 - 244.

Lin H, Phelan PL. 1992. Comparison of volatiles from beetle-transmitted Ceratocystis fagacearum and four noninsect-dependent fungi. Journal of Chemical Ecology 18, 1623-1632.

MacDonald W, Pinon J, Tainter F, Double M. 2001. European oaks—susceptible to oak wilt? In: Ash, C.L. (Ed.), Shade Tree Wilt Diseases. APS Press, St. Paul, MN, pp. 131–137.

Marini L, Haack RA, Rabaglia RJ, Toffolo EP, Battisti A, Faccoli M. 2011. Exploring associations between international trade and environmental factors with establishment patterns of exotic Scolytinae. Biological invasions 13, 2275-2288.

Merrill W. 1975. American chestnut and chestnut oak are not reservoirs of the oak wilt fungus in Pennsylvania. Plant Disease Reporter 59, 564-566.

Miller RB, Quirk JT, Christensen DJ. 1985. Identifying white oak logs with sodium nitrite. Forest Products Journal 35, 33 - 38.

Moore N. 2012. Roundwood imports and exports – an investigation. A study for the Forestry Commission. Contract CFS 03/09.

<u>http://www.forestry.gov.uk/pdf/InvestigationRoundwoodTradeData.pdf/\$file/InvestigationRoundwoodTradeData.pdf</u> [Accessed 9 February 2015].

Pinon J, Irwin H, Macdonald W, Tainter F. 1997. The susceptibility of European oaks to oak wilt. Phytopathology. 87(6 Suppl.): S114.

Rexrode CO, Lincoln AC. 1965. Distribution of oak wilt. Plant Disease Reporter 49, 1007-1010.

Rexrode CO, Brown D. 1983. Oak Wilt. Forest Insect and Disease Leaflet 29. Northcentral Forest Experiment Station, Minnesota, USA. 5 pp.

Schmidt E, Juzwik J, Schneider B. 1997. Sulfuryl fluoride fumigation of red oak logs eradicates the oak wilt fungus. Holz als Roh und Werkstoff 55, 315 - 318.

Sinclair WA, Lyon H, 2005. Diseases of Trees and Shrubs. Second edition. Ithaca, New York: Cornell University Press. 660 pp.

Southwood TRE. 1996. Philosophical Transactions of the Royal Society London B, 351, 1113-1129. <u>http://royalsocietypublishing.org/content/royptb/351/1344/1113.full.pdf</u>

Stein J, Binion D, Acciavatti R. 2003. Field guide to native oak species of eastern North America. USDA Forest Service FHTET-2003-01, pp 175.

Tainter FH, Ham DL. 1983. The survival of Ceratocystis fagacearum in South Carolina. European Journal of Forest Pathology 13,102-109.

True RP, Gillespie WH. 1961. Oak wilt and its control in West Virginia. Circ. No. 112. West Virginia Agricultural Experiment Station. 18 p.

Webber JF, Brasier CM. 1984. The transmission of Dutch elm disease: a study of the processes involved. In 'Invertebrate-microbial interactions' (eds J.M. Anderson, A.D.M. Rayner and D. Walton) pp. 271-306. Cambridge University Press.

Wilson DA, 2001. Oak Wilt: A potential threat to southern and western oak forests. Journal of Forestry, 99:4-11.

Wilson DA, 2005. Recent advances in the control of oak wilt in the United States. Plant Pathology Journal 4,177-191.

Whitford PB, Whitford KD, Whitford PC. (2007). A Marquette Country Savanna rexamined. Michigan Botanist 46, 25-32.

Yates, M.G. 1981 The subcortical fauna of oak: scolytid beetles as potential vectors of oak wilt disease. In: Last, F.T.; Gardiner, A.S., (eds.) Forest and woodland ecology: an account of research being done in ITE. Cambridge, NERC/Institute of Terrestrial Ecology, 116-117. (ITE Symposium, 8).

Date of production: 12th March 2015 (Vs 1.1), revised 30<sup>th</sup> July 2015 (Vs 1.2)

Version no.: Vs 1.2

Author (s): Joan Webber, Forest Research, Alice Holt Lodge, Farnham, GU10 4LH, UK

#### **Reviewers:**

John N. Gibbs<sup>,</sup> Ex Principal Pathologist, Forest Research

Richard Baker, Helen Anderson, Anastasia Korycinska, Melanie Tuffen The Chief Plant Health Officer Unit, Defra Plant and Animal Health, Sand Hutton, York YO41 1LZ © Crown copyright 2015

You may re-use this information (excluding logos) free of charge in any format or medium, under the terms of the Open Government Licence v.2. To view this licence visit <u>www.nationalarchives.gov.uk/doc/open-government-licence/version/2/</u> or email <u>PSI@nationalarchives.gsi.gov.uk</u> This publication is available at <u>https://secure.fera.defra.gov.uk/phiw/riskRegister/plant-health/pest-risk-analysis-consultations.cfm</u>

Any enquiries regarding this publication should be sent to Forestry Commission, Head of Plant Health Forestry Commission Cross Border Plant Health Service Silvan House 231 Corstorphine Road Edinburgh EH 12 7AT

Email: plant.health@forestry.gsi.gov.uk