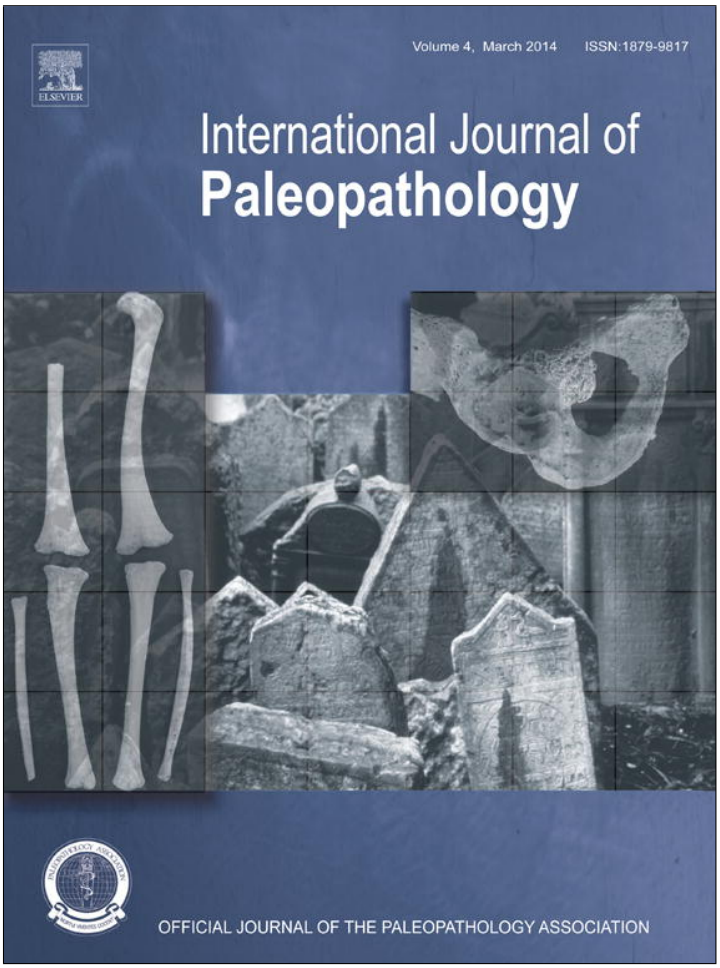

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Invited Commentary

Plant paleopathology and the roles of pathogens and insects

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ABSTRACT

Plant pathologies are the consequence of physical and chemical responses by plants to invasive microorganisms or to imbalances in nutritional or environmental conditions. Many factors determine the potential for plant disease infection and disease, but the primary components are the terrestrial host plant, the pathogen, the environment, and occasionally a biological vector. Pathogens typically are one of four major causative groups: viruses, bacteria, fungi, and nematodes. The vector often is a passive abiotic agent such as wind, water or soil, but it also may be an insect that actively facilitates transmission of the pathogen to a plant. Pathogenic invasion of plants may require sophisticated structures for penetration of host tissues and can elicit a range of host responses such as production of defensive compounds, callus tissue, galls and necroses to seal wounded or infected areas. Fossil diseases primarily are diagnosed from surface leaf structures internal tissues, categorized into damage types (DTs), important for tracking the evolution of herbivore and pathogen attack and host-plant response in time, space and habitat. The fossil record is a useful, underappreciated, but accessible archive of plant damage. We present an overview of pathogens and life cycles that involve insects in the production of these disease symptoms in fossil plants.

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1. Introduction

If one mentions the term paleopathology among the biological or geological community, commonly the first thought that registers is the archeological record of human diseases reflected in various skeletal abnormalities (Ortner, 2011). The vertebrate fossil record also comes to mind, consisting of atypical, histological malformations that result from disease, growth anomalies, predation, or accidents incurred during life. Rarely considered is the broader compass of associations among other fossil organisms. Among these other associations are data from plant fossils that include insect herbivory from the compression–impression record which is becoming increasingly robust (Labandeira and Currano, 2013), as well as the more modest yet significant records of invertebrate animal–animal interactions. Examples of such relationships include the altered tissues of insect hosts by their nematode parasitoids (Poinar, 2012), and the healed wounds surrounding unsuccessful drill holes in the shells of marine mollusks by gastropod predators (Kelley and Hansen, 2003).

These paleopathological relationships occur in compression-impression or permineralized deposits. Compression fossils are the flattened, carbonized remains of organisms found in rock matrix. Related impression fossils are the remains of weathered compression fossils, essentially comprising a flattened mold of the original organism without any organic material remaining. By contrast, permineralized fossils are three-dimensionally preserved organisms typically in a silica or carbonate matrix, that result from mineralizing fluids perfusing the organic material at a cellular level.

Our application of the term "plant pathology," used here in reference to the fossil record, is therefore more eclectic than generally applied to the study of modern plant diseases. In extant plant pathology, biological damage to plants inflicted through the activity of insect herbivores is excluded. However, in the same way that paleopathological studies of fossilized vertebrates explore the origins of all healed wounds incurred during the life of the organism, we also include injuries acquired through herbivory within our concept of plant paleopathology. This is an essential modification in comparison to modern studies of plant pathology, as it is often impossible to distinguish in fossilized specimens between the symptoms of primary plant pathogens and organisms entering the host through wounds created during insect herbivory.

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Table 1
Insect-vectored pathogens likely to leave a fossil record on plant tissues.

Pathogen system	Common name	Pathogen	Insect vector	Functional feeding group	Plant host	Organ(s) affected	Tissue(s) affected	Damage types[a]	Figure	References
Virus	Barley Yellow Dwarf	*Luteovirus* (barley yellow dwarf virus)	*Rhopalosiphum* spp. (Hemiptera: Aphididae)	Piercer-&-sucker	*Hordeum vulgare* and other cereals	Seed, stem and leaf	Endosperm, phloem	DT46, DT48, DT136	Fig. 1	Burnett (1989), Martin et al. (1990), Gray et al. (1993) and Agrios (1997)
Bacterium	Curcubit Bacterial Wilt	*Erwinia tracheiphila* (curcubit bacterial wilt virus)	*Acalymma vittata*, *Diabrotica undecimpunctata* – Coleoptera: Chrysomelidae	External foliage feeder	*Cucumis sativus* (cucumber) and other Curcubitaceae	Leaf, fruit	Xylem, phloem (vessels)	DT03, DT04, DT05, DT50, DT126	Fig. 2	Watterson et al. (1972), Goodman and White (1981) and Agrios (1997)
Fungus	Dutch Elm Disease	*Ophiostoma ulmi* (an ascomycete)	*Scolytus multistriatus*, *Hylurgopinus rufipes* – Coleoptera Scolytidae	Wood borer	*Ulmus americana* (American elm) & other *Ulmus* spp.	Stem (trunk), leaves as an effect	Xylem (vessels, wood), bark, fibers, tracheids	DT160, DT174, DT123, undescribed borings	Fig. 3	Pomerleau (1970), Brasier (1991), Smalley and Guries (1993) and Agrios (1997)
Nematode	Red Ring Disease	*Bursaphelenchus cocophilus* (an aphelenchoidid)	*Rhynchophorous palmarum* – Coleoptera, Curculionidae	Pith borer	*Cocos nucifera*, *Elaeis guineensis*, *Phoenix dactilifera* (coconut, date and oil palms)	Stem, foliage	Ground parenchyma, foliar mesophyll	DT160, DT174, DT221, undescribed borings	Fig. 4	Griffith (1987), Giblin-Davis (1990) and Agrios (1997)

[a] See Labandeira et al. (2007).

In this contribution, we therefore offer a more expansive and inclusive definition of paleopathology that extends beyond that of the vertebrate fossil record. Our intention is to recognize the types of pathogens and the effects that their life cycles have, in association with herbivorous insects, for the production of disease symptoms in fossil plants. To do this, we necessarily discuss examples of well studied and economically important pathogen life cycles from modern agriculture and forestry. Such an approach forms our concept of paleopathology as a discipline that seeks to understand the deep historical wealth of interactions between plants, their insect herbivores, and colonizing pathogens (Berry, 1923), but nevertheless is informed by modern plant pathology (Agrios, 2005). Plant paleopathology involves the presence of numerous substrates and opportunities for pathogenic invasion of plant tissues resulting from both the feeding activity of herbivorous insects as well as independent ingress of plant-host tissues. We will focus on plant diseases caused by pathogens of internal vascular-plant tissues that appear to have been related to insect feeding activities such as chewing, piercing-and-sucking, mining, galling, seed predation and penetrative oviposition during the past 420 million years (Labandeira, 1997; Labandeira et al., 2007; Prevec et al., 2009).

2. Plant pathology and insects in the fossil record

2.1. What are plant pathologies and pathogens?

A plant disease is a deleterious metabolic imbalance at the cellular level usually caused by microorganisms (Holiday, 1989). Plant pathogens consist of viruses, bacteria inclusive of mollicutes, parasitic green algae, parasitic higher plants, rare protozoans, fungi in the broadest sense, and nematodes (Agrios, 2005). Factors such as excesses or a surfeit of minerals and nutrients, extreme temperatures and humidity, and oxygen availability also cause disease symptoms in plants. Imbalances in these latter, abiotic, factors result in so-called "noninfectious plant diseases," whereas maladies that are induced by biotic factors are termed "infectious plant diseases" (Agrios, 2005).

