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***XYLELLA FASTIDIOSA* AND BACTERIAL LEAF SCORCH OF OAKS: SULIMINAL, SUBTLE, AND SUSPECT**

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ABSTRACT

The fastidious xylem-inhabiting bacterium, *Xylella fastidiosa*, is a widely distributed vascular pathogen occurring in a variety of plants and trees. Vectored by several insects (primarily leafhoppers), *X. fastidiosa* causes various symptoms including marginal leaf scorch, decreased fruit production, declining vigor, delayed bud break, stunting, dieback, and sometimes death in susceptible hosts. Surveys have documented a wide distribution of *X. fastidiosa* in oaks, and it is considered by some to be a debilitating pathogen in certain species, especially red oaks. What does all this portend for oak populations? What is the role of *X. fastidiosa* in oak decline? How does *X. fastidiosa* interact with other oak pathogens? This paper briefly reviews the state of our understanding, offers some hopefully relevant commentary, and poses some questions worthy of research attention.

Key words: Insect vectors, leaf disease, tree decline

The fastidious xylem-limited bacterium *Xylella fastidiosa* (Wells et al. 1977) is associated with, and in many cases is considered causal for, leaf scorch/scald and decline diseases in a wide variety of plant species (Hopkins 1977, 1989, Sinclair and Lyon 2005, CABI and EPPO - undated, Mizell et al. undated). With the possible exceptions of Taiwan and India (pending confirmation? – CABI and EPPO - undated), *X. fastidiosa* is known only in the western hemisphere (Global Invasive Species Database 2005, CABI and EPPO - undated,). In the United States, *X. fastidiosa* has been widely reported in association with leaf scorch/leaf scald and decline syndromes on a variety of broad-leaved fruit and shade tree species including members of the genera *Acer*, *Aesculus*, *Carya*, *Celtis*, *Cornus*, *Liquidambar*, *Morus*, *Platanus*, *Prunus*, *Quercus*, *Ulmus*, and *Citrus*. Overall, *X. fastidiosa* has been associated with 75-100 species of plants, both woody and non-woody, belonging to some 30 plant families. In many of these host plant species, the bacterium induces no noticeable symptoms of disease (Hopkins 1989, Sinclair and Lyon 2005).

Genetic, pathogenic, nutritional, and host-specificity variation is known within *X. fastidiosa* and unique strains are recognized (Hopkins 1989, Chen et al. 1995, Colletta-Filho et al. 2001, Mehta and Rosato 2001, Schaad et al. 2004, Schuenzel et al. 2005, Zhang et al. 2005, Gould and Lashomb 2006). Several groups of strains have been distinguished within *X. fastidiosa* based on DNA sequences, protein profiles, and host preferences (Schaad et al. 2004, Schuenzel et al. 2005). Schaad et al. (2004) have proposed three identifiable and potentially-useful subspecies, including subspecies *multiplex* which appears unique to elm, sycamore, oak, and maple (Mundell 2005). To date, however, the genus *Xylella* remains monospecific (Sinclair and Lyon 2005, Gould and Lashomb 2006, CABI and EPPO - undated).

X. fastidiosa is an insect-vectorized pathogen. According to Purcell (1989), virtually all insects that feed predominantly on xylem fluid are potential vectors of *X. fastidiosa*. Within its known range in North America, the most common insect vectors are leafhoppers (Cicadellidae) in the subfamily Cicadellinae (sharpshooters) and spittle bugs or froghoppers (Cercopidae). Specific vectors vary among host plant species and geographic locations. Extensive lists of known and potential insect vectors are available (Lashomb et al. 2002, Gould and Lashomb 2006, CABI and EPPO - undated, Mizell et al. - undated.).

Following introduction of *X. fastidiosa* into xylem elements of susceptible plants, symptoms of infection develop as the bacterium proliferates in the vascular system (xylem). Symptoms may include marginal leaf tissue necrosis (often in older leaves first), premature leaf abscission, decreased fruit production, decline in vigor, stunting and/or reduced growth, delayed bud break, dieback, and ultimately death (Hopkins 1989, Barnard et al. 1998, Sinclair and Lyon 2005, Gould and Lashomb 2006). This complex of symptoms is consistent with and thought to be largely related to reduced water supply to host plant tissues as the bacterium multiplies in xylem elements, although host-produced tyloses and gums, as well as pathogen-produced phytotoxins may be functional in some pathosystems (Hopkins 1989, Sinclair and Lyon 2005, CABI and EPPO - undated).

BACTERIAL LEAF SCORCH OF OAKS

Among the many broad-leaved trees affected by *X. fastidiosa* are several species of oaks (*Quercus* spp.), especially members of the red oak group. Surveys in several eastern states in the U.S. (Chang and Walker 1988, Haygood 1988, Blake 1993, Hartman, Eshenaur and Jarflors 1995, Barnard et al. 1998, Gould et al. 2004) have demonstrated that *X. fastidiosa* is widespread therein and commonly associated with oaks exhibiting leaf scorch and/or decline (Figure 1). Surveys reported from New Jersey (Lashomb et al. 2002, Gould and Lashomb 2006, Gould et al. 2007) suggest that bacterial leaf scorch (“BLS” – the common name of the disease attributed to *X. fastidiosa* infections) is spreading in red oak populations there. Similar to the insect vector situation (above), lists of oaks infected by *X. fastidiosa* are readily available (Lashomb et al. 2002, Sinclair and Lyon 2005, Gould and Lashomb 2006).

