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Anne Sicard, Adam R. Zeilinger, Mathieu Vanhove, Tyler E. Schartel, Dylan
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Anne Sicard,[1,2] Adam R. Zeilinger,[1] Mathieu Vanhove,[1]
Tyler E. Schartel,[3] Dylan J. Beal,[1]
Matthew P. Daugherty,[3] and Rodrigo P.P. Almeida[1]

[1]Department of Environmental Science, Policy and Management, University of California, Berkeley, California 94720, USA; email: rodrigoalmeida@berkeley.edu

[2]Biologie et Génétique des Interactions Plant-Parasite, UMR 0385, Centre de Coopération Internationale en Recherche Agronomique pour le Développement-Institut National de la Recherche Agronomique-Montpellier SupAgro, Campus International de Baillarguet, 34398 Montpellier CEDEX 05, France

[3]Department of Entomology, University of California, Riverside, California 92521, USA

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Abstract

The bacterium *Xylella fastidiosa* re-emerged as a plant pathogen of global importance in 2013 when it was first associated with an olive tree disease epidemic in Italy. The current threat to Europe and the Mediterranean basin, as well as other world regions, has increased as multiple *X. fastidiosa* genotypes have now been detected in Italy, France, and Spain. Although *X. fastidiosa* has been studied in the Americas for more than a century, there are no therapeutic solutions to suppress disease development in infected plants. Furthermore, because *X. fastidiosa* is an obligatory plant and insect vector colonizer, the epidemiology and dynamics of each pathosystem are distinct. They depend on the ecological interplay of plant, pathogen, and vector and on how interactions are affected by biotic and abiotic factors, including anthropogenic activities and policy decisions. Our goal with this review is to stimulate discussion and novel research by contextualizing available knowledge on *X. fastidiosa* and how it may be applicable to emerging diseases.

INTRODUCTION

In October 2013, scientists reported that the bacterium *Xylella fastidiosa* was present in the southern Italian region of Apulia (129). In most circumstances, an aggressive effort to contain and eradicate *X. fastidiosa* would have been implemented, thereby limiting the spread of the bacterium to other regions in Italy and Europe or even eradicating it. However, following the October 2013 report, expected and unexpected cascades of events led to the establishment of *X. fastidiosa* in various regions along the Mediterranean basin. As with past epidemics, the emergence of *X. fastidiosa* in Europe has motivated much research into the ecological and evolutionary drivers of the establishment and spread of this plant pathogen. Our goal in this review is to place recent knowledge on *X. fastidiosa* within epidemiological and agroecological contexts and outline how this knowledge may be applied to risk assessment and management of other emerging epidemics of this pathogen worldwide.

BRIEF HISTORICAL PERSPECTIVE

In 1892, Newton Pierce was the first to report on a disease caused by *X. fastidiosa* (111). Pierce studied an epidemic of California vine disease in Southern California that had devastating consequences to the grape industry in the region; the disease was later called Pierce's disease (PD). More than a century later, despite being the most studied and best understood *X. fastidiosa* disease, PD remains a significant problem to the grape industry in California (141). Other than a 1930–1940 epidemic that led to major breakthroughs such as the identification of insect vectors, *X. fastidiosa* and the diseases it causes remained poorly characterized until the late 1970s when it was first cultured in vitro (40). The bacterium remained a pathogen of interest primarily in the United States until 1987, when it emerged in Brazil and was associated with a citrus disease (21, 64). The impact of *X. fastidiosa* on the citrus industry led to the development of a research community in Brazil that eventually sequenced the genome of the bacterium in 2000 (132). At the same time, another PD epidemic in Southern California devastated the local wine industry after the establishment of an invasive vector, *Homalodisca vitripennis* (14, 137). These two epidemics initiated a two-decade period of intensive study of *X. fastidiosa* and resulted in an unprecedented accumulation of knowledge concerning this pathogen (42). Although research on *X. fastidiosa* continues to focus on grapevines and citrus, new epidemics in other crops in other regions of the United States, as well as the emergence of the pathogen in Europe, are bound to increase and diversify knowledge about this bacterium and the diseases it causes. In 2013, Purcell (115) provided a more thorough historical perspective of *X. fastidiosa* prior to current epidemics.

Emergence of *Xylella fastidiosa* in Europe

X. fastidiosa has been a target of concern for decades, including efforts to understand why it had not already established in Europe (e.g., 114). Before 2013, there were only sporadic reports of *X. fastidiosa* detection in Europe (50), but they were not independently confirmed and did not raise concerns. Thus, the 2013 report of a new olive tree disease associated with *X. fastidiosa* in Italy was troubling but not unexpected (129). The presence of *X. fastidiosa* in Europe led the European Commission to require mandatory surveys of the bacterium in member countries. These surveys led to the determination that *X. fastidiosa* is established in France (island of Corsica and southern region of Côte d'Azur) and Spain (Balearic islands and province of Valencia) (9, 44, 105, 124). Reports from other countries exist but do not appear to include open-air outbreaks and were limited to isolated detection events (**https://gd.eppo.int/taxon/XYLEFA/distribution**).

A strong case has been built linking the importation of *X. fastidiosa*–infected plant material from Central America to the emergence of the olive tree disease in Italy. Evidence shows that a single genotype has established in Apulia (60, 73, 87, 90) and that imported plant material infected with different strains of *X. fastidiosa* has been intercepted at ports and markets in different European countries (12, 87). A different picture, however, is emerging from France and Spain. Whereas Italy has a localized disease epidemic associated with a single pathogen introduction, the other two countries appear to have had multiple independent introductions of different *X. fastidiosa* genotypes that are widely spread in the landscape (9, 44, 105, 124). The lack of a localized disease epidemic and the spatial distribution of different genotypes (e.g., throughout Corsica and Balearic Islands) suggest that *X. fastidiosa* was not very recently introduced. We expect the temporal dynamics of these introductions to be understood better once more genomic and other types of data become available.

