Destructive tree diseases that are associated with ambrosia and bark beetles: Black swan events in tree pathology?

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Diseases that are associated with ambrosia and bark beetles comprise some of the most significant problems that have emerged on trees in the last century. They are caused by fungi in the Ophiostomatales, Microascales and Hypocreales, and have vectors in the Scolytinae (ambrosia and bark beetles), Platypodinae (ambrosia beetles) and Hylesininae (bark beetles) subfamilies of the Curculionidae (Coleoptera) (73, 102, 144). Some of these problems, such as Dutch elm disease (DED), have a long history, have been extensively researched, and are fairly well understood (23, 56, 70, 113, 149, 168, 169). In contrast, other similar diseases developed recently and are poorly or partially understood (2, 3, 42, 51, 73, 89, 94, 98, 114, 127, 137, 154, 158, 164, 165). Significant data gaps may exist for the ecology, epidemiology and management of the latter diseases.

The emergence and unexpected importance of these tree diseases are discussed in this article. An underlying factor in most of these interactions is the absence of a coevolved history

between the so-called "naïve" or "new encounter" host trees and the pathogens and/or beetles (27, 128, 174). For the ambrosia beetles, these interactions are associated with susceptibility to what are typically benign fungi and atypical relationships with healthy trees (ambrosia beetles favor trees that are dead or stressed). Interestingly, the pathogens for both the ambrosia and bark beetle-associated diseases often have symbiotic relationships with the insects that are not based on phytopathogenicity. Some of the most alarming and damaging of these diseases are considered below as "black swan events" (155).

Black Swans. Before 1697, improbable events and situations in Europe were known as "black swans" (at the time, all swans known to Europeans were white) (134). In that year, the black swan, *Cygnus atratus*, was discovered in Western Australia (38). Thereafter, "black swan" developed as a metaphor for a supposed impossibility that is contradicted with new information. For example, John Stuart Mill used black swan logical fallacy when identifying falsification, a key component in the scientific method (68).

In a recent book, Taleb (155) developed Black Swan Theory (BST). Unlike the "black swan" to which Mill referred (68), BST focuses on unexpected events of large magnitude and consequence (155). Taleb (155) recognized such events in diverse fields including finance, history, science and technology. He suggested that black swan events:

1) have extreme impacts;

2) lie outside the realm of regular expectations (they are rare); and

3) are unpredictable.

Although all ambrosia and bark beetle-associated diseases are not black swan events, several do fulfill the above criteria since they have large impacts, are uncommon surprises and are

unpredictable (2, 3, 51, 56, 73, 89, 94, 98, 114, 137, 154, 158, 164, 165) (Table 1). They are typically understood and appreciated only with the benefit of hindsight and subsequent research.

The Beetles and Beetle-associated Tree Diseases. The order Coleoptera contains more species than any other order in the animal kingdom and about 25% of all known life-forms (10, 75). About 40% (ca. 400,000) of all described insect species are beetles, and they are found in all of the world's major habitats except the oceans and North and South Poles. In the Coleoptera, the infraorder Cucujiformia contains 90 families and more than half of its known species; in turn, the Curculionidae in the Cucujiformia contains the Hylesininae, Platypodinae and Scolytinae sub-families which include the bark and ambrosia beetles (75).

There are ca 3,400 species of ambrosia beetles in 11 tribes. They evolved at least eight times from bark beetle ancestors and colonize the xylem, as opposed to the phloem habitat of bark beetles (45). Conifers were likely the ancestral hosts of these insects, but subsequent shifts to angiosperm hosts occurred, many of which were concurrent with the development of the ambrosia beetle lifestyle (45). Some reversion to conifer hosts occurred, but most extant ambrosia beetles are found in angiosperms. Extant bark beetles are also most common on angiosperms, although the most economically important species occur on pines.

Bark beetles feed on host phloem, but many species supplement their diets with symbiotic fungi (12, 20, 146). In contrast, most ambrosia beetles subsist entirely on fungi (14, 15, 17, 144). They are fungus farmers and one of four examples of agriculture in the animal kingdom (humans, attine ants and macrotermitine termites are the remaining three) (45, 118) (Fig. 1). The fungal symbionts enable ambrosia beetles to exploit a poor nutritional resource, the xylem, which they are unable to utilize directly (14, 60, 139). And for some of the bark beetles, fungal

symbionts provide sterols, nitrogen and other nutrients that are required for, or facilitate, development and reproduction (1, 12, 19, 20).

In a recent review of scolytine beetle-associated problems on trees, Hulcr and Dunn (73) considered the impact of these insects in new habitats. Although scolytines constitute less than 0.2% of all insect species, they have accounted for more than half of all interceptions at international ports of entry, due primarily to their association with packing materials that are made of nontreated wood (59). Beetles constituted 84% of the insects that were associated with wood materials imported into Chile and New Zealand, and 92% of the detections in the US (59). The increasing importance of these insects is mirrored in the scientific literature, as 50% of the journal articles on them have been published since 2008 (73).

Bark beetles. Bark beetles impact a wide range of forest and agricultural trees. They can kill trees by themselves (148), but are also associated with destructive diseases (5, 56), some of which are new (2, 158) or have increased in severity or geographic range (102).

Bark beetles are associated with diverse bacteria and fungi, the latter of which include members of the Hypocreales, Ophiostomatales, Microascales and Saccharomycetales. The relationships of the fungi with the beetles range from coevolved nutritional mutualisms, through consistent but not obligate associations, to more casual associations. Consistent relationships with fungi are thought to be less common in angiosperm-colonizing bark beetles (144), but it is on these trees that some new diseases have developed (2, 158).

Although the factors that result in the development of new bark beetle-associated problems are often obscure, three hypotheses are proposed for their development: 1) *biogeographical shifts*, in which new beetle vectors or tree hosts interact with fungi that are apparently benign in their original habitat (66, 103); 2) *pathogen-vector shifts* where pathogens join a pre-existing

beetle and tree association (2, 127, 166, 169); and 3) *host shifts* in which beetles and fungi that did not damage their original host tree(s) move to and damage a new host tree species (94, 158). These hypotheses are not mutually exclusive and some diseases have components of more than one of these scenarios. For example, both biogeographical shifts and pathogen-vector shifts may be involved with the DED pathogens and vectors that are discussed below.

Ambrosia beetles. The tree diseases that are caused by the fungal partners of ambrosia beetles are unusual. Typically, ambrosia beetles infest only dead or stressed trees since healthy trees are usually not suitable as brood sites (15). In new environments where ambrosia beetles are introduced and subsequently establish, most species continue to colonize only dead or stressed hosts. Some, however, attack living and apparently healthy trees (99, 136).

Why some ambrosia beetles attack healthy trees in the new locations is unclear. Hulcr and Dunn (73) suggested that olfactory miscues, in which the insects mistake healthy trees as potential brood sites, might be responsible. Alternatively, could attacks of healthy trees by some ambrosia beetle species (see 99) be relatively common? Might ambrosia beetles make tentative, but unremarkable, contact with healthy trees, which is only recognized when conspicuous damage or related disease problems develop? Greater understanding is needed for how, and the extent to which, these insects interact with healthy trees.

More significant, in the context of this review, is the phytopathogenicity of some of the fungal symbionts of ambrosia beetles. These fungi usually colonize the lining or more substantial portions of natal galleries (14, 150), but typically do not cause disease. Thus, the phytopathogenicity of some of the symbionts is unexpected (51, 89, 114, 120).

When ambrosia beetle infestations are associated with serious damage, a wide range of interactions is evident. At one extreme are ambrosia beetles for which there is no clear

involvement of the symbionts (6, 13, 99) (Table 1). In these cases, the beetle alone is reported to cause significant damage on the host tree. In other situations, ambrosia beetle-associated damage relies on significant infestation by the insect and associated disease that is caused by moderately virulent symbionts (89, 120, 161). [In this article, "virulence" is used to denote the relative degree to which a pathogen affects a host (148).] In these cases, substantial damage on the host tree would not develop without repeated insult by both the beetles and fungi. In the most extreme examples, single infections by the symbionts are capable of causing serious damage or host death (51, 53, 123, 133). In the latter situations, continued infestation by the beetle and multiple infections by the symbiont may occur, but are not needed to kill trees. Considering the significant damage that can develop in the above situations, all would fall outside the "typical" impact one might expect when ambrosia beetles interact with healthy host trees.

The factors that are involved in ambrosia beetle-associated disease are less clear than those for the bark beetle-associated diseases. Although Hulcr and Dunn (73) indicated that "The virulence that we see in the 'new pests' has in no case been documented to be their typical ecological strategy in their native forests," it is probable that at least some of the symbionts are pathogenic in their centers of origin, and that selective disease pressure occurs in these cases (133). Coevolved resistance that would develop in the host tree in these situations would involve the attrition, via disease, of susceptible portions of the host populations.

The fungal symbionts. The symbiotic fungi in ambrosia and bark beetles are often transported in specialized structures called mycangia (14, 144). Mycangia are located within mouthparts, the thorax, or elytra of these insects, and are found in females, males or both sexes, depending on the species (55, 144) (Fig. 2). They include invaginations of an insect's integument that are lined

with secretory structures that facilitate the acquisition and transport of these fungi (52, 153) or, when loosely defined, the above structures as well as pits, pockets and setae that enable the transport of these fungi (144).

Since the fungal symbionts can serve indespensible or beneficial nutritional roles and are often transported in specialized structures, it is not surprising that there can be a high level of specificity between the symbionts and beetles. For example, vertical transmission of symbionts (from one generation to the next) occurs in many species (4, 17, 92, 145), and coevolution has been demonstrated or suggested in some of the bark beetle x fungus interactions (92, 145, 148). Nonetheless, lateral transmission of symbionts from one species to another also occurs in ambrosia and bark beetles, and this includes symbionts that are plant pathogens (5, 34, 55, 56, 64, 127, 130, 148).

Six (144) indicated that lateral transfer was most common with non-mycangial fungi that were carried externally (phoretically) on an insects' exoskeleton. However, lateral transfer of mycangial species may also occur when a species interacts with another species' brood gallery and its associated fungi (145). Clearly, lateral transfer increases the numbers of potential vectors, and with this increase would come increased chances for symbiont transmission to a greater number of host tree species. In bark beetles, both vertical and lateral transmission of symbionts also occurs via fungus-feeding phoretic mites (70, 93, 117). Much less is known about fungus-associated phoretic mites on ambrosia beetles, but they also occur on these insects (Carrillo et al., unpublished). Whether they transmit fungal symbionts warrants study.

By reducing host tree defenses, phytopathogenic symbionts are thought to facilitate colonization of, and damage to, host trees by some bark beetles (148, 167). Recently, Six and Wingfield (148) evaluated this "classic paradigm." They suggested that phytopathogenicity

primarily benefits the fungi, rather than the beetles, and that bark beetles benefit from these associations when the fungi provide nutritional benefits but do not rely on their pathogenicity to facilitate host tree colonization. Although the conclusions of Six and Wingfield (148) are controversial, they are, nonetheless, thought-provoking and have stimulated additional research (167).

Similar analyses have not been conducted for ambrosia beetles. However, if their symbionts increased the numbers of dead or stressed host trees in which they could reproduce, these insects would evidently benefit from these associations. A greater understanding is needed for how interactions among ambrosia beetles, phytopathogenic symbionts and host trees develop and evolve.