A pathogen is an organism that causes a disease (Falkow, 1977), and as a parasite, it lives on or in another host organism and exists at the expense of the host. Pathogenicity is the ability of a parasite to interfere with its plant host's basic metabolic processes, and consequently cause disease (Lindgren et al., 1986; Casadevall and Pirofski, 1999; Shapiro-Ilan et al., 2005). Pathogens may be biotrophic parasites, deriving nutrition from living cells using strategies aimed at minimizing or delaying host responses, or they may be necrotrophic parasites, aggressively invading and killing host cells. Obligate (biotrophic) parasites can survive and reproduce only within a living host, whereas many other organisms are facultative, able to survive as either parasites or saprophytes that live on dead organic matter (Reis et al., 1999). The variety of plant hosts that a pathogen can attack may be monospecific, such as a single species, a particular organ or tissue, or oligospecific, a taxonomically less circumscribed group of related species, to broadly eclectic preferences of hosts that lack phylogenetic connection (Tsuchiya, 2004). Additionally, in the case of some fungal pathogens with complex life cycles, such as rust fungi, different life phases of the same species may exhibit specificity on different, alternate plant hosts. Typically there are three key factors that are involved in the development of infectious diseases, and their relationship is sometimes referred to as the "disease triangle" (Francl, 2001). The three sides of the triangle are (1), the plant host; (2), the pathogen; and (3), the environment. The plant host must be sufficiently susceptible both in its genetic predisposition and in its physical context. The pathogen must display acceptable levels of availability and abundance in order for propagules (dispersed

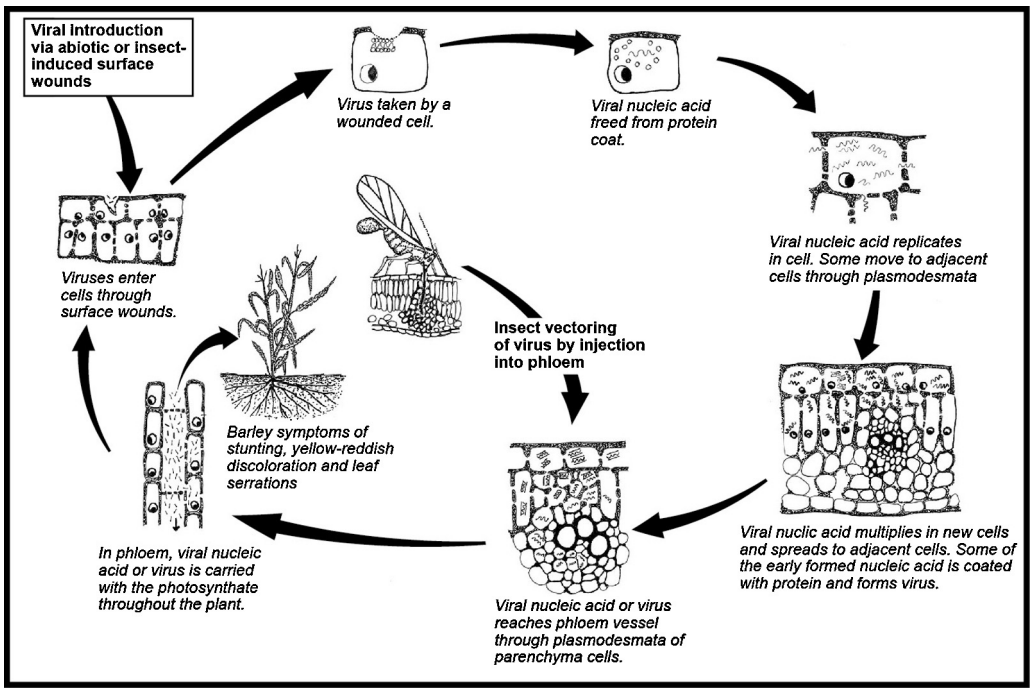

Fig. 1. The life cycle of barley yellow dwarf virus of cereal grains.

After Agrios (1997).

parts of the organism, such as spores) to encounter a suitable host and it must be sufficiently virulent to overcome innate host-plant defenses. The environment, including ambient temperature, humidity and other factors, must be conducive to infection and disease development. However, in the case of insect-transmitted pathogens, this calculus is further convoluted by the relationships of the vector to all three sides of the disease triangle.

2.2. What groups are responsible for plant pathologies?

The principle groups of pathogens that cause plant diseases and other histological abnormalities likely to be preserved in the fossil record are viruses, bacteria, fungi (and traditionally affiliated organisms), and nematodes (Dangl and Jones, 2001). Table 1 lists the major attributes and documentation for examples of these four major groups, whose life cycles involve insect vectors that are further detailed in Figs. 1–4. The role of insect transmission of pathogens is emphasized, particularly as the presence of necroses and other disease-related symptoms are frequently associated with insect damage in the modern world and fossil record. Nevertheless, members of other groups, parasitic green algae (Joubert and Rijkenberg, 1971), flagellate protozoans that include trypanosomes (Dollet, 1984), and parasitic angiosperms such as dodder and mistletoes (Dobbertin and Rigling, 2006) also cause disease symptoms in plants. However, because these groups are rare (e.g., Collinson et al., 1993), they are seldom encountered. These latter groups also cause surface lesions, plant tissue rots, wilts, leaf yellowing, root diseases, twig distortions, and other types of necroses that often are difficult to distinguish from the four major pathogen groups (Forsberg, 1975; Sinclair et al., 1987).

Abiotic causes, such as nutrient deficiencies (Masaka et al., 2008), excessively elevated or depressed ambient temperatures (Schaberg et al., 2011), inadequate oxygen availability (Licausi, 2011), and moisture stress (Board, 2008) can result in symptoms that are similar to pathogenic attack. Two examples from the Eocene of Argentina and Germany display leaf damage that probably is attributable to environmental factors (Fig. 5). Because of this damage pattern, especially necrotic regions along the basal segments of the primary and secondary veins (Fig. 5A), and a band of necrotic tissue encircling the leaf margin ("scorch symptom," Fig. 5B), an environmental cause such as water stress is more likely than pathogenic attack. However, pathogens also can elicit symptoms of water stress in plants by damaging the root system of the host, thereby inhibiting water uptake.

2.2.1. Viruses

A virus is a disease-causing pathogen that consists of a highly organized nucleoprotein that replicates parasitically only within cells of other organisms. Viruses lack the compartmentalization of an intact organism, are incapable of self-sustaining metabolism, and require the DNA replicating machinery of a cellular host for continued existence. Approximately 2300 species of viruses have been formally classified (King et al., 2012), many of which are responsible for a variety of destructive plant diseases. As viruses are incapable of dividing through fission and lack reproductive structures such as spores or cysts, they cause diseases not through production of noxious compounds such as toxins, but rather by incapacitating metabolic processes of their hosts that lead to abnormal cellular or tissue development. Examples of such debilitating consequences for plants include lesions, necroses, chloroses, ringspots, galls, blights, and similar diseases (Smith, 1935; Agrios, 2005).

Phytophagous hemipteran insects disproportionately transmit viruses, particularly as their elongate, stylate mouthparts are involved in piercing and sucking deep-seated plant tissues with the accompanying injection of viruses into plants (Uzest et al., 2007). One common entry point for plant viruses is the xylem and phloem of a host's vasculature, of which Barley Yellow Dwarf Disease is a prominent example (Table 1, Fig. 1). Barley Yellow Dwarf Disease is caused by the barley yellow dwarf virus (BYDV), a *Luteovirus* (Martin et al., 1990), that is transmitted by several aphid species, especially species of *Rhopalosiphum* (Hemiptera: Aphididae). *Rhopalosiphum* is the principle vector for BYDV on barley (*Hordeum vulgare*) and other cereal grasses (Poaceae) such as oats and wheat

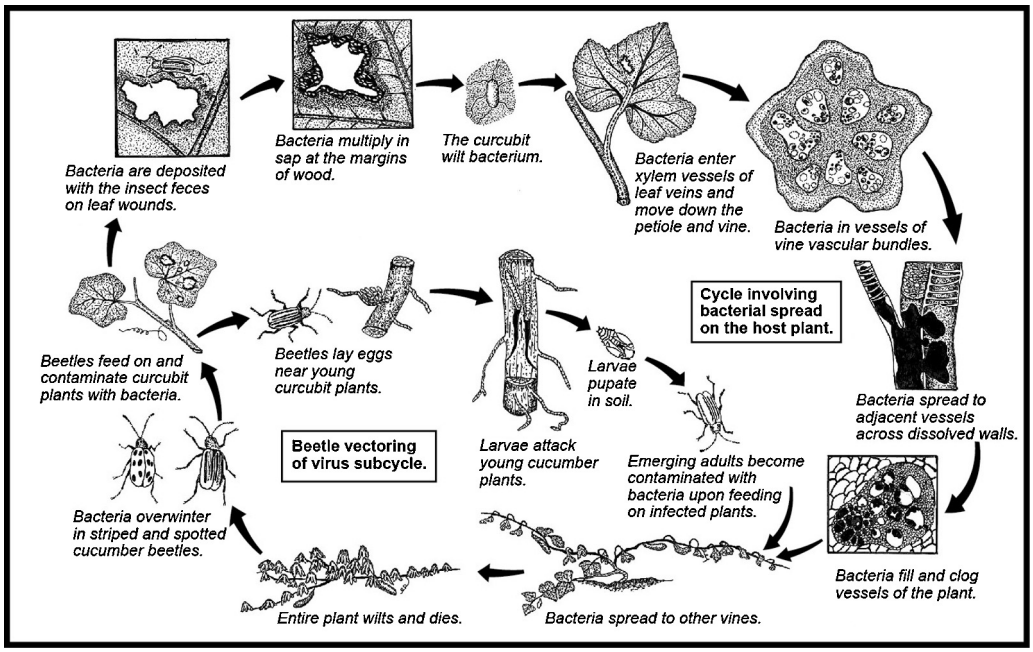

Fig. 2. The life cycle of bacterial wilt of cucurbits.

After Agrios (1997).

(Miller et al., 2002), as well as lawn, pasture and wild grasses. The life cycle of BYDV begins with injection of the pathogen by aphid feeding on barley leaves. The virus moves from epidermal and mesophyll tissues of the leaf into the vascular tissue, eventually invading the nutrient-conducting phloem and largely water-conducting xylem of the stem. The BYDV virus can overwinter in perennial grasses, remaining dormant and becoming virulent during the subsequent spring, at which time additional virally infected aphid feeding can augment viral pathogenicity through the breakdown of structural barriers between the host's cells. In warmer regions, virus-bearing aphids can survive year-round and perpetuate the disease cycle in wild hosts without interruption, providing a key source for new disease when they migrate in spring to cooler areas expunged of aphid populations during cold winter months.