ESSENTIALS OF *XYLELLA FASTIDIOSA* BIOLOGY

X. fastidiosa is a xylem-limited bacterium in the family Xanthomonadaceae (Gammaproteobacteria) that is an obligatory colonizer of plant and insect hosts (143). The list of plants associated with *X. fastidiosa* includes more than 350 species; we expect that number to increase as research on new epidemics in the Mediterranean accumulates (52). However, the list of host species is largely based on field-observed associations of *X. fastidiosa* hosts, both symptomatic and asymptomatic, and limited research has been done under controlled conditions to confirm these associations. Furthermore, despite this large number of host plants, *X. fastidiosa* is a bacterial species with various well-supported phylogenetic clades, each of which has a more limited host range. In fact, when pathogenicity to plants is considered, *X. fastidiosa* phylogenetic clades have a limited host range. An alternative view based on currently available data is that *X. fastidiosa* is associated with a large number of plant species as a commensalist, but a limited number of clades and particular bacterial genotypes are responsible for and specific to a small number of plant diseases.

Unlike plant disease, the association of *X. fastidiosa* with hemipteran vectors, the only means of natural dissemination, is not the outcome of particular insect-pathogen combinations. There are two main insect vector groups, the sharpshooter leafhoppers (Cicadellidae subfamily Cicadellinae) and spittlebugs (Cercopoidea, families Aphrophoridae, Cercopidae, and Clastopteridae), both of which are xylem-sap feeders (68, 131). In 1965, Frazier hypothesized that all insect species in these groups should be considered *X. fastidiosa* vectors, a proposal that has stood the test of time (57). Importantly, available data demonstrate that insect vectors are capable of transmitting all *X. fastidiosa* genotypes without specificity (4). Limited evidence exists for cicadas (Cicadoidea) as *X. fastidiosa* vectors; this group requires further investigation, considering the large populations in certain Mediterranean regions (77, 106).

Plant and Insect Colonization

Plant colonization by *X. fastidiosa* is a gradual process in which blockage of individual vessels due to bacterial population growth, associated plant responses (e.g., tyloses), and concomitant spread among vessels via pit membranes reduce sap flow in the xylem network (10, 95). Pathogen cell density mediates processes linked to plant colonization and movement, which are upregulated at low cell density; by contrast, *X. fastidiosa* populations switch to an insect acquisition and colonization phenotype that precludes further plant colonization at high cell densities (22, 96). The nature of this molecular switch is likely linked to *X. fastidiosa* biology (4). Xylem-sap-feeding insects are negatively impacted by plant water stress and increases in water column tension—phenomena that are also triggered by *X. fastidiosa* infection (7, 93). In addition, water-stressed plants often

exhibit symptoms, such as leaf scorch, that are common consequences of pathogenic *X. fastidiosa* infections (93). Research has shown that sharpshooter vectors visually discriminate against plants exhibiting leaf scorch symptoms, with or without *X. fastidiosa* (38, 92). As previously proposed, we interpret this cell-density-dependent behavior as an adaptation that permits the bacterium to colonize plants successfully yet limits excessive multiplication that would lead to disease symptoms and thus decreased host plant attractiveness to insect vectors. In fact, *X. fastidiosa* spread has been mathematically and experimentally demonstrated to peak prior to full disease symptom development, a point at which within-plant bacterial populations are large and acquisition by vectors is maximized, but insects strongly discriminate against such plants (39). Finally, although *X. fastidiosa* may negatively impact plants in different ways, the only evidence of potential negative effects on insect vectors is recent work demonstrating that the population size of a chitinase mutant strain did not increase in insect vectors when compared with a wild-type control (81), suggesting that vector colonization by *X. fastidiosa* involves chitin degradation. Although this could impact insect hosts, there is no direct evidence that *X. fastidiosa* colonization leads to vector behavioral or fitness-related changes.

Vector Transmission

Several aspects of *X. fastidiosa*–vector interactions relevant to plant-to-plant transmission have been studied, allowing for broad generalizations to be summarized. First, as mentioned above, there is no evidence of *X. fastidiosa* genotype-vector specificity, indicating that biological and molecular interactions between the pathogen and vector are phylogenetically conserved. Once acquired from plants, *X. fastidiosa* attaches to and colonizes specific regions in the foregut of vectors (i.e., precibarium and cibarium) to form a persistent biofilm (76, 81, 118). The fact that *X. fastidiosa* is propagative and persistent yet noncirculative in insect vectors has been demonstrated with several lines of evidence, including the lack of transovarial (58) and transstadial (5, 117) transmission of bacterium in vectors, microscopical observations of the foregut (6, 17, 118), transmission studies with aging insects (6, 69), and other approaches demonstrating bacterial persistence and multiplication in vectors (69, 76, 81).

Even though *X. fastidiosa* forms a biofilm on the cuticular foregut of insect vectors, there is no detectable latent period required for transmission (117). In other words, transmission may occur immediately after acquisition, even if a biofilm has not developed. However, recent work indicates there may be a positive correlation between bacterial populations in insects and plant infection (30), suggesting that the dynamics of fluid flow in vector foreguts, coupled with specific probing behaviors during xylem-sap feeding that may facilitate pathogen detachment, are associated with *X. fastidiosa* inoculation. Yet the specific insect probing behavior(s) associated with *X. fastidiosa* inoculation into plants remains to be determined.