Although the fungal symbionts often serve significant nutritional roles for these insects, uncommon symbionts that cause tree diseases are the focus of this article (Fig. 3). Below are summarized general attributes of ambrosia and bark beetle-associated tree diseases, their history and significance, and factors that are related to their development (Table 1). The most destructive and surprising of these diseases are highlighted as black swan events.

Leach's rules

The etiologies of ambrosia and bark beetle-associated tree diseases are usually determined soon after they become evident, since the causal agents are often easy to isolate and the completion of Koch's postulates is straightforward. Due to their small size and the common occurrence of secondary beetles species in damaged trees (which play no role in the development of these diseases), the roles of the respective beetle vectors may be less clear, at least during the early stages of a disease outbreak.

Leach (101) reviewed insect vectors of plant pathogens and established four rules to confirm that an insect was the vector of a given pathogen. He indicated that it was necessary to demonstrate: 1) a close association of the insect with diseased plants; 2) regular visits of healthy plants by the insect; 3) an association of the pathogen with the insect; and 4) the development of the disease in healthy plants after interaction with pathogen-infested insects. In general, Leach's criteria have been met for the vectors and diseases that are discussed below and listed in Table 1. However, in a wider consideration of these problems, probable and unlikely examples of pathogen vectors are also discussed (those for which Leach's criteria have not been met).

Dutch elm disease (DED)

DED is the most well-known example of the diseases that are discussed in this article. It is not a new problem but is considered here as a black swan event, since it: 1) has had a huge impact, especially on native elms in North America; 2) was uncommon (no other disease had impacted such a large group of forest and urban landscape trees over such a wide area), and 3) was an unpredictable surprise (70, 178). DED serves as a model for similar diseases for which far less is known. Biogeographical and pathogen-vector shifts for the DED pathogens, vectors and hosts are discussed below, and provide useful insight into the ecology and epidemiology of this and other bark beetle-associated diseases.

DED first appeared in Europe around the 1st World War, and was described in 1921 in the Netherlands (thus, its common name) and in 1930 in the USA (Ohio) (70, 178). DED spread rapidly in Europe and North America, affecting native elms, *Ulmus* spp., on both continents. It has killed over 40 million trees of American elm, *U. americana*, and remains a problem on native elms in Europe (23).

Two DED epidemics are associated with the so-called "nonaggressive" and "aggressive" strains of *Ophiostoma ulmi* (22). The nonaggressive strain [now *O. ulmi* (Fig. 4A)] caused the first epidemic but was not recognized until the second epidemic, caused by the aggressive strain (now *O. novo-ulmi*), developed in Europe in the mid-1900s. Subsequently, two subspecies of *O. novo-ulmi* were recognized: *O. novo-ulmi* subsp. *americana*, introduced on timber from North America to Europe in the late 1960s, and *O. novo-ulmi* subsp. *novo-ulmi*, which moved to Europe from the East in the early 1970s (22, 23). *Ophiostoma ulmi* and *O. novo-ulmi* have distinct cultural characteristics, temperature responses and virulence on various elm hosts. In general, both *O. ulmi* and *O. novo-ulmi* are highly virulent on American elms, whereas *O. ulmi* is moderately virulent and *O. novo-ulmi* is highly virulent on European elms (22).

Several species, interspecific hybrids and clonal selections of elm are part of the DED story (149). The American species are most susceptible and include the American elm (*U. americana*), rock elm (*U. thomasii*), and slippery elm (*U. rubra*). The European species are generally less susceptible than the American species, and they include the European field elm (*U. carpinifolia* = *U. minor*), English elm (known as *U. procera*, but which is probably a clone of *U. carpinijolia*), wych elm (*U. glabra*), and hybrids between *U. carpinifolia* and *U. glabra* (aka *U.* X *hollandica*). Asian species, including *U. pumila*, *U. wilsoniana*, *U. parvifolia*, and *U. japonica*, are highly tolerant [That is, endure infection without developing severe disease (177)]. Moderately tolerant selections of the new encounter elms have been selected, but the most tolerant genotypes are the Asian species and their hybrids with other species (149).

A better understanding of the causal fungi and past and current DED epidemics would be possible with information on the pathogens in their centers of origin. The response of the Asian elm species to DED has led to the presumption that the DED pathogens originated in Asia (56).

Although this hypothesis was questioned after the pathogens were not found during surveys in Asia (24), it was resurrected with a recent finding of *O. ulmi* and *O. novo-ulmi* subsp. *americana* in Japan (109). Although little is known about the DED pathogens in Asia, they are evolving in Europe: hybridization has been demonstrated between the *O. novo-ulmi* subspecies, and gene introgression from *O. ulmi* to *O. novo-ulmi* has been suggested (22, 23). Apparently, similar research has not been conducted in North America.

At least 10 bark beetle species have been indicted as vectors, only a few of which are significant (56, 168, 169). In Europe, the native large elm bark beetle, *Scolytus scolytus* (Fig. 5A), is a better vector than *S. kirschi*, *S. laevis* and *S. multistriatus* (Scolytinae: Scolytini) (Fig. 5B), whereas the introduced *S. multistriatus* is a better vector than the native *Hylurgopinus rufipes* (Scolytinae: *Hylesinini*) (Fig. 5C) in North America.

If the DED pathogens originated in Asia they were laterally transferred to the above vectors when they were introduced to Europe and North America. Rather than providing nutritional benefit for the vectors, the pathogens are apparently detrimental to the beetles. Webber and Gibbs (170) reported that fungus-free host tissue was necessary for the first stages of beetle development, and that gallery construction stopped when *Scolytus* larvae fed on tissues colonized by *O. ulmi sensu lato* (*s.l.*). Active portions of the insects' galleries developed in front of the advancing pathogen.

The DED pathogens are external contaminants of these insects (they are not transmitted in a mycangium). Conidia of the pathogen are produced in sticky masses that facilitate their acquisition and transport by their vectors as they emerge from host trees (Fig. 4A). The amounts of inoculum that they obtain when doing so is a determinant of pathogen transmission and disease development (169).

During studies on *U. americana* and *H. rufipes*, McLeod et al. (113) detected increased attraction of the beetle to trees that were infected by *O. novo-ulmi*. Four semiochemicals [(-)-3-pinene, (-)-a-cubebene, (+)-spiroaxa-5,7-diene and (+)-8-cadinene] from DED-affected trees attracted the beetle, and emission of the latter three compounds was upregulated in trees that were inoculated with the pathogen. McLeod et al. (113) concluded that *O. novo-ulmi* manipulates host trees to enhance their detection by *H. rufipes*, thereby improving the chances that the pathogen will be disseminated to a new host tree. Perhaps more likely, *H. rufipes* may have evolved an upregulated positive response to these compounds in stressed trees.

Several vector-associated factors influence transmission of the DED pathogens to host trees, including host specificity, numbers of generations that emerge in a season, body size, and the quantity and quality of pathogen inoculum that is carried (169). In Europe, the more efficient vector *S. scolytus* produces two generations per year compared to one for *S. multistriatus.* Webber (170) reported that the larger *S. scolytus* was more frequently associated with *O. ulmi* and *O. novo-ulmi* in its galleries and carried more inoculum more consistently than *S. multistriatus*; she recovered up to 350,000 spores on 51 of 52 individuals of *S. scolytus* vs $\leq$30,000 spores on 32 of 50 assayed *S. multistriatus*.

The development of DED in Europe and North America during the last century has several important aspects. Clearly, different pathogens have influenced the spectrum of new encounter host trees and the intensity with which they have been impacted. As susceptible elm genotypes have been decimated and new more tolerant hybrids and selections have been developed and deployed, the pathogen has been confronted with new hosts. Time will tell whether the new elms will effect change in these pathogens. Notably, diverse native and exotic bark beetle vectors of varying efficiencies have surfaced during these epidemics, and those that have been

studied appear to derive no nutritional benefit from their association with the DED pathogens. Although DED-compromised trees may provide increased habitat for these insects and attract at least one of the vectors (113), the DED relationship appears to be one of hitch-hiking, opportunistic phytopathogens on coincidental vectors (148). Some, but not all, of the bark beetle-associated diseases have similar relationships with their causal agents.

Examples of other bark-beetle associated tree diseases

Biogeographical shifts. The biogeographical shift hypothesis indicates that fungi that are benign in their original habitat can affect new encounter hosts when they are moved to the native ranges of these trees, or when the new encounter host trees are moved to areas in which the fungi reside. The DED pathogens are presumably examples of the former type of biogeographical shift. In contrast, diseases incited by species in the *Ceratocystis fimbriata sensu lato* (*s. l.*) complex may be examples of the latter type of biogeographical shift (66), and a bark beetle is a factor in the epidemiology of one of these diseases (137).

Ceratocystis fimbriata s.l. causes lethal canker and wilt diseases of economically important new encounter hosts (47, 140), but it is a non-aggressive colonizer of wounds on the original hosts in their native ranges (78, 156, 173). *Ceratocystis fimbriata s. l.* includes many tree pathogens (40, 41, 78, 87, 156, 163-165, 172). The wide host range of *C. fimbriata s. l.* led Webster and Butler (171) to propose that it was a species complex, a view that has continued to develop (140). As the phylogeny of this pathogen becomes better understood, it appears that *C. fimbriata sensu stricto* (*s.s.*) refers specifically to the sweet potato pathogen that was described by Halsted and Fairchild (61), and that other members of the *C. fimbriata s.l.*complex cause canker and wilt diseases on other host species (41, 78, 140, 163-165).

Species in the *Ceratocystis fimbriata s.l.* complex are moved easily with infected germplasm, infested soil, and on pruning implements. However, bark beetles disseminate some of these pathogens (2, 3, 137, 173), and ambrosia beetle dissemination has been suggested for others (57, 81). The seca and sudden decline diseases of mango, which are black swan examples of biogeographical shifts for *Ceratocystis*–incited diseases, are discussed below under pathogen-vector shifts.

In some biogeographical shifts, there is evidence for the evolution of the pathogenic symbionts. Brasier and Kirk (23) reported that hybridization occurred between *O. novo* subsp. *americana* and *O. novo-ulmi* subsp. *novo-ulmi* in several locations in Europe. Hybrids had similar growth rates and pathogenicity as the parental subspecies, and were significant components of the assayed pathogen populations. Brasier and Kirk (23) concluded that complex hybrid swarms of these pathogens were spreading on the continent.

Increased fitness and virulence of another biogeographical-shift symbiont was reported in China (103). *Leptographium procerum* (Fig. 4B) was introduced from the USA to China by the red turpentine beetle, *Dendroctonus valens* (Hylesininae: Tomicini) (Fig. 5E). Novel genotypes of the fungus were recently identified in China that were more pathogenic to the new encounter species *Pinus tabuliformis* than were USA genotypes of the fungus. The novel genotypes also induced the release of higher amounts of 3-carene, a primary attractant for the beetle, in inoculated seedlings (103). Lu et al. (103) concluded that *L. procerum* adapted to its new encounter pine hosts after its introduction to China.

Pathogen-vector shifts. A second observation on bark beetle-associated diseases indicates that some pathogens joined pre-existing beetle and tree relationships. Pathogen-vector shifts are

apparent for the DED pathogens, as well as the seca and sudden decline pathogens of mango (*Mangifera indica*) (Fig. 6).