Symptoms of the BYDV virus include plant-host stunting, decreased production of kernels, and incomplete development of kernel endosperm (Burnett, 1989). In addition, leaves undergo distortion, yellowing, and assume other hues of discoloration

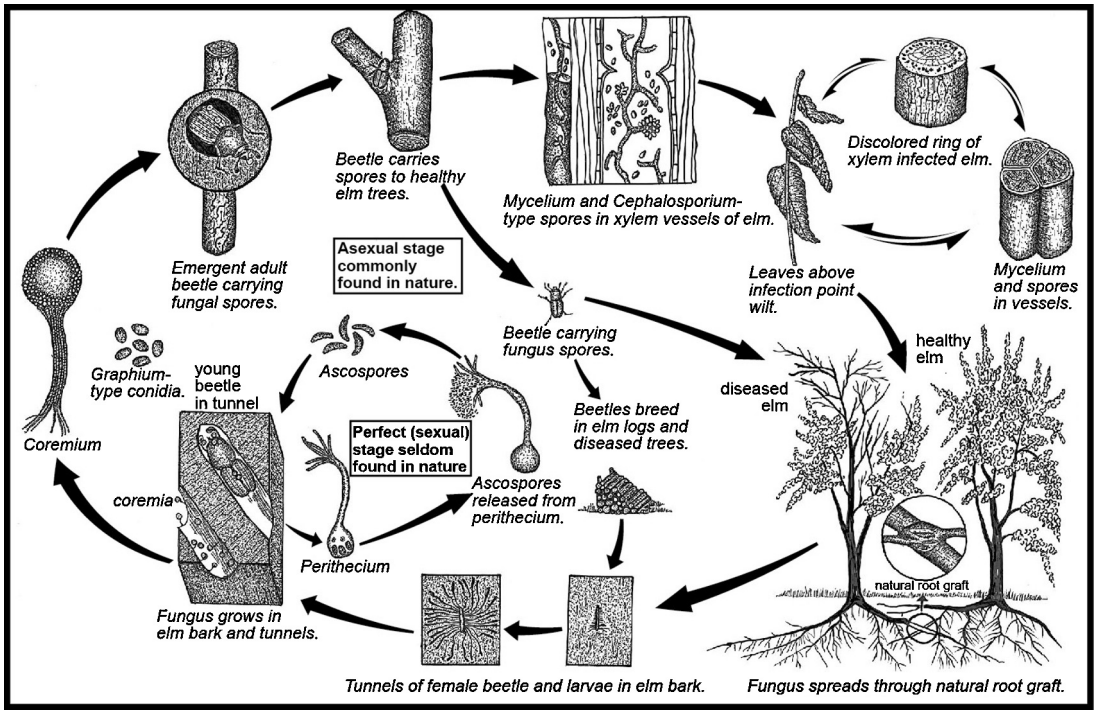

Fig. 3. The life cycle of Dutch elm disease of the American elm.

After Agrios (1997).

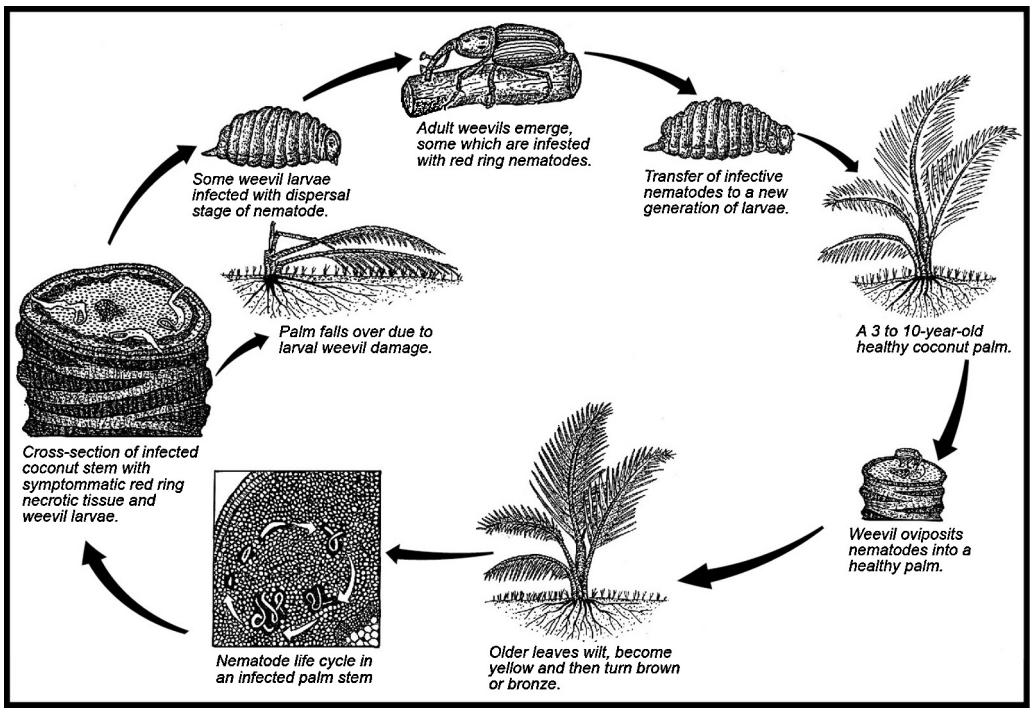

Fig. 4. The life cycle of red ring disease of coconut palm.
After Griffith (1987) and Giblin-Davis (1990).

occurring along leaf margins and tips (Gray et al., 1993). Such systemic foliar damage should be recognizable in the fossil record, as damage types (DTs), such as DT114, or conceivably could be confused with surface feeding that would include DT29, DT30, DT75 and DT97, detailed in version 3 of the *Guide to Insect (and Other) Damage Types on Compressed Plant Fossils* (Labandeira et al., 2007). In the *Guide*, structurally discrete and diagnostic damage types (DTs) are defined and described based on stereotyped patterns of damage caused by insects feeding on plants, such as distinctive modes of external feeding, piercing and sucking, leaf mining and galling. However, such damage could be indistinguishable from the consequences of environmental stress (Fig. 5). Another effect of viral attack is incomplete endosperm development that conceivably could be mistaken for seed predation. Notably, BYDV damage has been recognized in herbarium sheets, providing crucial information for documenting the pathogen's global colonization patterns (Malstrom et al., 2007).

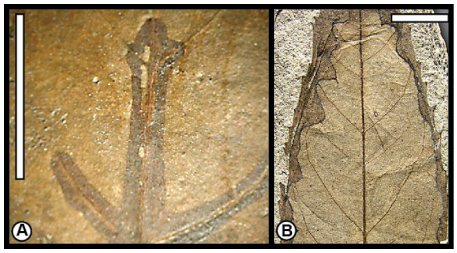

Fig. 5. Two distinctive foliar damage types (DTs) from the early Paleogene illustrate the tricky nature of distinguishing environmental damage from viral or bacterial damage. (A), A leaf specimen, representing DT221, from the middle Eocene of Menat, France, on a species of Platanaceae; the DT is comprised of a rather uniformly wide zone of darkly hued tissue along the midrib and adjacent secondary veins toward the leaf base (Labandeira et al., 2007). This specimen, MNHN 12660, on *Platanus schimperi* (Platanaceae), housed at the Muséum National d'Histoire Naturelle, in Paris. (B), An example of DT114, from the early Eocene of the Palacios de Loros 2 Flora, of Patagonian Argentina; the DT consists of a wide zone along the leaf margin of darkly hued tissue, the inner border of which is controlled by venation and stands in contrast to a much lighter-hued and extensive inner area (Labandeira et al., 2007). This specimen is housed at the Museo Paleontológia Egidio Feruglio, in Trelau, Argentina. Specimen PL2-1234. Scale bars, 10 mm.

2.2.2. Bacteria

Bacteria are prokaryotic organisms that have a cell membrane but lack membrane-bound organelles. Most have a cell wall, the exception being the mollicutes (Bové et al., 2003). Bacteria assume a variety of sizes and shapes, including rods, spheres, dumbbells, filaments and corkscrews (Madigan, 2012), but largely are indistinguishable in their external morphology. In classifying bacteria, features such as the properties of their cell walls (as revealed through Gram-staining), nutritional mode, motility, and more recently gene sequencing are considered. Bacteria reproduce through fission or by production of spores or spore-like bodies. As a result, they have inordinately elevated rates of population increase. Once a host is infected, bacteria can quickly produce disease symptoms. Over 5000 bacterial species have been described (Staley, 2006), but only about 120 species cause diseases in plants. These pathogenic forms are generally saprophytes that can be grown on artificial media (Agrios, 2005), but they also occur as parasites and epiphytically in tissues of their host plants.

A commonplace example of an insect-transmitted proteobacterial pathogen that results in plant damage is *Erwinia tracheiphila* (Enterobacteriaceae), responsible for cucurbit bacterial wilt, a disease affecting cultivated and wild species of cucumbers, squash and other gourds of the Cucurbitaceae (Table 1, Fig. 2). Symptoms include drooping and wilting of leaves on vines, eventually resulting in stem drying and hardening while simultaneously oozing a whitish, viscid, bacterial slime within inner tissues (Saalau-Rojas and Gleason, 2012). The pathogen overwinters in the bodies of 12-spotted and striped cucumber beetles such as *Diabrotica undecimlineata* and *Acalymma vittata* (Coleoptera: Chrysomelidae) that vector the bacterial pathogen through their feeding on cucurbit leaves (Watterson et al., 1972), resulting in the formation of necrotic tissue surrounding the feeding damage. From wounded foliar tissue, the bacteria invade vascular tissue, particularly xylem

vessels, forming deposits that block sap flow and promote leaf wilting (Goodman and White, 1981). The irregular zone of necrotic tissue surrounding margin and hole feeding by beetles on cucurbit leaves should be recognizable in the fossil record on the basis of similarities in feeding and disease pattern. Pathogenic *Erwinia* bacteria may be detectible by scanning electron microscopy that would reveal rods 0.5–1.0 × 1.0–3.0 μm in dimension.