Finally, in the context of pathogen spread, it is important to dissociate the capacity of an insect species to transmit *X. fastidiosa* from its epidemiological role. *X. fastidiosa* transmission efficiency is impacted by various factors, including vector species (e.g., 34, 113), pathogen genotype (88), host plant species during pathogen acquisition and inoculation (88), bacterial populations within plants (70) and in different tissues in the same plant (36), vector preference for host plant type (121, 131), host plant tissues (36, 120), disease symptoms (38, 92), and temperature (35, 39). Therefore, although a large number of species are potential vectors of interest because of their inherent capacity to transmit *X. fastidiosa*, few are of epidemiological relevance in a specific disease system due to the large number of factors that affect the transmission process (29, 67, 121). Although the factors listed above focus on the transmission process, aspects of vector and host plant biology and ecology, seasonality, and crop-management practices are also relevant.

Xylella fastidiosa Diversity and Host Plant Relationships

Human-mediated movement of infected plant material has contributed to long-range dissemination of *X. fastidiosa*, but currently available genetic data indicate that three of the five *X. fastidiosa* subspecies are allopatric in origin. *X. fastidiosa* subsp. *fastidiosa* originated in Central America, subsp. *multiplex* in North America, and subsp. *pauca* in South America. Intersubspecific genetic introgressions between subsp. *fastidiosa* and subsp. *multiplex* may have led to the emergence of *X. fastidiosa* subsp. *morus* (100). The origin of *X. fastidiosa* subsp. *sandyi* is still debated (4); recent genomic data suggest that this clade clusters within subsp. *fastidiosa* (60, 87, 89). Applying a phylogenetically representative genome sequence data set to *X. fastidiosa* taxonomy will likely clarify pending questions and lead to a revision of the group. As diversity is not a goal of this review, we direct readers to recent work on the topic (28, 60, 89).

Genetically typing *X. fastidiosa* was challenging because this bacterium is naturally competent (79) and populations undergo extensive homologous recombination (HR) within and among phylogenetic clades (3, 28, 75, 98–101, 103). In 2005, a multilocus sequence typing (MLST) scheme for *X. fastidiosa* was first introduced (130), leading to substantial improvement in understanding the phylogenetic relationships among isolates and how clades are organized in relation to both host plants and geographical location. When this article was prepared, 293 isolates from 76 plant species across three continents were publicly available in the *X. fastidiosa* MLST database (**https://pubmlst.org/xfastidiosa/**). In total, 55 sequence types (STs) are associated with 25 host plant families (for visualization of these data, see **https://microreact.org/project/Hkwv1v7tZ**). Note that the data set is geographically and host plant biased (e.g., most samples are from the United States, particularly California), limited in size, and based on a robust yet small number of loci ($n = 7$). Nevertheless, individual STs appear to be associated with a limited number of host plants. In other words, there is some host specialization in the form of disease relationships as determined from symptomatic plants in the field. ST-based *X. fastidiosa* identification using plant species previously infected elsewhere with a particular ST may be used to predict which host plant species are susceptible in a new region following an introduction. In other words, an almond-infecting ST previously described in the United States should also infect almonds in Spain, as was recently determined (124). We expect this hypothesis will be tested in a robust manner with a larger data set, given its potentially regulatory consequences for quarantine circumstances.

Predictability of Host Jumps

The potential for introduced pathogens to form novel associations with hosts and the ensuing consequences of these novel associations on disease dynamics are both of considerable interest because pathogens that form novel biotic associations in a naive geographic range may experience relaxed constraints on susceptibility and pathogenicity, which in turn facilitate disease epidemics (138, 139, 147). Disease epidemics can start as the result of a pathogen jumping to novel hosts. Host jumps are likely common throughout evolutionary history and occur through a three-step process (51, 146). In the first step, a novel host is exposed to the emerging pathogen. In the following step, if the pathogen and novel host are compatible, the pathogen successfully infects the novel host. In the final step, the pathogen must sufficiently transmit among individuals within the novel host's populations.

The quarantine pathogen status of *X. fastidiosa*, coupled with its ability to colonize a large number of host plant species, highlights the practical importance of being able to predict novel host susceptibility to an individual ST or a phylogenetic clade of *X. fastidiosa*. The underlying factors associated with *X. fastidiosa* host plant specificity remain to be determined, but concerns about this question are of immediate relevance in areas where *X. fastidiosa* has been recently introduced.

Using the above-mentioned MLST data set, we attempted to determine whether *X. fastidiosa* host shifts are predictable. Previous work with plant-pathogenic fungi, for example, indicated that pathogen host jumps tend to occur in plant species more closely related to those that the pathogen already infects (107). In other words, the closer the phylogenetic relationship between a host of concern and known pathogen host, the higher the likelihood that a host jump will occur. This elegant work follows expectations of coevolutionary theory and has had some success with plant-pathogenic fungi. Thus, we performed a comparative host–pathogen coevolutionary analysis to investigate host plant–pathogen relationships. Maximum likelihood and Bayesian phylogenies were generated for host species and *X. fastidiosa* STs to test for coevolutionary congruence (as described in 85). No significant coevolutionary fit could be established (**Supplemental Figure 1**). These analyses confirm the difficulty of predicting *X. fastidiosa* host jumps.

In summary, currently available data suggest that using taxonomy derived from MLST data sets, i.e., ST number, to infer *X. fastidiosa* host range is both useful and problematic. Assignment of an isolate to a particular ST suggests which plant species may be susceptible to pathogenic infections on the basis of available knowledge. By contrast, the use of ST does not permit inferences on potential host range or host jumps. The level of genetic and phenotypic diversity currently used to define *X. fastidiosa* subspecies, although helpful taxonomically and in a biogeographic context, lacks resolution to provide reliable information regarding host range. We expect large whole-genome-sequence data sets to assist in addressing these limitations.