Seca and sudden decline possess the black swan attributes of significant impact, surprise and unpredictability. For example, in Oman large areas of production that were grown on susceptible Omani rootstocks have been decimated by sudden decline (2). Prior to the development of seca and sudden decline, no routinely lethal disease was known on mango, and they remain the most destructive of all diseases on this important crop (127, 129).

Seca disease has been recognized since the 1930s in Brazil (138, 142, 166). Recently, Van Wyk et al. (165) described two new species with multigene geneologies, *Ceratocystis mangicola* and *C. mangivora*, in a collection of seca isolates from Brazil

Sudden decline, a very similar (identical?) disease of mango, was first recognized in Oman in 1998 and spread rapidly to affect other mango-production areas in that country (2). Sudden decline has probably been present, but misdiagnosed (105, 108), in Pakistan for as long as it has been in Oman. The sudden decline agent in Oman and Pakistan is *C. manginecans* (164). It is closely related to, but distinct from, *C. mangicola* and *C. mangivora* (165). Although *C. manginecans* had been presumed to originate in Brazil (66, 129, 165), there is no evidence that it occurs in that country. The original host(s) and geographic origin for *C. manginecans* are unclear, but there is emerging evidence that it is an Asian fungus that has come in recent contact with non-native *Acacia* spp. that are grown in plantations in Indonesia (156), Vietnam and Malaysia (M.J. Wingfield, unpublished) (Fig. 7). Likewise, the original hosts of *C. mangicola* and *C. mangivora* in Brazil are unclear. Given the benign host relationships and cryptic habitats (e.g. soil) that are often associated with these fungi, the orginal hosts of *C. mangicola*, *C. manginecans* and *C. mangivora* may remain obscure.

The mango bark beetle, *Hypocryphalus mangiferae* (Scolytinae: Cryphalini) (Fig. 5F), originated in the same areas in southern Asia where mango evolved (11, 30, 119, 176). It is now found worldwide on this tree, including many areas where seca and sudden decline are not found (124, 130). Despite assertions that "…specific vectors of *C. fimbriata* in Latin America are not known…" (47), *H. mangiferae* is a significant factor in the development of seca, as well as sudden decline (2, 3, 127, 137) (Figs. 6 B&C).

In Brazil, Ribiero (137) indicated that *H. mangiferae* produced galleries in seca-affected trees, and that it was found on healthy, as well as diseased, mango trees. Other scolytines were also associated with seca, including *Xyleborus* ambrosia beetles (137), but the latter insects were found only in diseased trees. In Pakistan, four beetles were associated with sudden decline, including *H. mangiferae*, *Sinoxylon* sp., *Xyleborus* sp. and *Nitidulidae* sp. (105, 164). However, only *H. mangiferae* was found in trees during the early stages of disease development (107). Leach's rules have been completed for *H. mangiferae* in Brazil and Oman (3, 137).

Infestation of *H. mangiferae* with the respective pathogens varies greatly in Brazil (1% of the assayed individuals) (138) and Oman (13-83%) (2). How *H. mangiferae* carries these pathogens and whether the insect has mycangia are not known. However, these results may suggest inconsistent phoretic associations with these pathogens. In Brazil, *H. mangiferae* was attracted to cultures of *C. fimbriata s.l.* in olfactometer tests, and larvae of the insect were raised to adulthood on the fungus (137). Since *H. mangiferae* consumes at least one of the seca pathogens, there is evidence for a beneficial symbiont x bark beetle interaction, as well as the previously mentioned antagonistic interactions (170).

Host shifts. New encounter host trees are also affected by bark beetle x fungus combinations that are benign on their original host(s). Thousand cankers disease (TCD) of black walnut (*Juglans*

nigra) was first recognized in Colorado, outside the native range of this tree (158) (Fig. 8). Soon after, it was described in other western states (94), and it spread recently to the tree's native range (58). TCD fits the criteria for a black swan disease, in that it is eliminating large numbers of black walnut in all of the above areas (high impact), was rare and unexpected, and was an unpredictable surprise. TCD kills trees and is the most destructive problem on mature specimens of this valuable tree. It remains an incompletely understood disease.

Geosmithia morbida, a new Hypocreales species, was reported as the cause of TCD (94). It has a bark beetle vector, *Pityophthorus juglandis* (Scolytinae: Corthylini) (Fig. 5G), which was first described in 1928 in New Mexico. As recently as 1992 *P. juglandis* was known only in that state, Arizona, California and Chihuahua, Mexico (37). The Arizona walnut (*J. major*), the insect's presumed original host, is not affected greatly by the beetle. *Geosmithia morbida* has been recovered from necrotic tissue adjacent to *P. juglandis* galleries in *J. major*, in which branch dieback and mortality did not develop (94).

Geosmithia spp. are found in diverse habitats, but are most commonly associated with bark and ambrosia beetles for which they are nutritional symbionts (95-97). Although Kolarık et al. (94) listed several studies in which these fungi had been examined as potential tree pathogens, *G. morbida* is the first for which phytopathogenicity has been documented (158). Isolates of *Fusarium solani* are also recovered from TCD lesions, and some of these cause significant, albeit smaller, lesions on inoculated *J. nigra* (158). The role that *F. solani* may play in the development of TCD is unclear. However, since *F. solani* replaces *G. morbida* in TCD lesions in Tennessee after late August (58), it could be an aggressive colonist of dead and dying phloem in these trees.

Unlike the previously discussed phytopathogenic species of *Ophiostoma* and *Ceratocystis*, which are virulent pathogens that can kill susceptible species after a single inoculation, *G. morbida* causes far less damage by itself (158). The lethal impact of TCD on *J. nigra* is associated with mass attack by *P. juglandis* and the numerous, associated lesions that develop in affected trees (thus the disease's common name) (Fig. 8C). Mass attack is also associated with, and is presumably required for, serious damage that is caused by some of the ambrosia beetle-associated diseases that are discussed in the next section.

Ambrosia beetle-associated diseases

In general, less is known about tree diseases that are associated with ambrosia beetles than with bark beetles. A range of symptoms and symptom severities are evident in the various ambrosia beetle situations. Only one of these diseases, laurel wilt, is clearly a systemic vascular wilt (51, 133). For most of these diseases, there is a more localized development of symptoms and tree and branch mortality depends on mass attack by the vectors and multiple infections by the symbionts (89, 114, 120, 161).

Some of the ambrosia beetle situations resemble those that are associated with bark beetles, in that the beetles damage trees without an apparent contributing role of the symbionts. Kuhnholz et al. (99) listed five "primary" attacking ambrosia beetles, three of which exhibit unusual aggression on healthy trees and may not rely on phytopathogenic symbionts: *Corthylus columbianus* (Scolytinae: Scolytini: Corthylina), *C. punctatissimus* and *Megaplatypus mutatus* (Platypodinae: Platypodini) (Fig. 5H) (6, 12, 79, 99, 122, 150) (Table 1). For example, fungi that were associated with *C. columbianus* activity on silver maple (*Acer saccharinum*) caused no significant damage on detached or intact host material when used in inoculation experiments

(79). And in recent reviews on *M. mutatus*, Alfaro et al. (6) and Smith (150) mentioned no role for its symbiont, *Raffaelea santoroi*, in the significant damage that this beetle causes on a very wide range of host trees (179).

Interestingly, for other ambrosia beetles that Kuhnholz et al. (99) listed as primary or secondary attackers of trees the roles that phytopathogenic symbionts may have played in causing tree damage (for example, *Platypus apicalis* (Fig. 5I) and *Platypus quercivorus*) (Platypodinae: Platypodini) (Table 1) was not mentioned. The assumption that ambrosia beetle-associated damage is caused solely or predominantly by the insect is common in the literature and highlights the need for cooperative research on these problems between entomologists and plant pathologists. This assumption may be responsible for some ambrosia beetle-associated tree diseases escaping wider attention. A disease that affects *Nothofagus* spp. is a good example.

For several decades, *Nothofagus* spp. and other native trees in New Zealand have been affected by a disease that stains sapwood, reduces the marketability of harvested timber and kills trees (8, 46, 53, 115, 123, 175). The disease is caused by *Sporothrix nothofagi*, which is transmitted by three species of ambrosia beetle, *Platypus apicalis* (Fig. 5I), *P. gracilis* and *Treptoplatypus caviceps* (formerly *P. caviceps*) (Platypodinae: Platypodini) (8, 53). The beetles attack healthy, as well as unhealthy trees, and use an aggregation pheromone to initiate mass attacks (116). Although numerous galleries of the beetles are found in affected trees, the fungus alone is capable of killing trees after artificial inoculation (123). Moisture stress induced by waterlogging or drought has been associated with enhanced tree mortality (46, 123). Despite the "well established" pathogenicity and impact of this fungus and its interaction with the ambrosia beetle vectors (123), publications on the problem often indicate that the insects cause the observed damage, rather than associated disease (8, 115).

Systemic and lethal damage caused by an ambrosia beetle symbiont. Laurel wilt (LW) is an exemplary black swan disease. In less than a decade, it has spread rapidly in the southeastern USA and killed millions of reday (*Persea borbonia*) and other native host trees in the Lauraceae (50, 51, 72, 130, 151). Most members of the Lauraceae that are native to the USA are susceptible to LW, and the current epidemic is dramatically changing the composition of southeastern forests with respect to this plant family. Yet, when it was first observed outside Savannah, GA, LW was not even recognized as a disease. Its puzzling development highlights the unusual and unpredictable impact it has had in urban environments and forest and agroecosystems in the southeastern USA

LW affects American members of the Lauraceae, which include significant components of Coastal Plain forest communities in the southeastern USA (Figs. 9 A & B), and rare members of the family that are threatened with extinction (50, 51, 72, 130, 131, 151). Avocado (*P. americana*) is affected in Florida (no. 2 producer in the USA, ca $55 million per year) (Figs. 9 C-E), and there is major concern that the disease will spread to California (no. 1 in USA, ca $350 million per year) and Mexico (world's top producer) (43, 44, 130, 133). Other members of the Lauraceae, which are keystone species in tropical American ecosystems (21, 29, 35), may be at risk. LW is caused by *Raffaelea lauricola* (51, 65), which has an Asian ambrosia beetle vector, *Xyleborus glabratus* (Scolytinae: Xyleborini) (Fig. 5J) (51).

In May 2002, *X. glabratus* was reported for the first time in the Western Hemisphere in Port Wentworth, Georgia (135). LW was recognized in the surrounding area in 2004, and by July 2012 had spread as far west as Jackson County, Mississippi, as far north as Sampson County, North Carolina, and as far south as Miami-Dade County, Florida (162) (Fig. 10). The disease's rapid movement has been due to the pathogen's mobile vector, the movement of infested wood

(redbay is a preferred barbeque fuel and is used by wood turners), and the presence of native and non-native plants throughout the southeastern USA that are susceptible to LW and on which the vector reproduces (36, 130). The genetically and pathogenically homogeneous population of the pathogen that is present in the USA indicates the establishment and spread of a single, asexually reproducing clone of the fungus (71; Hughes et al., unpublished).