CONSIDERATIONS AND UNANSWERED QUESTIONS

Some years ago, this author read a news item in a very popular trade journal that is widely distributed across the U.S. The headline read, “Bacterial Leaf Scorch on the Rise in the Southeast” – a headline clearly intended to signal some level of threat. Such headlines do little to clarify our understanding of this complicated disease scenario. To begin with, the headline presumes at the outset that someone (we) knows (know) what the baseline is; how much BLS did we start with? The reality is that we have no idea, and the “increase” to which the headline refers is likely an increase in the number of reports of BLS resulting from 1) the advent of technologies that facilitate detection and 2) an increase in professional interest and investigation. Of interest to this author is the fact that Dr. George Hepting (one of the “patriarchs” of Forest Pathology in the U.S.) apparently observed oaks exhibiting leaf scorch symptoms decades prior to our ability to detect and identify *X. fastidiosa*. He referred to the symptoms as “leaf dip” (D.H. Marx – personal communication). Recognition of such subtleties and misunderstandings is a must if we are to improve our understanding of BLS in oaks, as well as in other species.

Other factors demanding rigorous evaluation and interpretation when it comes to understanding BLS are environmental considerations and interactions with other diseases. For

example, it is generally recognized that the symptoms resulting from infections by *X. fastidiosa* are “generic” and can be produced by a variety of other causes such as salt damage, drought, other vascular infections, or root disease (Hopkins 1977, Lashomb et al. 2002, Gould and Lashomb 2006). Indeed, Virginia creeper (*Parthenocissus quinquefolia*) inoculated with *X. fastidiosa* failed to express significant leaf scorch symptoms unless subjected to a reduced water (drought?) regime (McElrone, Sherald and Forseth 2001).

How many surveys for or detections of *X. fastidiosa* have considered environmental conditions and have conclusively ruled out the occurrence (simultaneous or sequential) of other diseases? In Florida, for example, turkey oaks (*Quercus laevis*) with and without leaf scorch symptoms and, respectively, with and without detectable infections of *X. fastidiosa* were frequently observed side-by-side. How many of these trees (and which ones) were infected with *Armillaria* and/or *Ganoderma* – common and widespread root pathogens frequently associated with declining turkey oaks (Barnard et al. 1998)? We have no idea. May I submit that this question could be raised in pretty much every situation involving BLS of oaks?

Interestingly, I have read that “scale insects, borers, *Armillaria* root rot, and other biotic diseases may express themselves as secondary pests” on BLS-infected trees (Lashomb et al. 2002, Gould and Lashomb 2006). Do we know that BLS is always primary? Or, could it be secondary (Hopkins 1989)? Why do *X. fastidiosa*-infected and *X. fastidiosa*-free trees of the same species often occur side-by-side (Gould and Lashomb 2006, Barnard et al. 1998)? Is this a function of insect vector preference, genetic variation/resistance in the host, or environmental or pathogenic predisposition to disease development?

What about *X. fastidiosa* and “oak decline”? This phenomenon (oak decline) has been an issue in the southern U.S. for years (Tainter et al. 1990, Oak et al. 2004) and the distribution of oak decline for all intents and purposes can be superimposed on the known range of *X. fastidiosa*, and vice versa. Could there be a link? To this author’s knowledge, there has never been a serious attempt to find out.

There is much we do not know about *X. fastidiosa* and the various diseases with which it is associated. Statements by D. L. Hopkins (1989) perhaps state things best.

- “The combined list of natural hosts for all strains of *X. fastidiosa* ` evidently is limited more by the effort spent in the search for alternate hosts than by the actual host specificity of the bacterium.”
- “Except for a few host-pathogen combinations like PD of grapevine and phony disease of peach, *X. fastidiosa* could be considered a weak or opportunistic pathogen. Strains of *X. fastidiosa* often appear to survive as residents of the xylem vessels in symptomless hosts, but accumulate and produce disease symptoms only if the host is weakened by some other stress factor. ...Stress factors favoring *X. fastidiosa* diseases include drought, other diseases, root pruning with cultivation equipment, overproduction of fruit, normal fruit maturation, and senescence. In most hosts, symptoms of the diseases are not visible until either the time of fruit maturation or late autumn when the hosts are senescing.”
- “In addition to senescence apparently affecting susceptibility of hosts to *X. fastidiosa*, symptoms of the diseases – chlorosis, abscission of leaves, and acropetal symptom development – are also characteristic of plant senescence.”

- “...host senescence appears to be fundamental in diseases caused by *X. fastidiosa*, ...”
- “While other stress factors on the host may favor *X. fastidiosa*, chronic, nonlethal infection by the bacteria also may predispose its hosts to other pathogens and stresses. This seems especially to happen with the shade tree diseases.”
- “With many *X. fastidiosa*-associated diseases, it is difficult to determine whether the bacterium is the primary or a secondary stress factor. In some cases, a synergism with another pathogen or stress factor may be required for disease development.”

Given the complexities and sometimes conflicting realities, it is clear that a thorough understanding of the role of *X. fastidiosa* in scorched and/or declining oaks demands more investigation. Further, assuming climate change is inevitable (and I would hazard a guess that it is – one way or another), the nuances of pathogen X insect vector X environment interactions seem endless. Careful research and data interpretation are essential.

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Figure 1: Scorch symptoms on leaves of Texas red oak (*Quercus buckleyi*) typical of those caused by *Xylella fastidiosa* (photos taken in Austin, TX by Ron Billings).