ECOLOGICAL DRIVERS OF NOVEL DISEASE EPIDEMICS

In an effort to deconstruct pathways leading to the emergence of *X. fastidiosa* diseases and outbreaks, we devised a simplistic flowchart as a framework for discussion (**Figure 1**). On the one hand, pathogen and vectors may be introduced or endemic, and human-mediated dispersal has the potential to impact vector geographical distributions. On the other hand, climatic conditions and associated changes to biotic and abiotic factors in the environment may impact the distribution of *X. fastidiosa* and associated vector species in the landscape, potentially increasing or decreasing disease prevalence. Finally, changes to agricultural practices such as crop intensification, irrigation, pest control, fertilization protocols, adoption of mixed crop fields or cover crops, and a myriad of other modifications to how land is used could also positively or negatively impact the role of *X. fastidiosa* as a pathogen. Here, we explore how different factors affect the emergence of *X. fastidiosa* diseases (also see examples in **Supplemental Figure 2**). We note that the factors presented here are split into distinct sections for arbitrary reasons, whereas in reality they are interconnected and interdependent.

Contribution of Vectors to *Xylella fastidiosa* Outbreaks

The potential for *X. fastidiosa* to invade, establish, and expand beyond its current range is partly predicated on the presence and distribution of insect vectors in a novel range. *X. fastidiosa* vectors are globally distributed (33, 50, 121); however, most of these insect species are unlikely to contribute to novel *X. fastidiosa* outbreaks. In other words, the presence of vectors will not necessarily lead to outbreaks if *X. fastidiosa* is introduced, or is already present, in the landscape. Ultimately, the dynamic ecological interactions among vector, pathogen, and plants in the landscape are more likely to determine the importance of a particular vector species on pathogen spread than are the intrinsic biological characteristics of the insect. Factors that may modify the relative importance of a vector species in epidemics include, but are not limited to, pathogen genotype, vector phenology, vector natural infectivity (i.e., fraction of vectors in the population that are infective), vector diet breadth, vector mobility, plant phenology, plant water status, plant nutrient status, plant
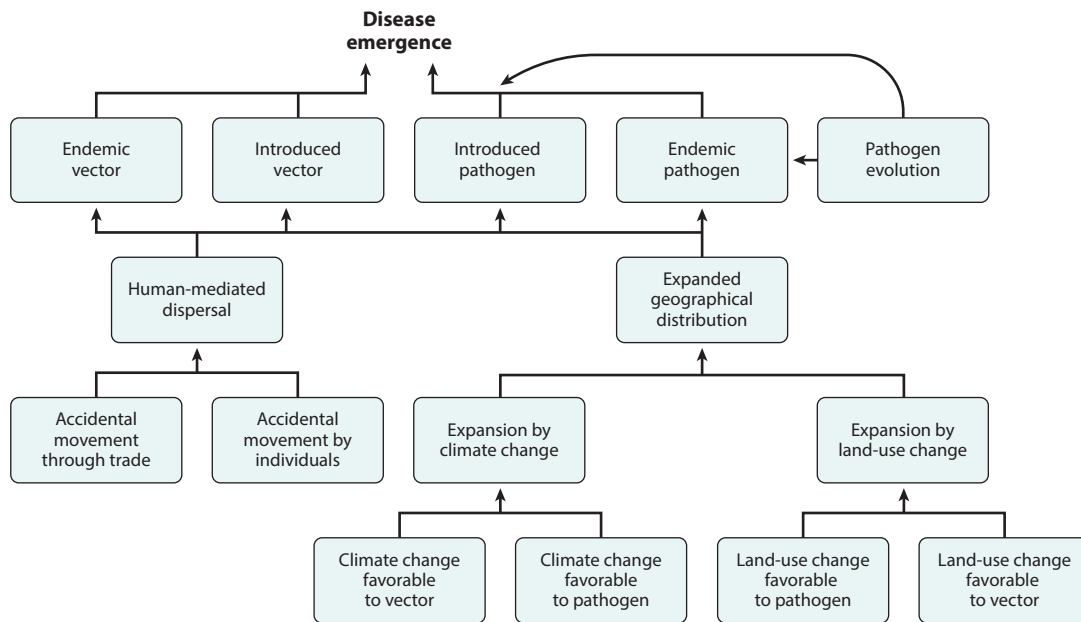
Figure 1

The emergence of *Xylella fastidiosa* diseases may be driven by a wide range of ecological factors, several of which likely interact with each other. Endemic and introduced insect vectors and pathogens play a central role in disease emergence, as they are subject to an array of factors that may contribute to novel disease or the geographical expansion and/or retraction of an existing disease. The flowchart highlights the hypothesized causal role of three major drivers of disease emergence: human-mediated introductions into new areas, climate change, and land-use change. Additionally, natural selection and other evolutionary processes may play a role. Pathogen evolution is likely to be most relevant for the emergence of novel *X. fastidiosa* disease, given the lack of vector species–*X. fastidiosa* genotype specificity. **Supplemental Figure 2** exemplifies how this simplified diagram may be helpful for understanding disease emergence dynamics.

community composition, agricultural practices, and climate. Some, but not all, of these factors have been explicitly tested in *X. fastidiosa* diseases (e.g., 39, 61, 78).

To our knowledge, there is one example of a vector introduction leading to *X. fastidiosa* epidemics. Introduction of the sharpshooter *H. vitripennis* to Southern California and subsequent outbreaks of PD in that region demonstrate that an invasive vector species can alter the dynamics of an established *X. fastidiosa* disease system. The large population size of *H. vitripennis*, rather than transmission efficiency, is thought to be responsible for that outbreak (14, 34). High population densities in citrus groves and spillover into adjacent vineyards are considered critical elements of the role *H. vitripennis* plays in driving epidemics (13, 110). So far, *H. vitripennis*–linked epidemics in Southern California represent a unique case among *X. fastidiosa* outbreaks owing to the invasive status of the vector. Invasion of infectious *X. fastidiosa* vectors into a new region represents a distinct but related mode for disease emergence. Although this scenario deserves more attention, there are no currently reported *X. fastidiosa* epidemics where it has occurred.