Recently, *R. lauricola* was recovered from specimens of *X. glabratus* from Japan and Taiwan (67). Since there are no reports of LW in the Americas prior to 2003, it is assumed that the beetle carried the pathogen when it was first detected in 2002, and that American hosts in the Lauraceae are all new encounters. An Asian member of the family, camphortree (*Cinnamomum camphora*), is affected by LW but often recovers after natural infection (152). Although there is scant information on the susceptibility of other Asian members of the family, coevolved resistance to LW may have occured in other members of the family in Asia, where there are no reports of LW, despite a great diversity of potential host trees in the Lauraceae. For example, at least four genera in the family that contain LW-susceptible species in the USA (*Lindera*, *Litsea*, *Persea* and *Sasafrass*) (49-51, 72, 130, 151, Hughes, unpublished), are widely distributed in Asia (35).

A single inoculation with as few as 100 conidia of *R. lauricola* can kill avocado and redbay (Hughes et al., unpublished). Within a week of inoculation, the pathogen colonizes the entire height of 1.5 m-tall, potted avocado plants (133). Xylem function is impaired as soon as 3 days after inoculation (before the development of external or internal symptoms of the disease), and reductions in hydraulic conductivity that develop in these plants are correlated with increased tylose formation in xylem lumina (76, 77). Tree mortality is associated with conductivities of 10% or less of those in healthy plants. Yet, even in these plants there is surprisingly little

microscopic evidence of the pathogen (77). How such a minor presence of the pathogen can induce such dramatic changes in its avocado host is not clear.

Why *X. glabratus* bores into healthy host trees has been investigated by entomologists and chemical ecologists. In general, plants in the Lauraceae have been most attractive to this insect. Mayfield and Hanula (111) reported that female *X. glabratus* (males are flightless and not involved in pathogen dispersion) were equally attracted to swampbay (*P. palustris*) and camphortree, which were more attractive than avocado and two other lauraceous natives in the USA, lancewood (*Ocotea coriacea*) and sassafras (*Sassafras albidum*). Other trees in the Magnoliaceae, Fabaceae, Fagaceae and Sapindaceae were less attractive or not attractive to the insect (62, 111, 112). α-copaene was identified as an important semiochemical attractant in lauraceous hosts, as well as the non-host lychee (*Litchi sinensis*, Sapindaceae) in which the beetle did not establish (82, 83, 121). Avocado seems to be a less preferred host for *X. glabratus* than other members of the Lauraceae. However, in no-choice tests female *X. glabratus* bored into plants of all avocado cultivars that were tested, resulting in the development of LW (112, 125). Thus, in this unusually specialized ambrosia beetle an important determinant of whether or not a particular tree species will be attacked appears to be the profile of volatiles that are emitted by the tree. In addition, *X. glabratus* is attracted to its symbiont, *R. lauricola* (74).

Lateral movement of fungal symbionts from one species to another is well known in bark beetles (145), and has also been reported among some ambrosia beetles (13, 50, 55, 130). To date, 14 ambrosia beetle species have been detected on avocado, and the mycangial mycofloras of the most common species on avocado, redbay and swampbay have been assessed (33, 34, 64, 82; Pena et al., unpublished; Ploetz et al., unpublished). *Raffaelea lauricola* has been isolated from eight ambrosia beetles, in addition to *X. glabratus* (34, 130, Campbell et al., unpublished).

Far more propagules of *R. lauricola* have been detected in *X. glabratus* than in the other species (on average, about 30 times more than the next greatest carrier), but the pathogen is found frequently in at least three of the latter species (34, Campbell et al., unpublished). For example, *R. lauricola* was recovered from 59% (70 of 118) of the assayed females of *X. ferrugineus* (Fig. 5J) and 51% (20 of 39) of the assayed females of *X. volvulus* (Fig. 5K), compared to 86% (43 of 50) of the assayed females of *X. glabratus* (34).

In recent no-choice studies with these insects, *R. lauricola* was transmitted to avocado by two species and to redbay by six species other than *X. glabratus*; LW developed after infestation with, respectively, one and five other species (34). Thus, other beetle species could be vectors of this pathogen. If so, they could help the pathogen expand its host range and may influence the development of LW, especially on hosts that support little or no reproduction of *X. glabratus*. Greater understandings of these possibilities are needed.

Mortality associated with localized damage caused by the symbiont. Other, recognized ambrosia beetle-associated tree pathogens are incapable of inciting lethal disease by themselves. For example, two diseases of oak (*Quercus* spp.) in Asia are caused by fungi that effect restricted damage in their hosts. For both of these diseases, mass attack by the respective vectors is associated with, and apparently required for, tree mortality (90, 102). Both diseases qualify as black swans, in that large swaths of native oak trees have been killed by unpredictable and hitherto unknown diseases (89, 98).

Japanese oak wilt (JOW) is caused by *R. quercivora* (98). Its vector, *Platypus quercivorus* (Platypodinae: Platypodini), transmits the pathogen to members of the Fagaceae, among which there is a considerable range in susceptibility (90, 120). Global warming has been proposed as a

reason for the development of JOW, as the beetle has extended its geographic range northward into the ranges of previously unencountered host species (80).

By artificial inoculation, Murata et al. (120) distinguished susceptible (*Quercus crispula* and *Q. serrata*) and tolerant (*Q. glauca* and *Castanopsis cuspidata* var. *sieboldii*) host taxa. Tylose formation was associated with xylem dysfunction in JOW-affected *Q. crispula* (98), and sapwood around inoculation sites in *Q. crispula* and *Q. serrata* became nonconductive before discoloration (symptom development) was evident (120). However, even in susceptible species symptoms of JOW stopped expanding 2 weeks after inoculation (120).

Korean oak wilt (KOW) is caused by *R. quercus-mongolicae* (89). Its vector, *P. koryoensis* (Platypodinae: Platypodini), transmits the pathogen to the highly susceptible Mongolian oak (*Q. mongolica*), and to a lesser extent to *Q. aliena* and *Q. serrata* (89, 102). Similar to the hypothesis of Kamata et al. (80), Kim et al. (89) proposed that climate change was responsible for the new encounter between *Q. mongolica* and *P. koryoensis*+*R. quercus-mongolicae.*

In a study that examined attack patterns by *P. koryoensis*, Lee et al. (102) noted that the severity of KOW increased as the density of entrance holes for the beetle increased; only in trees with high attack densities did severe disease develop. Lee et al. (102) also noted that larger trees tended to develop more severe disease than smaller trees. An aggregation pheromone that is produced by unmated males of *P. koryoensis* (which are responsible for initiating new colonies of this species) may play a role in the greater development of KOW on large trees (88).

Bigger trees also develop more severe symptoms of LW (51, 133). In the KOW and LW pathosystems the vectors may respond to visual cues. Attack densities of *P. koryoensis* on Mongolian oak increased with increasing stem diameters, and numbers of *X. glabratus* that were attracted to artificially baited stem silhouettes increased as did their diameters (102, 110).

Localized, nonlethal damage caused by ambrosia beetle symbionts. The tea shothole borer, *Euwallacea fornicatus* (Scolytinae: Xyleborini) (Fig. 5L), has been known for more than a century in southern Asia where it colonizes dead or declining members of at least 36 plant families (9, 26). It has also been reported to attack live agricultural crops, including tea (*Camellia sinensis*), avocado, citrus (*Citrus* spp.), cacao (*Theobroma cacao*) and rubber (*Hevea brasiliensis*).

Little information is available on damage that develops on most hosts of this insect. However, damage on tea is associated with a pathogenic associate of the beetle in the Hypocreales, *Fusarium ambrosium* (syns.: *F. ambrosianum*, *F. bugnicourtii*, *Monacrosporium ambrosium*) (9, 25, 143). Although this pathogen may cause damage on other hosts of *E. fornicatus* in Asia, reports on the beetle usually do not mention associated disease (26).

Within the last decade *E. fornicatus* has invaded many new areas in the Eastern and Western Hemispheres (31, 135). In the West, it was first reported in Panama in 1979 (91), and in the USA was first detected in Florida (2002) and California (2003); it is now widely established in the Americas (9, 154).

Recently, a branch dieback and canker disease was associated with *E. fornicatus* on avocado and other trees (32, 42, 114) (Fig. 11). This disease is much more severe in California and Israel than in Australia and Florida. Mass attack by the insect occurs in these trees, but trees are usually not killed. Rather, branches die and portions of the canopy are lost.

The new disease was originally attributed to *F. ambrosium* (32). However, recent work indicates that close relatives of this fungus are involved in different areas. Multigene geneologies distinguished *F. ambrosium* from two *Fusarium* spp. in Florida, another species of *Fusarium* in California and Israel, and a fourth species in Australia (42, 114; Freeman et al.,

unpublished; Kasson et al., unpublished). Artificial inoculations of avocado in Florida, California and Israel indicate that the new species are moderately pathogenic (42, 114, Ploetz et al., unpublished). In California and Israel, the great and surprising impact of Fusarium dieback on avocado and its unexplained (and expanding?) host range on other tree species (154) qualify this as a black swan disease.

Genetic variation has been detected in *E. fornicatus*. Although species delimitation is difficult in beetles that reproduce via inbreeding, COI mtDNA sequences suggest that the beetle populations in California and Israel are identical, but differ from those in Australia, Florida and Sri Lanka; both populations may be distinct from bone fide *E. fornicatus* (R. Stouthamer, personal communication). The insect in California was recently given a common name, the polyphagous shothole borer, to distinguish it from the tea shothole borer (154).

More research is needed on the diseases in the different locations as it is unclear whether differences in the beetle populations, fungi or other factors are responsible for disease that develops in the different areas. For example, is the severe damage that develops in California and Israel due to the pronounced Mediterranean environments that are found there, or are more aggressive beetles or virulent fungi involved in these areas than in Australia and Florida?

Predisposition. Ambrosia beetle-associated tree diseases develop in diverse situations. Although new encounters underlie many of the interactions, other factors are associated with some of these problems, including climate change and environmental predisposition.

Recently, Ranger et al. (136) summarized factors that could predispose plants to attack by ambrosia beetles. They listed flooding, drought, mechanical damage, freezing and ozone exposure as stresses that induce the release of stress-related volatiles by plants that would attract these insects. They emphasized that plants that appeared “healthy” could actually be

physiologically stressed. To their list one might add disease as a factor that could stress trees and make them more susceptible to attack by these insects.

Nurseries, in which high densities of a single or limited number of host species are produced, are another setting in which ambrosia beetle-associated tree diseases have developed. *Xylosandrus germanus* (Scolytinae: Xyleborini) (Fig. 2A) and *F. lateritium* were recovered from cankers on black walnut in the Midwestern USA (86). After artificial inoculation, the fungus caused cankers on this tree that were similar to those that were associated with *X. germanus* activity. Kessler (86) noted that this introduced (Asian) beetle was also associated with *Fusarium*-induced damage on *Quercus rubrum* (sic) in Germany. In Ohio, Dochinger and Seliskar (39) described a canker disease of tulip polar, which they demonstrated was caused by *F. solani*. Anderson and Hoffard (7) later reported a similar disease on this tree in a progeny trial in Ohio. Cankers were always associated with *X. germanus* activity and what they reported to be *F. solani*; cankers were not found on *X. germanus*–free trees. Although Anderson and Hoffard (7) did not isolate the fungus or conduct pathogenicity work, they suggested that an ambrosial symbiont of *X. germanus* was responsible for this disease.