2.2.3. Fungi

Fungi are responsible for the vast majority of plant diseases. There are seven major groups of fungal-like and fungal pathogens. The three fungal-like pathogens are the Myxomycetes or slime molds, the Plasmodiophoromycetes, responsible for root diseases, and the Oömycetes that cause root and stem rots (Agrios, 2005). These groups do not reside in the Kingdom Fungi, but traditionally have been grouped with fungi and historically have been studied by mycologists. The four groups of true fungi consist of the Chytridiomycetes (chytrids); Zygomycetes (zygospore-bearing soft rots); the very diverse Ascomycetes that cause diseases such as blights, vascular wilts and leaf-spot, including the Imperfect Fungi lacking known sexual stages; and the Basidiomycetes (club fungi), of which yeasts and mushrooms are the most familiar (Isaac, 1992). Ascomycete diseases are some of the best known, including the notorious Chestnut Blight of the eastern United States (Anagnostakis, 1987), responsible for the disappearance of many widespread stands of American chestnut (*Castanea dentata*) caused by the fungus *Cryphonectria parasitica* (Pyrenomycetes: Diaporthales), that bears perithecial fruiting bodies.

Of equal importance is Dutch Elm Disease (Smalley and Guries, 1993; Brasier, 1996), caused principally by the pathogen *Ophiostoma novo-ulmi* (Pyrenomycetes: Ophiostomatales), a perithecium-producing ascomycete (Table 1, Fig. 3). This *Ophiostoma* species is transmitted by bark beetles (Coleoptera: Scolytidae), particularly the American elm bark beetle *Hylurgopinus rufipes* and the European elm bark beetle *Scolytus multistriatus*, which feed on trunk tissues of the American elm, *Ulmus americana* (Brasier, 1991). The life cycle of this interaction is complex (Pomerleau, 1970), and begins with a female adult bark beetle landing on an elm tree infected with the fungus *O. novo-ulmi*. The beetles preferentially seek out weakened or dying trees for laying their eggs. A beetle bores through the bark and constructs a capacious central gallery with two opposite rows of small outpocketings, or niches, each of which receives a deposited egg. After the eggs hatch, the larvae form a series of radiating tunnels within the cambial and phloem tissues of the bark, approximately at right angles to the central gallery chamber. It is at this stage of active growth that the larvae acquire abundant asexual sticky spores (conidia) produced by the *O. novo-ulmi* fungus in bark crevasses and other areas adjacent to the larval tunnels. The conidia adhere to the larva's cuticle and some are ingested as the larvae construct an expanding tunnel network.

After pupation, the adults emerge from the host tree and seek healthy trees, where they feed on the bark in crotches of young branches (Webber and Kirby, 1983). They vector spores primarily through the feeding wounds they produce, or later when they bore into the bark of mature parts of the tree to create their egg galleries. Soon thereafter, the fungus reaches the vascular tissues of the inner trunk, and spores are generated asexually through yeast-like budding of mycelia that enter the xylem vessels (Webber and Brasier, 1984). Eventually these spores are carried upstream in the sap flow, and they invade the rest of the tree. The spread of the fungus elicits a host response causing occlusion of xylem vessels through gumming and production of tyloses (Elgersma and Miller, 1977), eventually involving tracheids, fibers and parenchymatic cells surrounding the conductive tissue (Newbanks et al., 1983; Martin et al., 2005). The effects of the diseased vascular tissue rapidly become apparent in the wilting, curling and yellowing of leaves and browning of newly grown twigs of the host's canopy (El-Touil et al., 2005; Newhouse et al., 2007). The presence of two generations of beetles produced per season ensures the efficient spread of the disease. Possible recognition of Dutch Elm Disease in the compression fossil record would be consistent with surface feeding damage of DT30 on twigs and small branches, DT27 and DT75 on foliar material involving marginal necrotization, and gall-mimicking DT123 involving cupping and thickening of foliage that could represent evidence of wilting and damage from desiccation (Labandeira et al., 2007). However, the detection of spore- and fruiting body-producing mycelial networks in trunk tissues undoubtedly would require high-resolution microscopy of permineralized fossil tissues.

2.2.4. Nematodes

Nematodes are a significant cause of disease in plants. After arthropods, nematodes are the second most taxonomically diverse animal clade in terrestrial ecosystems. Approximately 1000 nematode species are known to feed on or otherwise associate with live plants. As plant parasites, nematodes are responsible for significant agricultural losses, attacking roots, stems, foliage, and flowers of a wide variety of ornamental and crop plants, including grains, legumes, fruits, nuts, turf grasses and vegetable and fiber crops. As small, unsegmented worms, nematodes range from 300 to 1000 μm long, and are endowed with a protractible anterior stylet involved in puncturing plant cells, from which protoplasts are consumed. Nematode life histories involve four juvenile stages that are interrupted by molts, with development completed from two to four weeks. In most species, the third and fourth juvenile stages constitute the infective phase that involves obligate feeding on a particular plant host. Soil microhabitats adjacent to plant hosts are crucial for completing the life cycles of plant pathogenic nematodes, where they have a saprophytic existence. Nematodes either are dispersed to potential plant hosts through slow, self-generated movement, or alternatively by external agents such as larger animals, floods, dust storms, or other types of abiotic transport.

One of the better-known insect-transmitted nematode pathogens is Red Ring Disease (Magalhães and Miguens, 2008), caused by *Bursaphelenchus coccophilus* (Table 1, Fig. 4) (Blandón and Viáfara, 2008). The insect vector is the American palm weevil, *Rhynchophorus palmarum* (Coleoptera: Curculionidae), and uncommonly species of *Metamasius*, the sugarcane weevils. These weevils oviposit and feed on wounded ground parenchymatic tissues in the trunks, leaf bases, internodes and petioles of the coconut palm, *Cocos nucifera*, and in the process transmit *B. cocophilus* at any stage of the pathogen's life cycle (Giblin-Davis, 1990). Infestation affects healthy palms, and targets early- to late diseased individuals. After hatching, nematode-infected weevil larvae consume parenchyma, including pre-existing, Red Ring-infected tissue. After the weevil larvae pupate and emerge as adults, typically a few hundred infective, third-instar nematodes are carried by the weevil to infect or re-infect additional palm trees. The iterative process of weevils transmitting pathogens to the plant host is augmented by the co-occurrence of various stages of the nematode life cycle, resulting in a gradual increase in infectivity as individuals develop from eggs to adults.

The principal symptom of Red Ring Disease is a red to orange discoloration of upper stem and petiolar parenchymatic tissues deployed as a cylinder several cm inward from the trunk outer surface (Griffith, 1987). Although neither weevil larvae nor nematodes consume vascular tissues, xylem and phloem conductive tissues can become blocked with gum-like deposits that hinder the flow of nutrients and water to and from canopy foliage. The cessation of vascular tissue transport results in external pathogenic symptoms of wilting, yellowing and eventual browning of leaves closest to infected trunk tissues (Griffith, 1987; Giblin-Davis, 1990). This

is followed by the spread of foliar deformation to the rest of the canopy, usually resulting in tree death. Recognition of nematode mediated diseases in the compression and permineralized fossil record would be consistent with a combination of damage to affected plant parts, such as borings and associated necrotic tissue in stems, overall foliage dieback, and leaf wilting accompanied by anomalous foliar surface features.

2.3. How do pathogens attack plant-organs and tissues and how do plants fight back?

The best context for the pathogenic attack of plant tissues and organs is that of the disease cycle. Within the cycle, the pathogen is dispersed to the host plant typically by wind, water or an insect (Fitt et al., 1989). Given the appropriate conditions, the host is inoculated through viral or bacterial attachment to a host by fungal spores, germinating plant seeds, or hatching nematode eggs (Gramaje and Armengol, 2011). This is followed by penetration of the plant surface by the pathogen either directly or through natural openings such as stomata, lenticels or hydathodes (Zheng et al., 2010), or often by insect feeding damage that produces lesions and consequently exposes internal tissues to the external environment (Stafford et al., 2009).

After successful penetration, by which a pathogen gains entry into a host plant, infection ensues as the pathogen contacts and alters susceptible cells for the procurement of nutrients and other sustaining metabolic compounds. During this phase, the pathogen invades and colonizes a host region. If the pathogen is a virus or a bacterium, a large number of approximately equal-sized disseminules are dispersed as particles to new, uncolonized host tissues. If the pathogen is a fungus, there is an increase in its absolute size through the production of a mycelial mat that grows into the plant host (Bolton et al., 2006). A subsequent phase is the production and dissemination of pathogen propagules through either directed movement, as in fungal hyphal growth, or nematode movement. More common is dissemination through external agencies such as wind or insect transmission, and to a lesser extent by water and human dispersal (Fitt et al., 1989). Once dispersed, the pathogen may overwinter or otherwise survive an inclement interval, typically in an encysted state within the primary or alternate host plant, in a medium such as the soil, or in the bodies of their insect vectors. Eventually the pathogen emerges, producing inoculum that may infect a new host plant to continue the cycle (Montarry et al., 2007). Symptoms of viral and bacterial infection can include foliar wilt, yellowing (chlorosis), browning of major veins, and splotches of necrotic tissue (Bolton et al., 2006).

Most fungi penetrate their hosts through growth of fine hyphae produced by an existing mycelial mass or from a germinating spore. In some pathogenic forms, the termini of the hyphae are modified into a specialized swelling, the appressorium, that develops at a suitable penetration site, in response to thigmotropic, chemotropic or environmental stimuli and that may cover several host epidermal cells and their cell-wall boundaries (Kolattukudy et al., 1995; Mendgen et al., 1996). Appressoria have been shown to generate enormous turgor pressure, and this force acting on a narrow penetration peg, in conjunction with the action of enzymes secreted by the appressorium at the site of penetration, can effect direct penetration through the host cell wall, or indirect penetration through stomata or between adjacent cells (Kolattukudy, 1985; Kolattukudy et al., 1995). Fungi also can penetrate plant hosts through lacerations, punctures, borings or other types of lesions (Wang and Sletten, 1995), particularly those made by insects (Villari et al., 2012), but also wounds made by other pathogens. Other conduits for fungal penetration result from physical processes, such as wind damage that cause breakage or rupture of tissues (Schaberg et al., 2011), or along natural openings, such as stomata and secretory tissue apertures, similar to invasion by viruses or bacteria.