Supplemental Material >

Introduced Pathogens in Landscapes That Favor Spread

Introduction of *X. fastidiosa*–infected plant material has been associated with many cases of disease emergence across the world. Among the most dramatic examples is the introduction of *X. fastidiosa* subsp. *fastidiosa*, which led to PD in the United States more than a century ago

(102, 148), its subsequent geographic expansion to Taiwan (140) and Spain (104), and the emergence of *X. fastidiosa*–related diseases in both countries. Another example is the introduction of plum-infecting *X. fastidiosa* subsp. *multiplex* from North America to South America; in this case, there is also evidence that the invasive strain contributed to the emergence of citrus variegated chlorosis (CVC) and coffee leaf scorch outbreaks in Brazil (28, 103). These examples of *X. fastidiosa* outbreaks are the result of successful introduction and establishment of this pathogen in new environments where the same crop plant species were cultivated.

Role of Nonsymptomatic Pathogen Reservoirs

Given its large number of asymptomatic host species, *X. fastidiosa* can easily be introduced and established in a new environment. Under natural conditions, most *X. fastidiosa* hosts are asymptomatic but can act as reservoirs that contribute to *X. fastidiosa* establishment, maintenance, and spread. The importance of asymptomatic reservoirs on disease epidemiology varies among pathosystems; for example, they appear important for PD in Northern California (2, 115) but not for CVC in Brazil (83). In addition, different pathogen multiplication rates have been observed in noncrop plants, suggesting that different hosts may contribute differentially to spread (e.g., 119). Therefore, it is important to consider the role of plant community composition and the prevalence of asymptomatic hosts on pathogen maintenance in the environment, how those are distinct from the relative role these plants may have on disease epidemiology, and how the role of reservoirs is highly dependent on the pathogen-host-vector system. The degree to which reservoir hosts contribute to epidemics may be related to the relative importance of primary versus secondary spread. Estimating primary and secondary spread is also not a trivial task; however, Parry et al. (108) describe a statistical method using spatiotemporal data on disease incidence for that purpose. Lastly, wild plants also maintain *X. fastidiosa* genetic diversity. They may also be hosts for mixed infections and, thus, potential facilitators of gene flow.

Climatic Influences on the Distribution and Emergence of *X. fastidiosa* Diseases

Climate is an important driver of *X. fastidiosa* disease. It influences the distribution and dynamics of vector populations, where and how host plants are grown, pathogen transmission, and plant-pathogen infection dynamics. Manipulative studies have shown that higher temperatures generally favor greater feeding rates (136) and survival of sharpshooter vectors (135), higher transmission efficiency (35), higher *X. fastidiosa* multiplication rate (54), shorter incubation period and latency in plants (39), and greater persistence of infections in plants (86). These effects translate to different patterns in the field, especially with respect to plant infection; for example, lower winter temperatures are associated with seasonal reductions in *X. fastidiosa* infection level (133) and recovery of some infected plants (53, 84, 116). Similarly, a combination of higher temperature and precipitation is associated with higher vector population densities in the field (e.g., 61), and precipitation is positively associated with *X. fastidiosa* establishment or spread (15, 16). These documented effects of climate may further explain broadscale patterns of *X. fastidiosa* pathosystems, such as the regional distribution of an invasive vector in the western United States (71), geographic differences in overwinter recovery of infected plants (86), the restricted distribution of many *X. fastidiosa* diseases to climatically moderate coastal regions for much of the United States (114), and the ultimate distribution of *X. fastidiosa* disease in invaded regions of Italy and the Mediterranean (15, 16). Collectively, available evidence indicates that understanding the precise nature of climatic relationships will yield insights into which regions not currently affected are most suitable for disease emergence if *X. fastidiosa* is introduced.

Given underlying influences of temperature and precipitation on *X. fastidiosa* pathosystems, changes to climatic conditions may alter *X. fastidiosa* disease distribution or incidence. In general, global circulation models predict that many areas will experience warmer winter and nocturnal temperatures (8, 72, 125), which in turn will contribute to increased precipitation in some areas but more frequent or severe drought in others (59, 65, 125). Such changes may alter the suitability of areas where *X. fastidiosa* is not currently established and expand or shift disease distribution in nearby regions where it is endemic or has successfully invaded. Climate change may also lead to novel *X. fastidiosa* epidemics through the convergence of *X. fastidiosa* and novel host distributions. Alternatively, climate change may impact established or endemic *X. fastidiosa* pathosystems. In these cases, the agricultural system will likely migrate gradually to more suitable climates, as is happening for grapevine cultivation on the US West Coast (144), thereby contributing to the persistence of the pathosystem even if disease prevalence is somewhat altered. Thus far, studies that quantify the impact of climate change on *X. fastidiosa* distribution are largely lacking. An exception is a pair of studies that projected the potential extent of the *X. fastidiosa* invasion in Italy and throughout the region (15, 16). Those studies estimated that temperature and precipitation during the driest and wettest months contribute strongly to *X. fastidiosa* suitability, yet there were modest effects on disease expansion assuming current climate change projections. Additional studies of this type are needed, ideally drawing on a broader set of occurrence records for multiple *X. fastidiosa* subspecies, to generate robust predictions regarding the emergence of novel epidemics.

Other Anthropogenic Factors

Anthropogenic factors also play a role in generating novel pathogen-host associations that lead to disease epidemics. Human-mediated transport and introduction of pathogens through new trade routes among previously disconnected countries (1) and increasingly efficient means of transportation are believed to be responsible for generating cosmopolitan pathogens as well as for increasing the frequency and magnitude of disease outbreaks worldwide (32, 139). Human conversion of natural habitats into agroecosystems and monocultures as well as farming practices that place susceptible host populations in close proximity to each other further exacerbate the effects of introducing pathogens into a novel range (139). As a result, the combination of climate change and other anthropogenic factors generates serious concerns regarding the introduction and spread of invasive species, vectors, and pathogens at local to continental scales (11, 32, 49, 109, 126, 134).