In none of the above cases was the pre-existing health of trees examined. Given the wide range of factors that influence ambrosia beetle attraction to trees (136) and the unusual development of these diseases in nursery settings (the same diseases often did not develop in natural settings), the affected trees may have been predisposed to beetle attack. Although the extent to which predisposition also impacted the development of symbiont-associated disease in these situations is unclear, similar factors are well-known contributors to the development and severity of plant disease (141). Currently, there is a very poor understanding of the abiotic and biotic stresses that influence the outcomes of these interactions.

Summary

Recently, there has been an alarming increase in ambrosia and bark beetle-associated diseases of trees (73). These diseases have developed in forest ecosystems, urban landscapes and agricultural settings, under varied environments, and in geographically diverse locations (2, 42, 51, 56, 73, 89, 94, 98, 114, 133, 137, 149, 169). The most significant of these diseases have had huge and surprising impacts. Their unpredictability and dramatic increases to importance qualify them as black swan events in tree health (Table 1).

Despite the recent uptick in the appearance of these diseases, they are not new problems. The first pathogen with a demonstrated bark beetle vector was recognized almost a century ago, and the first pathogen associated with ambrosia beetles was detected at least 40 years ago (56, 115). Their increased prevalence has often been associated with activities that facilitate encounters of new or naïve host trees. Anthropogenic expansion of the geographic ranges of the pathogens and their vectors are responsible for most of the disease outbreaks (147), and movement of infested materials via international trade may be most important (59).

Better understandings are needed for how and why these diseases develop. For example, next to nothing is known about how the interactions among insects, fungi and host trees evolve. Why are some of the usually benign symbionts phytopathogens? Are the new encounter phytopathogens also pathogenic in their native ranges and, if so, to what extent do they exert disease pressure in those areas that selects against susceptible portions of resident host populations? And is some degree of virulence necessary for all symbionts to colonize the walls of natal galleries, or are unique attributes found in the phytopathogenic symbionts? Answers to

these questions might facilitate predictions of which symbionts could become problems in new environments and on new host trees.

To date, only general correlations have been made between anthropogenic and environmental factors and the probability that exotic scolytines would invade a given area. Marini et al. (106) determined that the value of imports could be used to predict numbers of exotic Scolytinae, and that warmer and wetter climates in the USA were correlated with their occurence. To develop better predictive capabilities for these problems, identification is needed of additional criteria that are related to the establishment of these insects and the phytopathogenicity of their symbionts.

Ophiostoma novo-ulmi, the *Ceratocystis* mango pathogens, and *R. lauricola* are exceptional among the phytopathogenic symbionts, in that a single inoculation of a susceptible tree species is usually sufficient to cause lethal disease development (2, 51, 133, 149). In most situations in which ambrosia and bark beetles are vectors, the pathogens cause moderate damage and relatively small lesions develop when trees are artificially inoculated (114, 120, 161). In nature, these interactions result in severe damage or tree death only after mass attacks by the vectors. Aggregation pheromones that are produced by bark beetles and the platypodine ambrosia beetles play significant roles in the mass attack phenomenon, and are key factors in the development of some of the diseases that are associated with these insects (88, 116). In contrast, the xyleborine ambrosia beetles do not produce aggregation pheromones, but rely on semiochemicals and other cues to identify host trees (76, 83, 110, 121). In general, much more information is needed on how these insects recognize suitable host trees and initiate development of the various diseases.

One could hypothesize that some of the above vectors would kill or severely damage trees by themselves, especially when their symbionts do not cause extensive damage in artificial

inoculation studies. Testing such a hypothesis might be possible with bark beetles that have non-nutritional phytopathogenic symbionts. However, due to their obligate reliance on, or the great benefit that is provided by, the nutritional symbionts, obtaining ambrosia beetle or bark beetle vectors that are not infested with the latter fungi is typically not possible.

Rather than debating whether an insect or fungus causes more damage in a given situation, it might be more useful to evaluate these insects and fungi as symbiotic units. Management of these diseases is often difficult, since host resistance is usually poor and chemicals that might be used for control (e.g. fungicides and insecticides) are either not available or are cost-prohibitive (126, 132, 133, 149). New ways to manage these problems might be possible with better information on how the various components of these pathosystems interact and result in disease development. In vectors that have been studied intensively, several different symbionts have been recovered from a single species (63, Ploetz et al., unpublished). Thus, with an improved understanding of the factors that result in mycangial colonization by these fungi it might be possible to alter mycangial content to eliminate or reduce the amounts of a phytopathgenic symbiont that is carried by a vector. Likewise, additional knowledge on the factors that are involved in the attraction of these insects to healthy and stressed trees might enable better and more effective means by which their ability to transmit these pathogens to healthy trees could be managed.

These diseases are poorly understood and holistic understandings are lacking for virtually all aspects of the various disease cycles. Nonetheless, as these problems become more commonplace (i.e. as the most serious and surprising problems stop resembling black swan events) significant biological and pathological traits may be revealed that illuminate some of their mysterious aspects. Interdiction via quarantine should play a prominent role in managing

these problems (59). Likewise, once a problem has established in an area an enhanced awareness is needed of the risks that are posed by the movement of infested materials. In this regard, the transport of infested firewood plays a particularly dangerous role (159). For all of these tree diseases, improved detection, prediction and management should be major goals of future research.

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Table 1. Examples of ambrosia and bark beetle-associated damage on trees					
Species	**Host tree(s)**	**Symbiont(s). Host impact**	**Leach's rules?[a]**	**Black swan disease?[b]**	**References**
Ambrosia beetle (Platypodinae: Platypodini)					
Austroplatypus incompertus	*Eucalyptus* spp.	*Ambrosiella* sp. Wood quality degraded in living trees with an unclear involvement of symbiont	No	No	12, 85
Euplatypus parallelus	*Pterocarpus indicus*	*Fusarium oxysporum*. Lethal wilt. Symbiotic relationship with beetle suggested, but Leach's rules not fulfilled	No	No	28
Megaplatypus mutatus	numerous	*Raffaelea santoroi*. Mortality with no known impact of symbiont	n/a	n/a	6, 150, 179
Platypus quercivora	*Quercus crispula*, *Q. serrata*	*Raffaelea quercivora*. Japanese oak wilt. Mortality in susceptible species after mass attack by vector	Yes	Yes	82, 90, 120, 161
Platypus koryoensis	*Quercus mongolica*,	*Raffaelea quercus-mongolicae*. Korean	Yes	Yes	89, 102

	Q. aliena and *Q. serrata*	oak wilt. Mortality in susceptible species after mass attack by vector			
Platypus cylindrus	*Quercus suber*	*Ophiostoma quercum* and other Ophiostomatales recovered from beetle, but Koch's postulates and Leach's rules not completed	No	No	18
Platypus apicalis, *Platypus gracilis*, *Treptoplatypus caviceps* (formerly *Platypus caviceps*)	*Nothofagus menziesii*, *N. solandri* var. *cliffortioides*, *N. fusca*, *N. truncata* et al.	*Sporothrix nothofagi*. Mortality and/or reduced wood quality caused by symbiont	Yes	Unclear early history	46, 53, 115, 175
Ambrosia beetle (Scolytinae: Xyleborini)					
Corthylus columbianus	Numerous angiosperms in eastern USA. Prefers vigorous living hosts.	No apparent role for symbiont or other fungal associates. Causes significant economic degrade of timber.	n/a	n/a	12, 79, 99, 122

Corthylus punctatissimus	Diverse angiosperms in eastern North America	No apparent role for symbiont. Lethal to young *Acer saccharum*, which is girdled at root collar	n/a	n/a	12, 48, 99
Corthylus sp.	Alnus plantations in Colombia	Pathogenicity documented for associated fungi, *Fusarium solani* and *Ceratocystis* sp.	No	No	180
Euwallacea fornicatus	*Camellia sinensis* and other angiosperms	*Fusarium ambrosium*	Yes	Unclear history	9, 25, 143
"*Euwallacea fornicatus*"	*Persea americana* and other angiosperms	*Fusarium* sp. causes branch dieback and is disseminated by what may be a distinct relative of *E. fornicatus* in California and Israel	Yes	Yes	42, 114
Xyleborus glabratus	*Persea americana*, *P. borbonia*, *P. humilis*, *P. palustris* et al.	*Raffaelea lauricola*. Laurel wilt. Lethal development in susceptible species after a single inoculation	Yes	Yes	51, 65, 133
Xylosandrus	*Juglans nigra*,	*Fusarium lateritium*, *F. solani* Cankers	No	No	4, 7, 39, 86

germanus	*Liriodendron tulipifera*, *Quercus rubra*	and severe damage on trees in nurseries but Koch's postulates and Leach's rules not completed.			
Bark beetle (Hylesininae: Tomicini)					
Dendroctonus frontalis	*Pinus* spp.	*Entomocorticium* sp. A and *Ceratocystiopsis ranaculosa* are not pathogens. Beetle causes significant damage on its own, and is repelled by phytopathogenic symbiont, *Ophiostoma minus*.	n/a	n/a	16, 69
Dendroctonus ponderosae	*Pinus* spp.	*Grosmannia clavigera*	Yes	No, at least on all but recent hosts	150, 160
Dendroctonus valens	*Pinus* spp.	*Leptographium procerum*	Yes	Unclear magnitude in China	103, 104

Bark beetle (Scolytinae: Corthylini)					
Pityophthorus juglandis	*Juglans* spp. (*J. nigra* most susceptible)	*Geosmithia morbida* causes thousand cankers disease	Yes	Yes	94, 158
Bark beetle (Scolytinae: Cryphalini)					
Hypocryphalus mangiferae	*Mangifera indica*	*Ceratocystis manginecans* causes sudden wilt of mango in the Middle East	Yes	Yes	2, 3, 164
Hypocryphalus mangiferae	*Mangifera indica*	*Ceratocystis mangicola* and *C. mangivora* cause seca of mango in Brazil	Yes	Yes	66, 137, 165
Bark beetle (Scolytinae: Scolytini)					
Scolytus scolytus, *S. kirschi*, *S. laevis*, *S. multistriatus*, *Hylurgopinus rufipes* et al.	*Ulmus americana*, *U. thomasii*, *U. rubra, U. minor*, *U. procera*, *U. glabra*, et al.	*Ophiostoma ulmi*, *O. novo-ulmi* subsp. *americana* and *O. novo-ulmi* subsp. *novo-ulmi.*	Yes	Yes	22-24, 56, 168, 169

[a] Leach (101) published four rules to determine whether an insect was the vector of a plant pathogen. He indicated that: 1) the insect must be closely associated with diseased plants; 2) the insect must make regular visits to healthy plants; 3) the insect must be

associated with the pathogen/causal agent; and 4) visitation of healthy plants by pathogen-infested insects results in the development of the disease.

[b] Black swan diseases possess the following criteria: 1) they have high impacts on a given host species, 2) lie outside the realm of regular expectations (they are rare); and 3) are unpredictable (155). Only in hindsight are these events appreciated and understood.

Figures

Figure 1. Gallery of an ambrosia beetle, *Euwallacea* sp., in an avocado tree affected by Fusarium dieback in Israel. Note eggs and larvae of the insect, and mycelium of the causal agent, *Fusarium euwallaceae*, on which the larvae are feeding.