Fungal invasion of internal tissues results in several major responses. At the biochemical level, inhibitors are produced that include a variety of antipathogenic phenolic compounds, phytoalexins, and toxins (Ferreira et al., 2006). One of these biochemical defense mechanisms is a hypersensitive response (Morel and Dangl, 1997), involving rapid localized host cell death in response to pathogen attack, effectively isolating the pathogen from healthy tissues (Agrios, 2005; Hofius et al., 2011). This is one of the most important disease resistance responses in plants, and it may be induced by fungi, bacteria, viruses and nematodes (Lindgren et al., 1986; Bauer et al., 1995). A related response is the production of abscission zones by the host, leading to excision of portions of the plant such as leaf lamina immediately surrounding the site of the infection (leaving a “shot-hole” appearance to the leaf), or to premature defoliation and fruit drop. This strategy removes the disease agent entirely from the plant. Hypersensitive response caused solely by pathogens may in some cases be differentiated from damage following insect herbivory by certain micromorphological features. In the case of plant damage from mandibulate insects, features such as vein stringers and a characteristic pattern of scalloping at the cut edge, indicate stereotypical feeding behaviors and particular mouthpart morphologies (Labandeira, 1997, 1998).

At the histological level, prominent induced structural defenses include the formation of a thickened peridermal response tissue, or callus, forming on the outer layers of bark, the rhytiodome (Mullick, 1973, 1977), Callus also forms in roots or stems with secondary growth tissues, at the point or surface of infection, which seal infected tissues and prevent nourishment of the pathogen (Cleary et al., 2012a). Calluses are deployed as scab-like dead tissue (Tippett et al., 1983), which is uniform in size, shape and texture for particular host-plant–pathogen combinations (Forsberg, 1975; Sinclair et al., 1987). In these or other organs, including leaves and fruits, distinctive fruiting structures, such as pycnia or cleistothecia, are produced toward the center of the necrotic infection zones, and are surrounded by one or more concentric rims of callus associated with hypertrophy or hyperplasy of the adjacent tissues (Biggs, 1984; Isaac, 1992). Similar structures occasionally are encountered in the leaf fossil record (Labandeira et al., 2007; DT49 on p. 13). Other defenses are formation of sheaths surrounding individual hyphae or fascicles of hyphae, and deposition of gums and resins (Cleary et al., 2012b), which have a rich fossil record as amber and copal that frequently incorporate the vector of the fungus (Poinar and Buckley, 2007), but rarely reveal evidence of the fungal disease (Poinar and Poinar, 2005). Profuse production of gums and resins often occurs in woody tissues of conifers and flowering plants as a defense from pathogenic fungi and those wood-boring insects that particularly target cambial tissues as a larval food resource (Villari et al., 2012).

Nematodes penetrate their plant hosts with a special oral stylet (McClure and von Mende, 1987), providing entry into an individual cell or the intercellular spaces of tissues. While nematodes themselves produce disease symptoms in their hosts, they may also vector viral or bacterial diseases that produce, especially in roots, features such as galls (Vovlas et al., 2008), lesions and excessive branching. In above-ground shoots, nematodes are responsible for the yellowing and wilting of foliage (Brown et al., 1995), as well as swelling and shortened internode segments in stems and distortion of buds and leaf tissue. With the probable exception of galls, much of the invasive plant damage caused by nematodes elicit host responses similar to other pathogens, including stem rots, foliar wilts, chloroses, necroses, and especially growth disorders (Viglierchio, 1971).

2.4. What about galls?

Plant galls are productions of atypical tissue that result in hypertrophy (enlarged cells), hyperplasy (excessive proliferation of cells) and other types of malformations induced by the direct developmental intervention of a biological agent (Raman, 2011). Consequently, galls are fundamentally parasitic relationships in which the metabolic machinery of the plant host is developmentally controlled by an organism, usually an immature stage that is lodged within the host plant. Most vascular plant galls are induced by hormones and other substances secreted by insects and mites, although viruses, bacteria, fungi and nematodes also create galls in a wide variety of host plants, and depending on the causative agent, on any part of the plant.

Because of their three-dimensional structure, frequent distinctive morphology, and targeted tissue and organ specificities, galls from viruses, bacteria, fungi and nematodes should have a detectible fossil record. For example, a viral gall, rice gall dwarf phytoreovirus (RGDP), occurs on rice, *Oryza sativa*, and other grass hosts from Eastern Asia (Hibino, 1996). RGDP results in stunting, galling, and surface darkening of foliar tissue that is transmitted by several species of the hemipteran insect vector *Nephrotettix* (Cicadellidae) through the host's phloem tissue (Omura et al., 1980). Similarly, the bacterium *Rhizobium radiobacter* causes crown gall disease in a wide variety of dicotyledonous angiosperms. This pathogen results not only in the production of tumorous growths on leaves, but also the transfer of small segments of DNA from a bacterial plasmid into host cells that are incorporated into the plant's genome (Chilton et al., 1977). Another example is the oömycete fungus *Albugo candida*, which is responsible for white blister rust on plants of the mustard family (Brassicaceae), producing symptomatic fungal pustules, also known as "warts" that occur within green islands of senescing foliage (Isaac, 1992), or galls on stems and petioles under conditions of systemic infection. Perhaps the most distinctive of all nematode galling agents is *Meloidogyne incognita*, which causes a noticeable gall in root tissues of the tomato plant *Lycopersicon esculentum* (Riker and Riker, 1936).

3. Insect-mediated plant pathologies in the compression–impression fossil record

We present several examples from our examination of the deep-time fossil record of plant–insect interactions involving pathogen damage. Our material comes from the compression-impression record of the Cenozoic (early Paleogene), Mesozoic (late Early Cretaceous), and Paleozoic (Late Permian). These specimens (Figs. 5–8) display leaf-damage symptoms consistent with pathogen attack or, less commonly, extreme environmental conditions such as severe temperatures or nutrient deficiencies. Our examples often are associated with particular DTs (Labandeira et al., 2007) that likely provided an entry for pathogens into inner leaf tissues, some of which may have been transmitted by the insect herbivore culprits. Other instances represent passive colonization of leaf surfaces by disseminating agents such as fungal spores or wind-dispersed viral particles. These plant pathogens should be recognizable in much of the compression fossil record, given what is known about their preservation potential in more exceptional deposits (Stubblefield et al., 1984a, 1984b). Potential identification of fossil plant pathogens, however, requires that the following three questions be addressed.

3.1. What signs and symptoms are important in the identification of plant pathogens?

Signs are evidence for the presence of the actual pathogenic agents, such as bacterial ooze, fungal hyphae and spores, whereas symptoms are reactions of a plant host to a pathogen. A common symptom is tissue necrosis occurring adjacent to insect-damaged tissue, which in some cases represents pathogen colonization of open wounds resulting from insect feeding, such as various types of excisions on leaves (Fig. 6A, B, E and F) (Prevec et al., 2009). Other kinds of pathogenic necroses result from independent colonization unrelated to insect feeding, including fungal splotches on leaf surfaces initiated by germination of dispersed spores (Isaac, 1992). Certain distinctive features of these two modes of pathogenic damage can be used coarsely to identify pathogens. For bacterial interactions, symptoms include leaf spots (Fig. 7Q), zones of epidermal puffiness indicative of leaf wilt and curling (Labandeira et al., 2007; DT123, p. 14), and chloroses and other color changes which form distinctive bands of altered tissue paralleling the leaf margin or central primary veins (Figs. 5, 6G, I–K and 7L). Commonly, pathogenic bacteria gain entry into plants via hydathodes (a specialized, water-exuding pore) along leaf margins, and result in lesions and associated necrotic zones that extend from the leaf margin to the midrib. These symptoms also may closely resemble those induced by water stress. Miniscule "fingerprint" patterns can occur on foliage surfaces that are uncannily like modern viral diseases (Fig. 8). Fungal examples include patterns in the migration of reaction tissue fronts across infected leaf laminae (Figs. 6D and 7F, G, M and N), distinctively callused tissue rims at the edges of fungal splotches (Fig. 7O and P), tissue specific foliar galls with distinctively textured surfaces (Fig. 6G and H), and the occurrence of successive generations of diagnostic sexual and asexual spore-generating structures, such as pycnidia and uredia (Dilcher, 1965). With exceptional preservation, signs such as spores, vegetative body fossils (e.g., fungal hyphae), bacterial rods and nematode stylets are another source of evidence for identification.

3.2. What evidence is available to identify plant pathogens?

Even though body fossils of terrestrial viruses, bacteria, fungi and fungi-like organisms, and nematodes are rare, it is the damage that they leave on plants that overwhelmingly will provide evidence for pathogen interactions in the fossil record. Viral diseases tend to show broad but subtle textural alterations of leaf surfaces, emphasizing reaction fronts (e.g., Figs. 7Q and 8), and lack the reproductive structures that would indicate fungal presence. Viruses may produce relatively uniform damage resembling nutrient deficiencies or the effects of extreme microclimate conditions (Fig. 5A and B). Such assignments are based on the relatively constant width of the darkened affected zones, also consistent with phenomena such as nitrogen deficiency or leaf scorch (Masaka et al., 2008). Viral damage typically causes ring spots, mosaic chlorotic patterns, localized distortions of the leaf lamina, leaf roll, generalized stunting, blight, and cankers, frequently inseparable from fungal damage. An exception to this list of effects is splotches of distinct, almost perfectly concentric minute ridges, or ring spots (Smith, 1935), that match damage in some, rare fossil leaves (Fig. 7M and N). However, such damage also could represent reaction fronts from certain epiphyllous fungi (Sinclair et al., 1987). A second example of a typically virally produced pattern of damage is mosaic patterns from chloroses. Other than ring spots and mosaic chloroses, it is difficult to diagnose viral diseases in living plants and particularly for fossil material. Apart from these exceptions, viral damage would be difficult to distinguish from other pathogenic diseases, nutrient deficiencies, somatic mutations and especially nonviral abnormalities of foliar surfaces.