Inadvertent transportation of pathogens with agricultural products is one means by which the first step of pathogen host jumping may be affected (32, 139). The recent introduction of *X. fastidiosa* into Italy may be one such case. Bosso et al. (15, 16) found that areas of intensive agriculture, complex cultivation patterns, olive groves, annual crops associated with permanent crops, orchards and vineyards, forest (essentially oak woodland), and Mediterranean shrubland were predictor variables associated with *X. fastidiosa* occurrence in Italy. In addition to these agricultural factors, *X. fastidiosa* infection in Italy may spread to multiple alternative hosts, although the distributions of these alternative hosts and the extent to which they co-occur with olive crops are not well understood (91, 112, 128, 129).

Whereas the introduction of pathogens into novel ranges may facilitate the first step of host jumping, agricultural practices can influence the degree or extent of pathogen-host plant compatibility and, thus, the disease prevalence and severity. In agricultural systems, increasing diversity of resistance genes within host plant populations can lead to reduced pathogen prevalence (18, 45, 145, 149). However, when there is little genetic diversity in resistance genes within a host plant population, a pathogen adapted to that particular host genotype may rapidly increase in

frequency and lead to disease outbreak or epidemics (139). Agricultural practices where tracts of land are planted with monoculture crops with limited resistance diversity may thereby provide ideal conditions to facilitate pathogen establishment and spread. This is the case for most *X. fastidiosa*–susceptible crops and their cultivation practices; most are perennial plants such as grapevine, citrus, and almond trees that are vegetatively propagated as clones and planted in large numbers as monocultures.

In summary, human-mediated transportation and agricultural practices may be influential in determining the extent of *X. fastidiosa* distributions and, thus, the severity of potential *X. fastidiosa* epidemics. Introductions of *X. fastidiosa* into regions where (*a*) novel host plants are available, (*b*) little diversity of resistance to *X. fastidiosa* infection (and thus greater degrees of compatibility with *X. fastidiosa*) is present, and (*c*) limited spatiotemporal heterogeneity exists among compatible host species may promote *X. fastidiosa*–related disease epidemics. Yet on the basis of evidence from previous *X. fastidiosa* introductions where conditions may have imposed constraints on *X. fastidiosa* establishment and spread, two end results of *X. fastidiosa* introduction and establishment appear possible: the re-emergence of a known disease in a new area and/or the emergence of a new disease involving a new plant host (4).

XYLELLA FASTIDIOSA EVOLUTION AND ADAPTATION

Besides infecting the hosts to which it was preadapted, *X. fastidiosa* can also increase its host range. The major evolutionary forces likely involved in novel host adaptation include mutations, homologous and nonhomologous gene flow, and selection. Prophage regions represent a significant proportion of *X. fastidiosa* genomes, but they are also highly variable and may not be involved in host adaptation. Furthermore, although no experimental proof yet exists, available data provide indirect support for various processes leading to host adaptation.

Support for Homologous Recombination on Host Adaptation

HR may be one of the main drivers of *X. fastidiosa* evolution and adaptation (4, 28). Compared with point mutations, HR has been estimated to contribute approximately three times more to *X. fastidiosa* genetic diversity at the nucleotide level (130). HR in *X. fastidiosa* consists of an exchange of typically small DNA fragments (usually ~1 kb) between related genotypes (80). Because *X. fastidiosa* is naturally competent (79), coinfection of plant hosts and/or insect vectors may be a requirement for HR, and as both occur (23, 31), genetic exchanges and thus recombination could happen in both environments. How common coinfections are in nature and how conducive these environments are for recombination remain to be determined, but the sympatric coexistence of multiple genotypes likely facilitates such processes. Although competency may be a conserved trait in *X. fastidiosa*, there are differences in transformation efficiency among subspecies and closely related strains (75).

Data indicate that *X. fastidiosa* subspecies have evolved in allopatry but have been brought into contact (human-mediated processes) on multiple occasions. These events provide clear examples of HR between endemic and introduced *X. fastidiosa* genotypes (e.g., 28) that are often associated with disease emergence. For example, recombination between endemic *X. fastidiosa* subsp. *pauca* and introduced *X. fastidiosa* subsp. *multiplex* in South America may have facilitated pathogen adaptation to citrus and coffee and, thus, are linked to the origin of coffee leaf scorch and CVC outbreaks in Brazil (28, 103). In a similar manner, *X. fastidiosa* subsp. *morus* infecting mulberry seems to have originated from large-scale HR events between *X. fastidiosa* subsp. *fastidiosa* and subsp. *multiplex* (100). These examples emphasize the risk of introducing new genotypes in areas

where this pathogen has already been detected, as it increases the chances of gene flow and might thus result in an expansion of host range and the emergence of new diseases.

Although HR has been associated with emerging diseases, *X. fastidiosa* lineages that have split and are host specific may also recombine but remain genetically and ecologically isolated. Phylogenetic evidence indicating that CVC emerged from coffee-infecting *X. fastidiosa* has accumulated (e.g., 28). However, coffee- and citrus-infecting *X. fastidiosa* in Brazil, for example, are host specific and phylogenetically distinct (3), even though these crops and respective diseases occur sympatrically and share insect vectors. In fact, when adjacent citrus and coffee orchards were surveyed, all isolates were genetically split into coffee or citrus groups, indicating strong biological isolation (56). In summary, although HR has been frequently associated with *X. fastidiosa* host jumps and new diseases, even frequent gene flow among sympatric and closely related clades of *X. fastidiosa* may not be enough to break down biological barriers associated with host colonization. In other words, host adaptation and specificity appear to carry ecological costs and possibly select for aggressiveness.