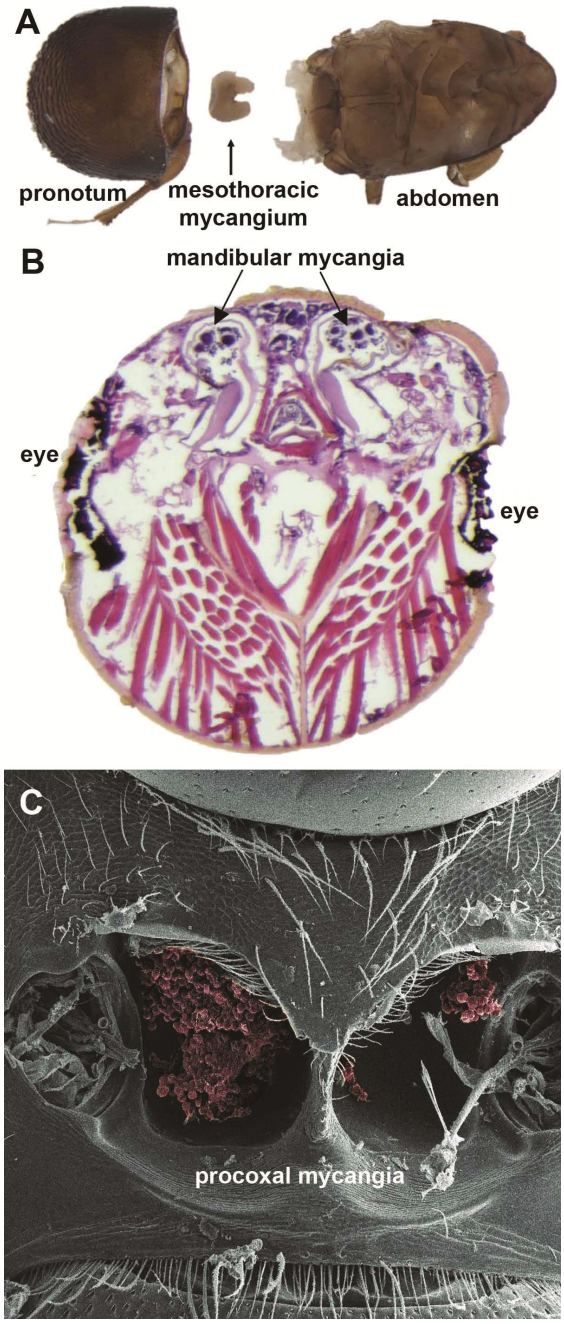

Figure 2. Mycangia are found in diverse locations in ambrosia beetles: A) mesothoracic mycangium, a pair of fused pouches extracted from between the prothoracic and mesothoracic nota in *Xylosandrus germanus* (Scolytinae: Xyleborini); B) paired mandibular mycangia in cross section of a female head of *Xyleborus affinis* (Scolytinae: Xyleborini); and C) paired procoxal mycangia (deep pits) in *Monarthrum* (Scolytinae: Scolytini: Corthylina). The dense contents of the mycangia in B and C are spores of the fungal symbionts of these insects.

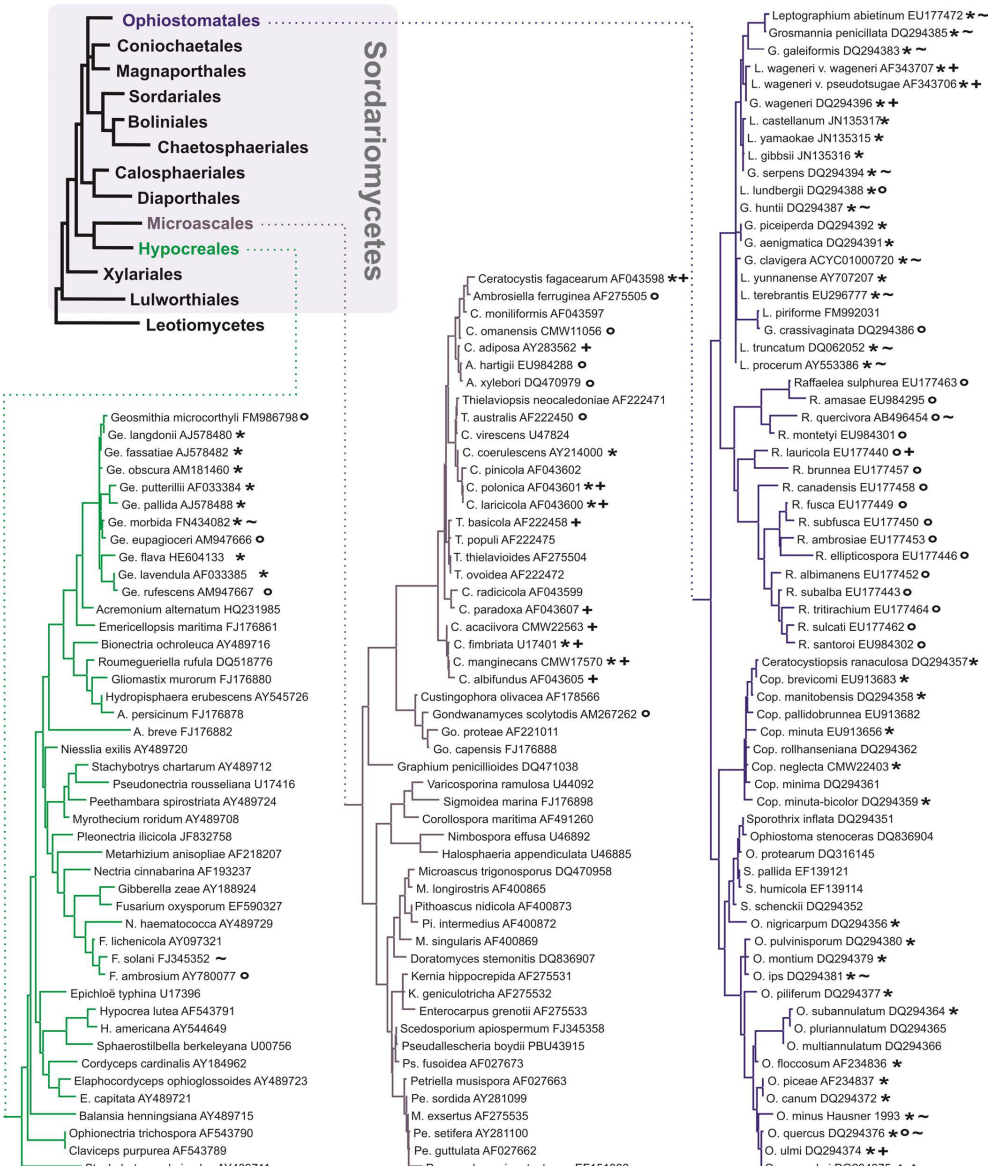

Figure 3. Phylogeny of fungal symbionts of ambrosia and bark beetles, based on maximum likelihood analysis of ribosomal large subunit sequences [conducted online with PhyML 3.0 (http://www.atgc-montpellier.fr/phyml/) and the GTR+I+G model]. Placement of the Ophiostomatales, Microascales and Hypocreales in the Sordariomycetes is indicated in the upper left. Bark (*) and ambrosia beetle (o) symbionts, and whether the phytopathogenic fungi can kill trees on their own (+) or are less virulent (~), are indicated in the phylogenies. The data matrix consisted of 214 taxa and 771 unambiguously aligned nucleotides. Species names are followed by their respective GenBank accession numbers.

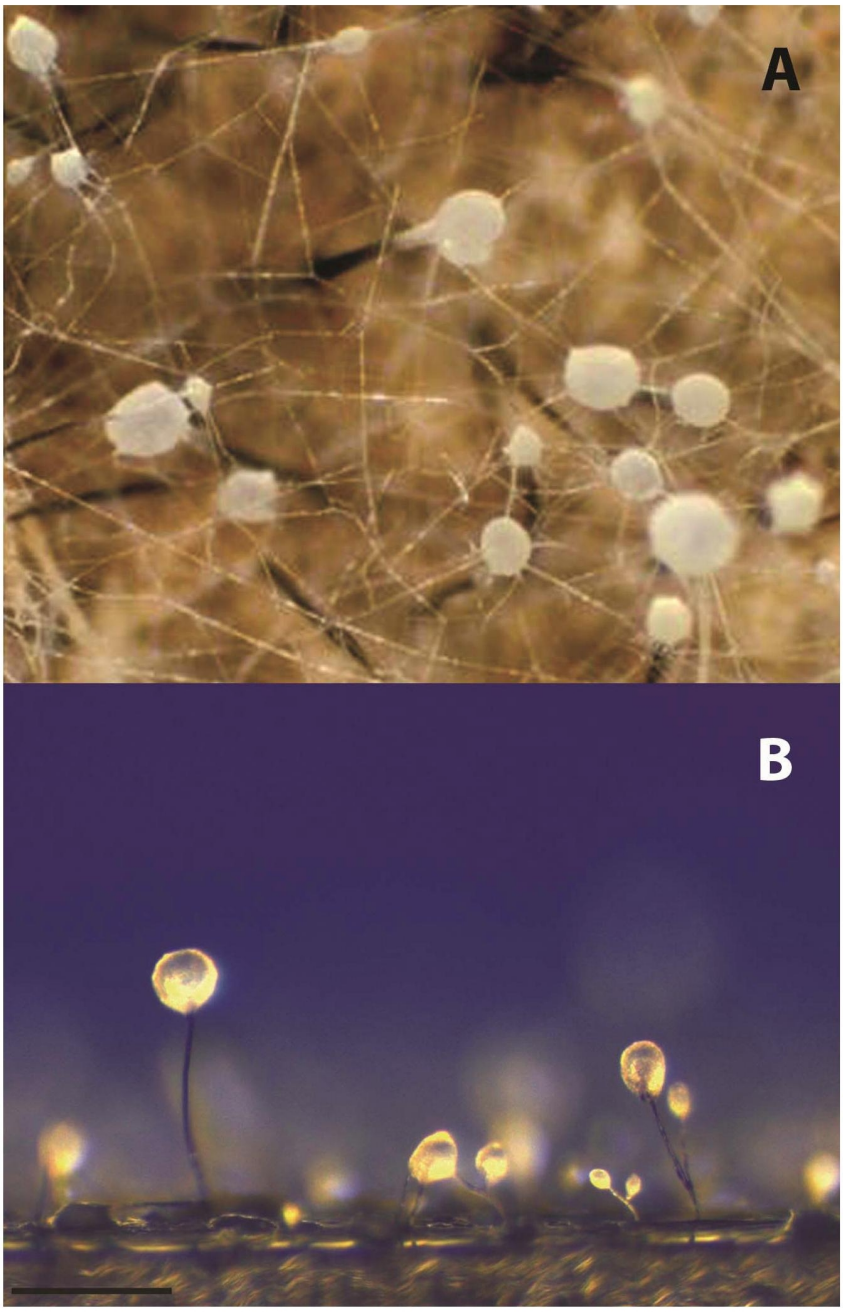

Figure 4. The fungal symbionts of ambrosia and bark beetles often produce sticky spores that facilitate their acquisition and dissemination by their vectors. In A, the *Graphium* asexual stage of *Ophiostoma ulmi* has produced white masses of conidia at the end of dark synnemata. A culture of *Leptographium procerum*, on which stipes and their associated conidial droplets are evident, is shown in B.