Although bacterial foliar damage is difficult to separate from viral and fungal damage, the best case for bacterially infected tissues consists of wedge-shaped lesions that originate along leaf margins, indicating bacterial ingress along hydathodes. Another

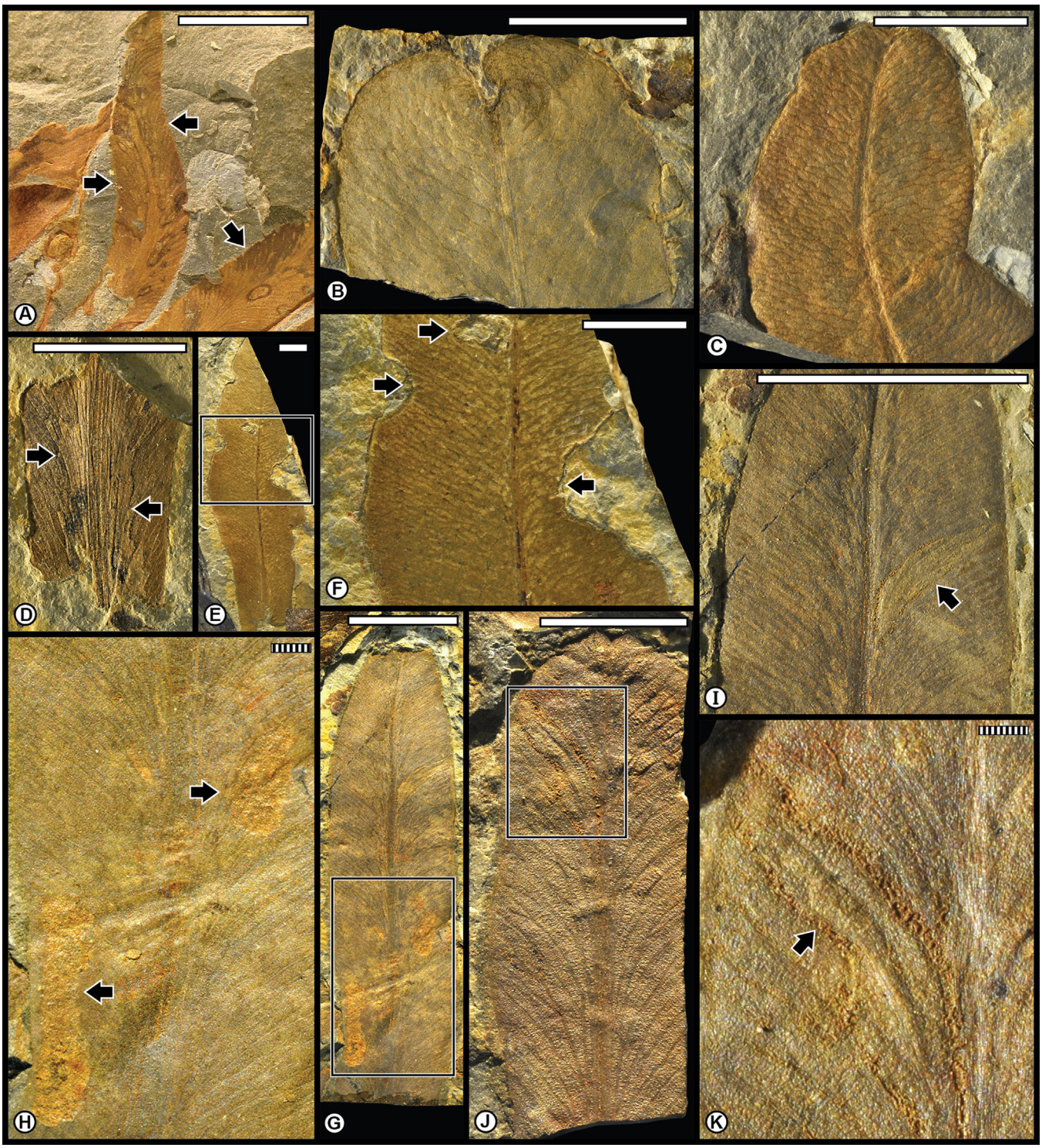

Fig. 6. Evidence for primary colonization of foliar surfaces and secondary pathogenic invasion of tissues resulting from insect herbivory on glossopterid plant hosts from the Late Permian New Wapadsburg Pass locality (A), and Kwa Yaya (B–K) of the Karoo Basin, South Africa. The glossopterid leaves hosting the fungal damage at Kwa Yaya are affiliated overwhelmingly with the K2 series of leaf morphotypes (Labandeira & Prevec, unpublished data; K2 morphotypes are typically associated with *Lidgettonia* fructifications, consisting of small, narrow leaves with fine meshes at a moderate to steep angle to the midrib). (A) Systemic fungal alteration of foliar tissues associated with extensive ovipositional damage (DT136) between the leaf midrib and margin, on two host morphotype W-1a leaves (specimen WN-42-20). (B) Fungal damage affecting surface tissues adjacent to apical foliar damage (DT13) of host morphotype K4a (specimen KY260a). (C) Systemic fungal alteration of surface tissues (hyperplasy) on host morphotype K5a, associated with ovipositional damage (DT101) adjacent to the kink at center-right (specimen KY333). (D) Fungal damage occurring along the leaf margin of morphotype K2, separated from unaffected tissue by an undulatory reaction front (specimen KY250b). (E) Glossopterid leaf morphotype K5a exhibiting margin feeding (DT12) and hole feeding (DT02) with thickened reaction rims at edge excisions associated with possible secondary fungal damage (specimen KY832). (F) Enlargement of external damage area in (E), showing possible fungal damage at arrow. (G) Elongate-ellipsoidal, full-depth galls occurring between the midrib and margin of leaf morphotype K2b, displaying a pockmarked surface texture (specimen KY712b). (H) Enlargement of two galls outlined in the rectangular template at (G). (I) Enlargement of a lenticular shaped area in the apical portion of the leaf in (G), showing inner necrotic tissue and an enveloping callus rim. (J) A necrotic region similar to (I), on host morphotype K2a (specimen KY422a). (K) Magnified portion of specimen in (J), illustrating differences in surface texture between inner area and surrounding unaffected tissue. All specimens are housed in the Albany Museum, Grahamstown, South Africa. Scale bars: solid, 10 mm; striped, 1 mm; dotted, 0.1 mm.

Fig. 7. Examples of fungal and bacterial modification of early angiosperm foliar tissues from the late Early Cretaceous (late Albian) Dakota Formation of Kansas and Nebraska, USA. All specimens are on the lauralean host *Crassidenticulum decurrens* Upchurch and Dilcher (1990), from Braun's Ranch Locality, Cloud Co., Kansas, except for (Q), which is from the Rose Creek locality, Jefferson Co., Nebraska. (A) Fungal damage, consisting of ovoidal splotches with prominent reaction rims and angulate extensions extending medially along the secondary venation. UF4820. (B) Enlargement of splotch at upper-right square in (A). (C) Enlargement of splotch at lower-left square in (A). (D) Another example of fungal damage as in (A). UF12508. (E) Enlargement of splotch at central square in (D). (F) Fungal damage, without medial angulate extensions. UF12508. (G), Enlargement of splotch at upper-left rectangle in (F) showing a circular central fructification. (H) Two examples of fungal damage, each with an angulate extension directed toward the midrib. UF12691. (I) Enlargement of two fungal splotches outlined in the template at (H). (J) Several examples of fungal damage, consisting of circular splotches with undulatory margins and lacking angulate structures, occurring along the leaf margin. UF16228. (K) Enlargement of detail in the rectangle at the lower-left in (J). (L) DT114; see Fig. 5A. UF16130. (M) Probable bacterial damage, showing approximately concentric ridges of callused tissue representing reaction fronts of the advancing pathogen. UF12658. (N), Enlargement of rectangular area at center-left in (M). (O) Fungal damage of a broadly ellipsoidal patch of necroses with a small, centrally-positioned fructification. UF12677. (P) Enlargement of rectangular area at center-top in (O), displaying a central fructification. (Q) Fungal damage covering almost the entire leaf surface, consisting of complexly anastamosing pathogen reaction fronts. UF16143. All specimens are housed at the Florida Museum of Natural History, Gainesville, Florida, USA. Scale bars: solid, 10 mm, striped, 1 mm.

example is "scorch" symptoms, which also could be attributed to water stress. One type of damage uncommonly present in the paleobotanical record involves epidermal puffiness and associated cupping of individual leaves, typical of modern bacterial wilts, leaf spots and blights (Webster et al., 1983), an example of which is DT123 (Labandeira et al., 2007). However, such damage is reminiscent of mite and thrips damage to surface tissues on a variety of modern foliage (Johnson and Lyon, 1991).

It is the fungi, however, that can be most readily identified, attributable to the presence of distinctive reproductive and vegetative features, as well as by the overall size of the damage (Isaac, 1992). Fig. 7 illustrates a variety of fungal damage on the chloranthaceous angiosperm host, *Crassidenticulum decurrens*, from the latest Early Cretaceous Dakota Formation of the midcontinent, U.S.A. Some of these epiphyllous fungi, for example, consist of a matted layer of mycelia on or within the epidermis (Fig. 7A–E, H–K). This distinctive type of leaf surface alteration has been assigned to DT58 (Labandeira et al., 2007). Another probable instance of fungal damage is DT136 (Labandeira et al., 2007), in which there are patches of necrotic tissue that connect multiple insect oviposition scars along the margins of glossopterid leaves (Fig. 6A). Various types of external foliage feeding, such as the leaf-margin excisions of DT12 and DT13, appear to have been infected later by fungi (Fig. 6A, B, E and F), and in some instances the rim of infected tissue is different in texture and hue from that of unaffected blade tissue (Anderson and Anderson, 1985: Pl. 77, Fig. 2a and b).