Laterally Transferred Elements

Laterally transferred elements such as plasmids and integrated prophages play an important role in *X. fastidiosa* evolution and genetic diversity and could contribute to the ability of this species to infect new hosts. The *X. fastidiosa* flexible genome primarily comprises plasmids, integrated prophages, and genomic islands. The integrase genes of prophages could contribute to both the introduction of new genes and rearrangements. The most evident differences observed between *X. fastidiosa* subspecies genomes seem to be linked to phage-like sequences. This observation has led several authors to propose that laterally transferred elements mediate the divergence of lineages leading to host specificity (43, 98, 142). Interestingly, in one studied case, the horizontally transferred gene pool was independently regulated in relation to the core genome (98). Whether laterally transferred elements are indeed involved in host speciation remains to be determined, but if they were, they would have to be shared by isolates infecting a given host.

Several plasmids harbored by different strains of *X. fastidiosa* have been described (122), but the functions of a significant fraction of plasmid-encoded genes remain unknown. These plasmids seem to lack some modules that would confer selective advantages to the strains harboring them (122). Nevertheless, one plasmid possesses a functional type IV secretion system enabling conjugation within and between subspecies (19, 123). Exchanges of plasmids have also been reported within (66) and between subspecies (60, 66). These conjugative plasmids could increase the chance of acquisition of new features involved in pathogenicity or exploitation of new resources and adaptation to new environments. Even in the absence of direct selective pressure, conjugative plasmids may confer a fitness advantage to their host (46, 55).

Mutations and Host Selective Pressure

On the basis of four strains belonging to four subspecies, Doddapaneni et al. (48) have estimated that the average SNP and INDEL frequencies are 0.01 and 0.02, respectively, per base pair of DNA. Although the effects of point mutations on *X. fastidiosa* adaptation to new hosts have been less studied than the role of HR, the recent infection of olive trees by *X. fastidiosa* subsp. *pauca* isolates highlights their importance. Studies (60, 87, 90) have shown that all isolates from olive trees in Italy belong to the same ST that also contains Costa Rican isolates infecting oleander and coffee plants. Given the small number of SNPs that differentiate strains infecting olive trees from those infecting oleander or coffee, adaptation to this new host in Apulia likely results from a few mutations under selective pressure. Interestingly, infection of olive trees by this bacterium also

occurs in Brazil and Argentina (27, 62). The olive strains from Brazil belong to another ST that includes coffee strains, pointing toward convergent evolution; by contrast, the strains in Argentina remain uncharacterized.

After a host jump, strong host selective pressures may purge less-fit genotypes from populations (24). Doddapaneni et al. (47) noticed that long internal branch lengths with groupings of short terminal branches that correspond to host-specific phylogenetic clades characterize phylogenetic trees of *X. fastidiosa* isolates. The authors hypothesized that this tree topology was the result of strong selective pressures. The lack of cross-infection between sympatric isolates infecting different hosts seems to corroborate this idea (e.g., 3, 56, 63). With few exceptions, most *X. fastidiosa* strains appear to be able to cause disease in only one or few crop species, pointing toward strong host speciation (e.g., 3, 101). In addition, replacement of local isolates (local populations) has not been reported to occur in nature, suggesting that isolates adapted to a given environment might be recalcitrant to invaders that could be less fit to local conditions (26). The genetic basis of host specificity remains to be determined, although whole-genome comparison of highly specialized isolates occurring in sympatry is likely to provide insights into this question.

Hypothesis on Evolution of Host Adaptation

Plant diversity is much higher in natural environments than in the ecological systems of mono-cultures of perennial hosts affected by *X. fastidiosa*. Because *X. fastidiosa* vectors are generally polyphagous, the probability that a given isolate in a natural environment will encounter the same host genotype is reasonably lower than in a monoculture. We suggest that a diverse plant community limits bacterial adaptation to a given host, selects for generalist genotypes that are not pathogenic, and generates and sustains a genetically diverse *X. fastidiosa* population in the landscape (**Figure 2**). As previously discussed, the prevailing hypothesis on *X. fastidiosa* biology posits that conflicting biological and ecological processes mediate within and between host colonization (22). In crop systems, once *X. fastidiosa* has adapted to a new host, isolates might be selected for fast movement and multiplication within plants, which increase the likelihood of plant-to-plant transmission (70). Faster movement and multiplication within plants are correlated with virulence and aggressiveness (e.g., 41, 94, 95). Purifying selection in these environments might be a strong factor in reducing pathogen diversity.

In other words, as *X. fastidiosa* in crop systems becomes more adapted to a particular host species, it also becomes more aggressive because it spreads among hosts more efficiently (**Figure 3**). Such strong selection would purge less-fit genotypes from populations, leading to highly specialized pathogens. Here, HR events within populations would lead to reduced diversification, as replacing alleles would be similar. However, owing to sequence divergence and ecological isolation, HR events with unrelated genotypes are both rare and associated with genes that do not determine host specificity. Genes important for the colonization of individual host species (i.e., associated with host specificity) are likely recalcitrant to HR, and a recipient bacterial strain is less fit, removed from the population, and therefore not detected. In summary, we propose that *X. fastidiosa* specialization in monoculture agricultural systems leads to more aggressive pathogen genotypes. We also postulate that such aggressiveness is maximized, and modulated, at a threshold point where the development of plant disease symptoms becomes incompatible with vector host preference behaviors as well as pathogen acquisition and consequently decreases rates of *X. fastidiosa* spread.