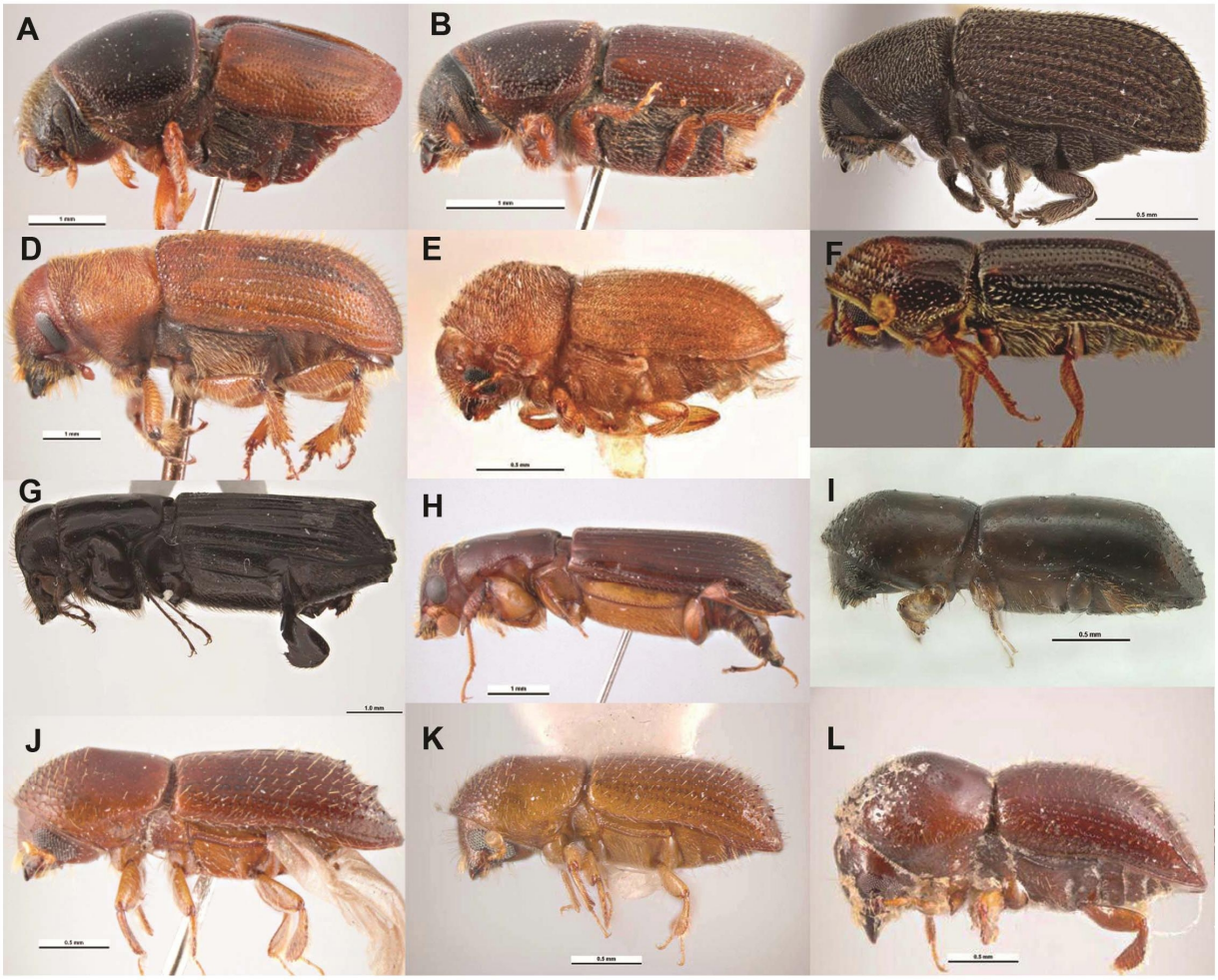

Figure 5. Insects that are discussed in the text include bark beetles: A, *Scolytus scolytus* (Scolytinae: Scolytini); B, *S. multistriatus*; C, *Hylurgopinus rufipes* (Scolytinae: Hylesinini); D, *Dendroctonus valens* (Hylesininae: Tomicini) E, *Hypocryphalus mangiferae* (Scolytinae: Cryphalini); F, *Pityophthorus juglandis* (Scolytinae: Corthylini); and ambrosia beetles: G, *Megaplatypus mutatus* (Platypodinae: Platypodini); H, *Platypus apicalis* (Platypodinae: Platypodini); I, *Xyleborus glabratus* (Scolytinae: Xyleborini); J, *X. ferrugineus*; K, *X. volvulus*; and L, *Euwallacea fornicatus* (Scolytinae: Xyleborini). The scale for A, B, D, G and H is 1 mm, and for C, E, I, J, K and L is 0.5 mm; F was without a scale.

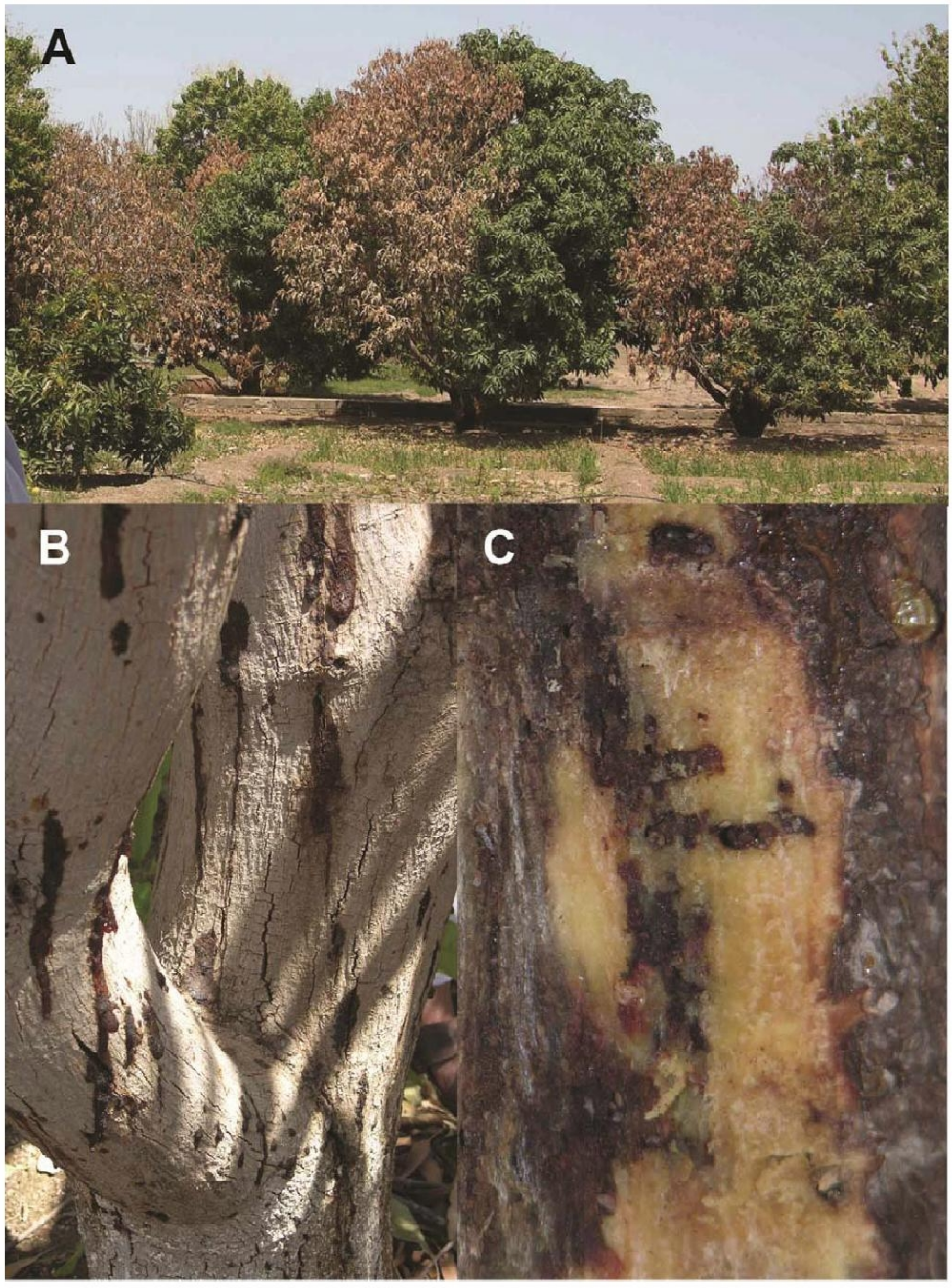

Figure 6. Symptoms of sudden wilt of mango (*Mangifera indica*) in Oman, caused by *Ceratocystis manginecans*: A, death of all or portions of trees; B, external evidence for boring activity of the bark beetle vector of *C. manginecans*, *Hypocryphalus mangiferae*; and C, necrosis of the cambium (left) in which galleries of *H. mangiferae* are evident (center).

Figure 7. Lesions in the cambium of *Acacia mangium* resulting from systemic infection by *Ceratocystis acaciivora*, a close relative of *C. manginecans* in Southeast Asia. Initial infections are closely associated with infestation by scolytine beetles, although the nature of this association has not been established.