These damage patterns could be confused with bacterial necroses when reproductive structures are absent. Confirmation of a fungal identity may be provided by the presence of distinctive perithecial, pycnidial, or other spore-producing bodies (Fig. 7F, G, O and P). These structures are usually circular, hardened, and located centrally within the lesion, in the most mature region of the infection. However, fungal spores or spore-containing structures such as asci often are not enclosed by such structures, and definitive attribution to fungal or fungal-like organisms relies solely on spore morphology (Agrios, 2005).

Plant damage and diseases attributable to nematodes are more difficult to ascertain, particularly on foliage. However, nematode mediated damage exhibits a preponderance of marked tissue hypertrophy and hyperplasy, intercellular cavities and galleries in root and stem cortical tissues, macroscopic surface lesions, large root galls and cysts, and radically distorted foliage. Evidence for a nematode vector would rely on multiple and separate signs and symptoms that point to a broader syndrome of infection of stems, foliage and perhaps other organs.

While there is significant overlap of these disease symptoms among the four types of pathogens, particularly for foliar tissues, separation potentially can be made by detection in the fossil record of distinctive damage features on modern stem and root tissues. One aspect of the paleobotanical record that may be helpful in identifying pathogens involves patterns of host specificity. A recurring, conspicuous feature is stereotyped fungal damage with lens-shaped necrotic areas on leaves occurring along secondary veins that extend toward the leaf midrib (Figs. 6G, I–K and 7A–E, H and I; Müller, 1982). By contrast, an encompassing pattern of complex reaction fronts covering broad surfaces of leaves (Fig. 7Q) is present on several unrelated host taxa in the same mid-Cretaceous deposit. These types of fungal damage previously were described in more recent compression-impression deposits from the middle Eocene of the Mississippi Embayment (Dilcher, 1965; Daghlian, 1978), where fungal fruiting structures, probably apothecia, were preserved near the centers of necroses, revealing indurated and thickened fungal mycelia that contrasted to unaffected tissue of the adjacent leaf surface.

3.3. What insect likely was responsible for transport of certain pathogens?

The last step in recognition of plant pathogens in the fossil record is to ascertain whether the dispersal mode was abiotic or facilitated by an animal vector. If there is infection by a pathogen that gains ingress through a wound caused by an insect, it is impossible a priori to say whether that pathogen arrived there with the insect, or if the pathogen was windblown, transmitted by water, or introduced by other abiotic processes. No assumption can be made about whether or not a pathogen entering a site of confirmed insect herbivory was brought there by an insect. Nonetheless, a repeated occurrence of one particular type of insect damage that is associated with a well-circumscribed and stereotyped set of disease symptoms may indicate a connection. It is important that, in lieu of any evidence for an insect vector–pathogen–disease symptom linkage, it is equally possible that there was occurrence of an opportunistic fungus already present on the leaf surface, prior to the arrival of the insect.

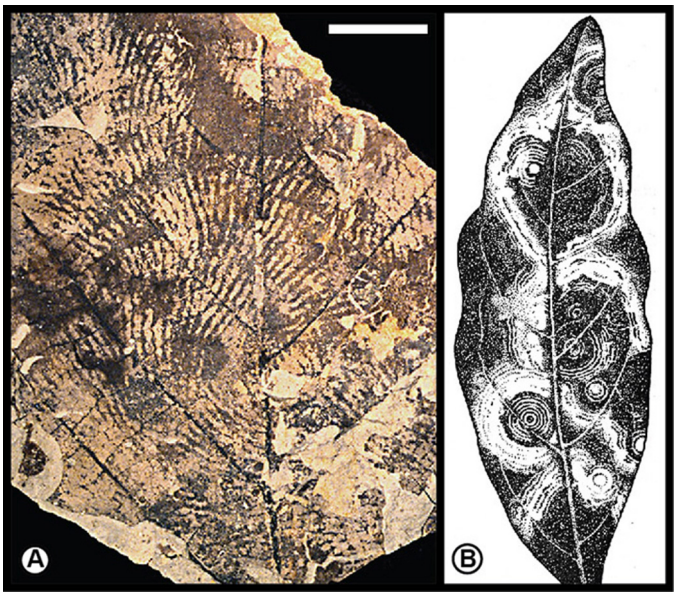

Fig. 8. Modern and possible fossil viral damage on foliage. The fossil insect-mediated damage (DT23, Labandeira et al., 2007), is attributed to skeletonization in (A), but perhaps is better affiliated with viral alteration of surface tissue, of which a modern example is shown in (B). The DT23 on a leaf of *Corylites* sp. (Betulaceae) in (A) was collected from the late Paleocene Fort Union Formation, Washakie Basin, Wyoming. A leaf of *Solanum capsicastrum* (Solanaceae), shown in (B), is infected with tomato spotted wilt virus, exhibiting ". . .concentric rings and line patterns characteristic of this virus on certain hosts" (Smith, 1935, p. 37).

If a direct association can be demonstrated between an insect vector and a known transmitted pathogen with recognizable disease symptoms, then a culprit should be identifiable, though plant-host specificity will vary. An animal vector, particularly an insect or a mite, may reveal some level of stereotyped plant damage, such as a stylet or oviposition insertion scar, or alternatively the edge of a chewed leaf, that would be linked to adjacent pathogen damage(Fitt and Armengol, 2011). A good example is the transmission of the causative agent of Pierce's Disease, *Xylella fastidiosa*, by the glassy-winged sharpshooter, *Homalodisca coagulata* (Hemiptera: Cicadellidae) on cultivated grape, *Vitis vinifera*, which results in a distinctive spectrum of symptoms affecting every organ and most tissues of the host plant (Hopkins and Purcell, 2002). The best identification relies on the close association of the pathogen, plant damage, and animal vector on the same plant-host organ. Identifications also could be made if spatially more distant symptoms occur among two or more host-plant organs of foliage, stems, roots or reproductive organs. Another straightforward identification would be an association of distinctive bark-beetle galleries in the trunk with foliar symptoms consistent with Dutch Elm Disease (Table 1, Fig. 3).

A more difficult case would involve uncharacteristic plant damage of external foliage feeding whose excised or abraded tissues are associated with equally nondiagnostic leaf blotches attributable to viral, bacterial or fungal pathogens. Biological knowledge of recent plant–insect–pathogen associations would be important to

buttress a proposed association. Such corroboration of an insect and secondary fungal infection would constitute an uniformitarian approach to the more recent part of the fossil record and would be less applicable to the deeper past. An example of a modern obligate plant–fungal association applicable to the relatively recent fossil record would be the diagnosis of fungal shot-hole damage occurring on a leaf of *Laurophyllum lanigeroides* (Lauraceae) in the 48-million-year-old Messel deposit of west-central Germany (Schaarschmidt, 1992). This damage is similar in appearance to shot holes on modern laurel caused by multiple bacterial and fungal pathogens which is one of the most striking examples of this disease symptom. Nevertheless, these shot holes could be mistaken for the excised, near-circular leaf cases by the Heliozelidae (Dziurzynski, 1958), a leaf-mining moth lineage commonly known as shield-bearers, or for hole-feeding by other insect groups. This example highlights the challenges faced when ascribing a particular damage type to a specific herbivore or pathogenic agent.

3.4. Can plant diseases be recognized in the preangiospermous fossil record?

With the exception of conifer diseases that are a principal concern of the timber industry (Sinclair et al., 1987; Johnson and Lyon, 1991), angiosperms have been overwhelmingly the focus of modern plant pathology because of their dominance in natural ecosystems and in the agricultural sector (Agrios, 2005; Figs. 1–4). Nevertheless, for plant paleopathologists, approximately the first three-fourths of the macroscopic fossil record of terrestrial plants consists of nonangiospermous vascular plants, principally mosses and liverworts, lycophytes, sphenophytes, ferns and a wealth of gymnosperm seed plants (Krings et al., 2012), lineages of which have a rich record of interactions with insects and mites (Labandeira, 2006; Prevec et al., 2009). Consequently, it is important to document and assess the role of those surviving lineages of nonangiospermous vascular plants in hosting plant diseases, and in particular to identify the disease signs that potentially can be recognized in the fossil record. It is evident that modern nonangiospermous plants are significantly affected by plant diseases, frequently identifiable by a variety of externally evident signs (Sinclair et al., 1987; Johnson and Lyon, 1991). With the exception of certain conifer taxa, most of these lineages are of minimal economic value, although other taxa such as lycopod species of *Huperzia*, bracken, certain cycads, *Ginkgo biloba*, and the gnetalean *Ephedra* have been used extensively as ornamentals in horticulture, as well as in herbal remedies for human ailments and diseases (Wu et al., 1995; Ling et al., 1995; Sati and Joshi, 2011), and have yielded extracts used in agriculture and the pharmaceutical industry (Young et al., 1997; Read et al., 2000; Roy et al., 2000; Saxena and Harinder, 2004; Womack and Burge, 2006).

Cryptogamous plants have been minimally assessed for disease. Mosses and liverworts apparently lack diseases (Saxena and Harinder, 2004). However, some liverworts host fungal glomeralean endophytes (Chambers et al., 1999; Davis et al., 2003) that probably serve mutualistic rather than parasitic roles. Similarly, some modern lycopods host glomeralean endophytes (Read et al., 2000; Winther and Friedman, 2007) that form associations extending deep into the Paleozoic permineralized record (Taylor and Krings, 2005; Krings et al., 2005; Krings et al., 2007; Krings et al., 2011). Like lycopods, horsetails possess few diseases (Freeberg, 1962), except for a variety of maladies that affect silica metabolism (Ellis and Ellis, 1985; Quarles, 1995; Epstein, 1999), and especially bacterioses that are induced by strains of *Pseudomonas syringae* that afflict wheat and secondarily target *Equisetum* (Pasichnik et al., 2012). The considerably more diverse ferns also host diseases (Irvine et al., 2008), but the focus overwhelmingly has been on the diseases of globally distributed and economically significant bracken, *Pteridium aquilinum*, susceptible to several diseases (Cunningham, 1927; Angus, 1858; Nienhaus et al., 1974; San Francisco and Cooper-Driver, 1984; Webb and Lindow, 1987; Irvine et al., 2008). Historically, bracken has been targeted for biological control because of its toxic effects on grazing livestock (Alcock and Braid, 1928; San Francisco and Cooper-Driver, 1984; Womack and Burge, 2006).