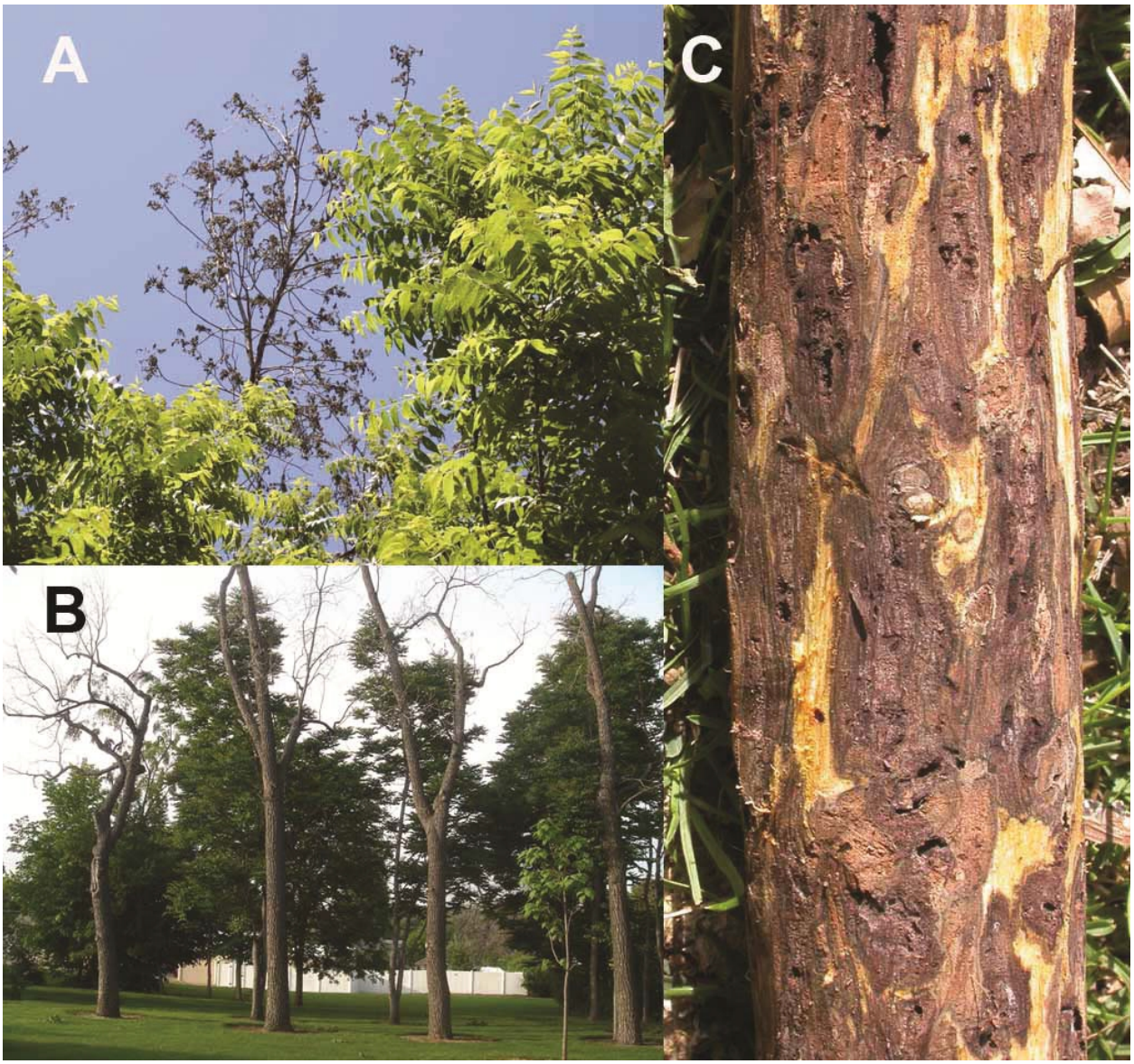

Figure 8. External symptoms of the thousand cankers disease of black walnut (*Juglans nigra*) develop initially as: A, dead terminals and progresses to B, death of the entire tree. Beneath the bark of affected trees, C, numerous cankers develop and eventually coalesce to produce large necrotic patches. Mass attack by *Pityophthorus juglandis*, the vector of the thousand canker pathogen, *Geosmithia morbida*, was needed for the serious damage in B and C to develop.

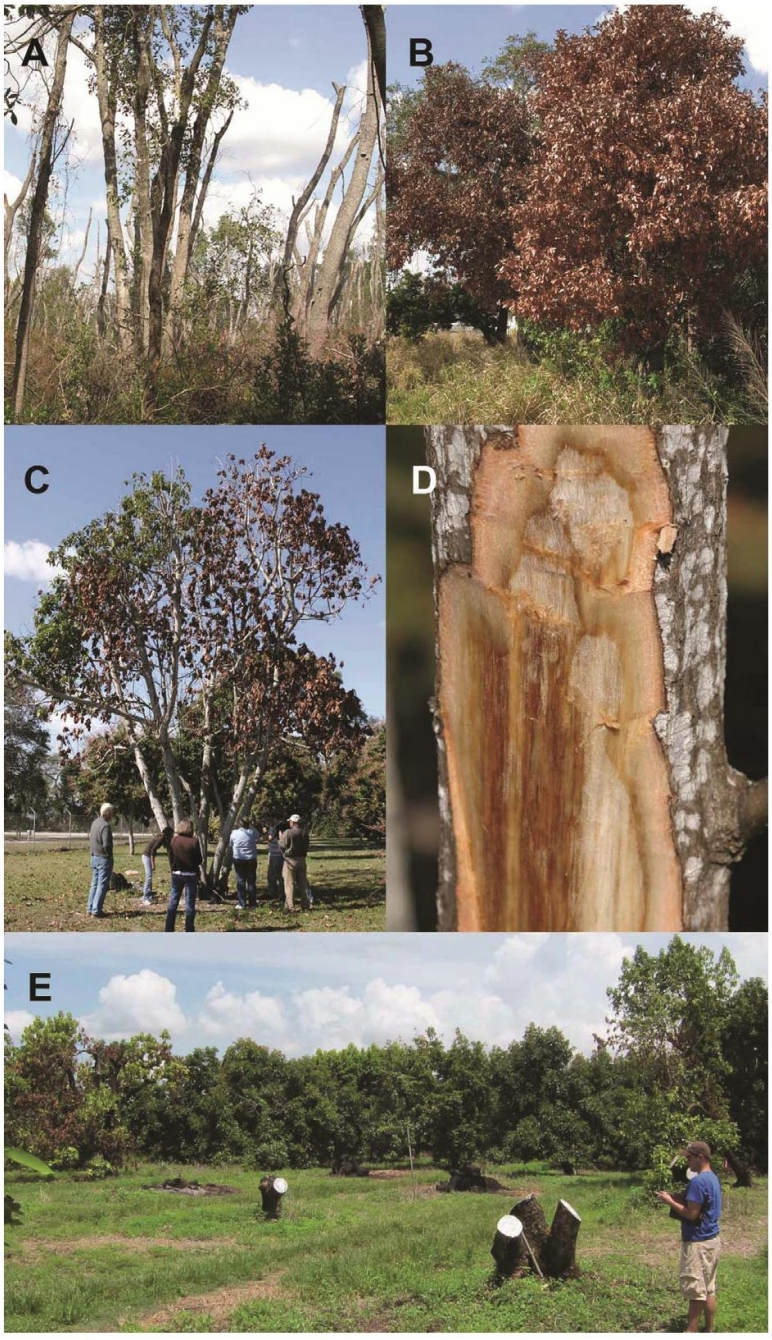

Figure 9. Destruction of redbay (*Persea borbonia*) by laurel wilt in, A, northeastern Georgia. Redbay is killed so rapidly that leaf abcission layers do not form and affected trees retain leaves for a year or longer, as in B, whereas avocado (*P. americana*) defoliates within weeks, as in C. Discoloration of sapwood occurs in all hosts, as on avocado, D. Focal development of laurel wilt occurs in commercial stands of avocado in South Florida, E, due to spread of the pathogen, *Raffaelea lauricola*, via root grafts.

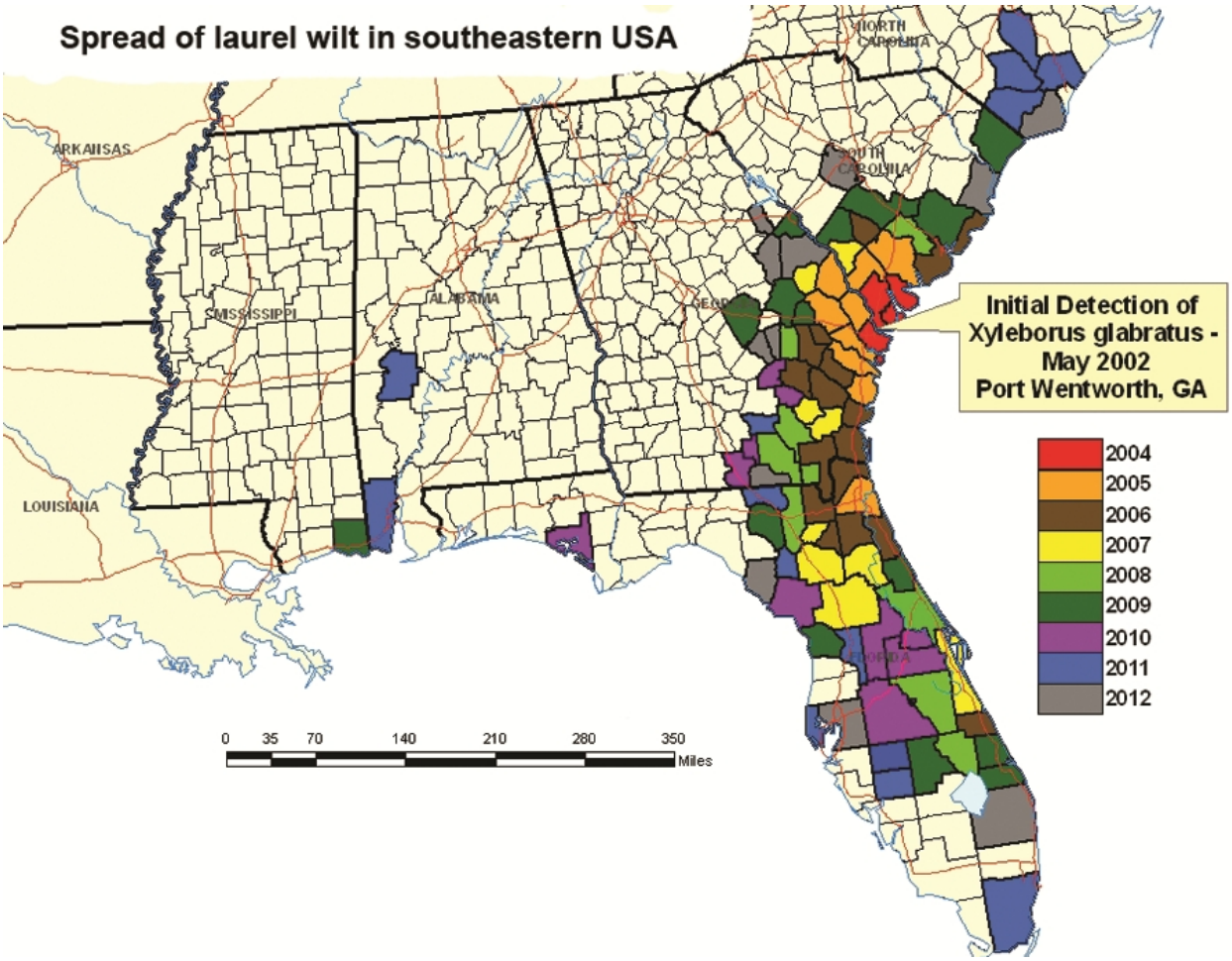

Figure 10. Annual spread of laurel wilt in the southeastern USA. The year in which a county had its first confirmed case of the disease is color coded as indicated in the legend.

Figure 11. *Euwallacea* spp. (Scolytinae: Xyleborini) and *Fusarium* spp. are associated with diseases of several tree species. Boring of one of the *Euwallacea* spp., the polyphagous shothole borer, results in the production of conspicuous “sugar volcanos” of the seven-carbon sugar, perseitol, on avocado, and in the inset necrotic patches that were caused by the fungus are evident.

Author biosketches.

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Michael M. Wingfield completed his early education in South Africa and obtained a Ph.D. in Plant Pathology from the University of Minnesota in 1983. His primary research interests concern the biology and control of tree pests and pathogens, with a strong global perspective. He established the Tree Protection Co-operative Programme (TPCP) in 1990 to minimise the impact of pests and pathogens that threaten commercial forestry in South Africa. The TPCP was the catalyst for the establishment in 1998 of the Forestry and Agricultural Biotechnology Institute (FABI; www.fabinet.up.ac.za) at the University of Pretoria, of which he is the founding director. Mike has served in many distinguished positions and has received numerous awards and honors for contributions to education, research and industry, in South Africa and elsewhere in the world. He is a fellow of scientific societies including the Royal Society of South Africa, Academy of Sciences of South Africa, the Southern African Society for Plant Pathology, and the American Phytopathological Society, is one of the few honorary members of the Mycological Society of America.