The four extant lineages of gymnospermous seed plants – cycads, *G. biloba*, conifers and gnetaleans – have a better documented spectrum of plant diseases than cryptogams. Cycads exhibit signs of several diseases, including necrotic stunt virus on *Cycas revoluta* (Kusunoki, 1986), tobacco ringspot virus on *Zamia furfuracea* (Baker, 2007), leaf-spot and leaf-freckle damage on several species (Tandon and Bilgrami, 1957), as well as other occurrences involving single species (Dodger, 1972; Jian et al., 2006). By contrast, the more intensively examined *G. biloba*, the remaining extant species of a vast ginkgophyte clade dominant during the Mesozoic, possesses numerous known diseases (Aoki, 1997). The diseases of *G. biloba* are overwhelmingly dominated by fungi, including the gray mold of *Botryotinia fuckeliana* (Takano, 1992), pink disease of *Erythricium salmonicolor* (Abe, 1992), thread blight of *Ceratobasidium anceps* (Ito, 1958), canker from various species of *Fusarium* (Takai, 1965), violet root rot from *Helicobasidium mompa* (Kobayashi, 1977), brown leaf-spot disease from *Hendersonia ginkgonis* (Naito, 1952), web blight from *Thanatephorus cucumeris* (Kobayashi, 1977), and other malformations (Young et al., 1997). *G. biloba* also has vesicular arbuscular associations that are mutualistic with fungi (Bonfante-Fasolo and Fontana, 1985).

Like *G. biloba*, conifers bear a considerable number of pathogens that are too numerous to mention here (Gilmour, 1966; Sinclair et al., 1987; Johnson and Lyon, 1991). As an exemplar, we emphasize *Agathis*, a southern hemisphere member of the Araucariaceae that forms a major native component of managed and natural forests of New Zealand, eastern Australia, Papua New Guinea, Malaysia, and adjacent islands in Indonesia and Melanesia. A variety of diseases inflict damage on the leaves and wood of *Agathis* (McKenzie et al., 2002), including die-back caused by *Phytophthora heveae* and *P. cinnamomi* (Podger and Newhook, 1971; Dodger, 1972; Gadgil, 1974), pycnia-bearing rusts caused by *Aecium balansae* and *A. fragiformae* (Punithalingam and Jones, 1971; Hadi et al., 1996), pink disease inflicted by *Corticium salmonicolor* (Suharti, 1983), and abnormal premature leaf abscission of *Hendersonula agathi* leaves (Young, 1948). Lastly, diseases of the Gnetales, the fourth major lineage of gymnospermous plants, are more poorly known than the three preceding gymnosperm groups, with the possible exception of *Ephedra*, also known as joint-fir or Mormon tea. *Ephedra* is particularly attacked by rust fungi, with disease symptoms preferentially expressed on leaves (Khan, 1928; Roy et al., 2000). Many of the aforementioned diseases of modern nonangiospermous vascular plants present symptoms that affect foliage features, fruit development and trunk structure that should be recognizable with extensive, but cautious, inspection of the fossil record. However, barring stereotypical disease symptoms or signs of the pathogenic organisms, identifications of the culprit may be impossible in many cases.

4. An overview of pathogens in the permineralized fossil record

It is difficult to confirm pathogenesis in the compression-impression fossil record, as cellular detail, and hence signs of the pathogen frequently are not preserved. However, the permineralized fossil record, where cellular details of the host and pathogenic agents can be recognized, provides us with greater opportunity to identify host–pathogen interactions. A difficulty in interpreting the

presence of a fungal or other potential pathogen within host tissues is that the host likely was dead when it was preserved, and was exposed to the effects of non-pathogenic, saprophytic organisms. In addition, while alive, the plant may have hosted endophytic fungi that did not elicit a disease response (Krings et al., 2009, 2011) and cannot be considered unequivocally as having been pathogenic, despite its presence within the tissues of the plant. Only the clear presence of a host reaction to an invading organism, or the presence of specialized hyphae such as haustoria that are known to be adaptations of an obligately biotrophic mode of life, are considered here to be adequate evidence for an observed pathogenic interaction.

Although there is fossil evidence of terrestrial prokaryotes and eukaryotes from the Precambrian interval of 1200–1000 Ma, including forms attributed to bacteria (Horodyski and Knauth, 1994), the earliest plant-associated bacteria have been reported from Cretaceous amber deposits (Schmidt and Dilcher, 2007; Adl et al., 2011). For viruses, although experimental evidence indicates that silicification is possible (Ladier and Stedman, 2010; Orange et al., 2011), and molecular phylogenetic studies suggest deep-time origins (Delwart and Li, 2012), there is no indication of a fossil record until the Early Cretaceous. Interestingly, these Cretaceous virus fossils occur in association with insects preserved in amber (Poinar and Poinar, 2005). Plant-associated nematodes, however, virtually lack a fossil record, with the sole, spectacular discovery of a nematode associated endophytically within an Early Devonian land plant from the Rhynie Chert (Poinar et al., 2008).

Fungi provide us with the best fossil record of pathogenesis in plants. The terrestrial fossil record of fungi extends to the late Silurian (Sherwood-Pike and Gray, 1985), and the mycorrhizal associations evident in permineralized material from the spectacular Early Devonian Rhynie Chert of Scotland, document the earliest plant–fungal interactions in the terrestrial record (Remy et al., 1994; Taylor and Taylor, 1997). These commensal relationships probably were essential to the success of the first land plants in colonizing the terrestrial environment (Pirozynski and Malloch, 1975). The silica-permineralized Rhynie floras also preserve several examples of parasitic interactions, such as the infection of the green alga *Palaeonitella* and various spores by chytrids, and fungal infection of the early land plant *Nothia aphylla* (Hass et al., 1994; Remy et al., 1994; Taylor et al., 1992, 1995; Taylor and Taylor, 1997; Krings et al., 2012). To a lesser extent, the carbonate-permineralized Calhoun Coal of the Illinois Basin, and other Late Carboniferous coal-ball deposits, also are a primary source of detailed hyphal structures, spores and fructifications, and examples of apparent plant pathogens. For example, fungi found associated with hypertrophied tissues of the seed *Nucellangium*, and the gymnospermous cone *Lasiostrobus* produced cells with wall appositions and excessive resin as an apparent response to fungal hyphae residing within their tissues (Stidd and Cosentino, 1975; Stubblefield et al., 1984a, 1984b).

More recent work on permineralized material from Antarctica reveals a variety of plant–fungal interactions, such as the first mycorrhizae on the widespread Gondwanan plant *Glossopteris* (Harper et al., 2013), and tyloses present in permineralized Jurassic conifer wood (Harper et al., 2012). Tyloses are cell wall extensions of parenchymatous cells that protrude into the pits of adjacent tracheids, creating occlusions and blockages. This form of plant defense and response to injury has been documented from the Carboniferous Period, although these early examples have been characterized as a physiological response to water stress rather than to pathogen attack (e.g. Scheckler and Galtier, 2003). Younger examples of fungal diseases on angiosperm hosts of Paleogene age can be directly related to extant fungal–plant interactions. An example is well-preserved silica permineralized material from the middle Eocene Princeton Chert locality in southern British Columbia, Canada, that documents diseases such as smuts and tar-spot on diverse angiosperm hosts (Le Page et al., 1994).

5. Conclusions and prospects for the future

We have presented an expanded view of what the field of paleopathology encompasses, including plants, pathogens, and their vectors. As conceived herein, paleopathology has established links to a variety of subdisciplines that examine the modern and fossil aspects of botany, entomology, plant–insect interactions, plant pathology, microbiology, mycology, climatology, and related basic and applied fields. The principal links between modern and fossil studies of plant damage are through studies of plant pathology and plant–insect interactions, which require knowledge of disease-inflicting organisms, plant host responses, and arthropod and nematode vectors. For about 425 million years, the direct evidence of these three major elements of plant pathology has been recorded in the fossil evolutionary archive of plant damage. This potentially extended record houses evidence for environmentally induced disorders as well, such as extreme temperature traumas and moisture and nutrient deficiencies in plants, particularly in foliage.

Although there is significant phenotypic overlap in the symptoms induced in plants by the four major types of pathogens, there should be an increased focus on circumscribing specific micromorphological indicators linking arthropod transmission, pathogen attack and host-plant response. In addition, a greater emphasis on the body-fossil record of the pathogenic organisms themselves would supplement their trace-fossil effects on other organisms, and may better reveal pathogen identities on necrotized tissues and thus enhance diagnoses from the record of plant damage. Consequently, an augmented inventory of diagnosable pathogen-induced damage types can be assembled to track the evolution of plants to their pathogens and associated vectors in time, space, and habitat. To date, the best progress in identifying pathogens in the fossil record has involved exquisite, three-dimensionally preserved material, such as silica- and carbonate preserved Paleozoic deposits, and Cretaceous and Cenozoic ambers. However, the greatest opportunity for tracking pathogens may well come from two-dimensionally preserved compression-impression material, available from a far greater diversity of localities in space, time and habitat, which offer a much greater expanse of surface area, particularly of foliage, important for identifying pathogen symptoms. New developments in scanning electron microscopy linked to electron dispersal spectroscopy, epifluorescence microscopy, high resolution X-ray computerized tomography, time-of-flight secondary ion mass spectroscopy, and image tiling should resolve the identities and effects of pathogens that heretofore were difficult to document or interpret.

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