DISEASE HARM TO AGROECOSYSTEM SERVICES

X. fastidiosa emergence around the world and associated disease epidemics have generated widespread concern and controversy, owing in large part to effects on agroecosystems. Disease
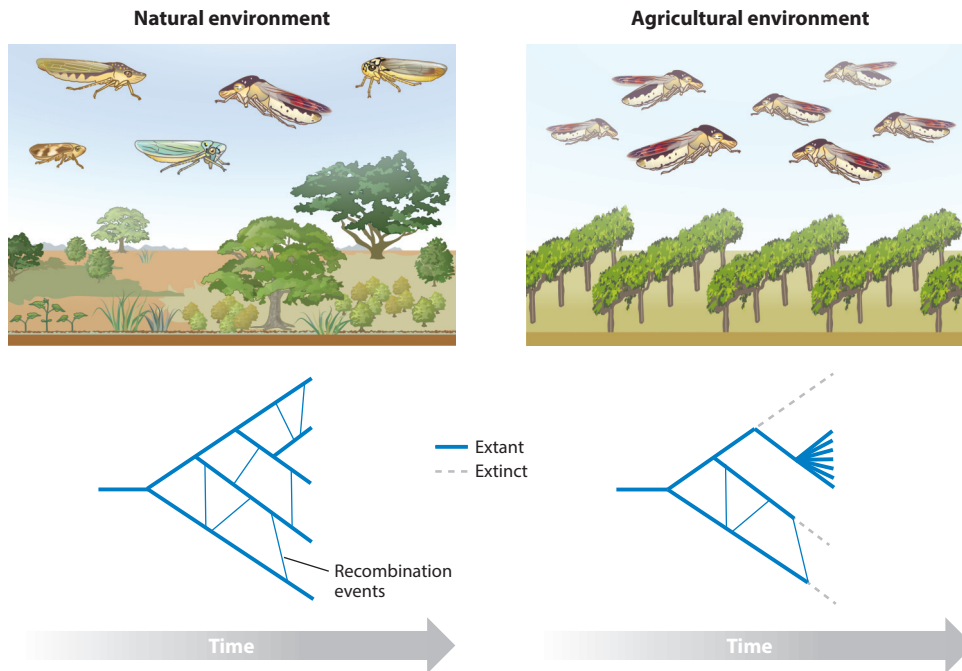
Figure 2

Conceptual model of the ecological pressures on *Xylella fastidiosa* genetic diversity in natural and agricultural settings. In low-diversity crop systems, the pathogen may benefit from clonality and be under selection for more aggressive strains, which would have a higher likelihood of being disseminated from plant to plant. In diverse plant communities, species of polyphagous vectors would transmit strains to various host species; the ecological pressures selecting for a wide host range may limit pathogen virulence for a particular plant species. Accordingly, ecological isolation in agricultural environments may limit the frequency and/or impact of homologous recombination on genetic diversity, resulting in highly specialized *X. fastidiosa* genotypes.

epidemics caused by *X. fastidiosa* broadly harm the provisioning of agroecosystem services in two ways: by reducing crop yield and through nontarget effects from disease management (**Figure 4**). Complete economic accounting of yield losses from *X. fastidiosa*–associated diseases is beyond the scope of this review and has rarely been undertaken. Tumber et al. (141) estimated that PD costs the California grape-growing industry US$104 million per year in reduced yield, management costs, and regulatory costs. This does not include the fraught and difficult accounting of indirect losses due to disease, such as reduced ecosystem services.

The ways in which *X. fastidiosa* reduces agroecosystem services are socially contingent. Our review thus far has focused on the ecological and evolutionary drivers of disease emergence (e.g., host jumping, climate change, and pathogen introduction). However, disease emergence is only one of several events or conditions required for agroecosystem harm. How people and institutions respond to disease emergence mediate the agroecological consequences. This is a particularly difficult problem for *X. fastidiosa* diseases, a technically challenging organism to perform research on, as politically difficult decisions such as eradication efforts must be devised and implemented quickly with limited or incomplete information. For example, *X. fastidiosa* was associated with the olive decline disease in Italy in 2013 (129), but proof of causality was not published until 2017 (i.e., fulfillment of Koch's postulates) (127).
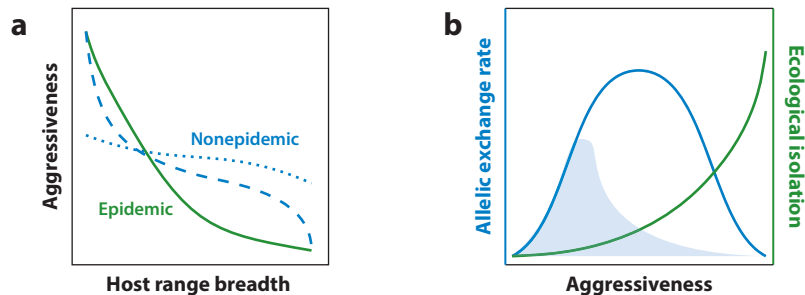
Figure 3

Hypothesized consequence of trade-offs between pathogen genotype aggressiveness and host range on recombination. (*a*) Epidemic *Xylella fastidiosa* genotypes (*green line*) may be highly aggressive to a few host plant species, with mean aggressiveness decreasing sharply as host-range breadth increases. Conversely, nonepidemic genotypes (*blue lines*) may behave more like generalist pathogens, that is, more aggressive but not necessarily pathogenic to a larger number of host plant species. Here, we present two scenarios: one in which the trade-off between aggressiveness and host breadth is strong (*blue dashed line*), and another in which it is much less so (*blue dotted line*). (*b*) Aggressiveness is expected to increase with ecological isolation (i.e., host specialization). As ecological isolation increases, *X. fastidiosa* genotypes encounter and exchange alleles with distinct genotypes less often. Host specialization, in turn, leads to purifying selection and sequence divergence, further reducing the frequency of recombination events. Yet strains that are not aggressive enough do not reach high populations in the landscape, regardless of host breadth, and they have limited opportunities to recombine. Therefore, the frequency of recombination events peaks at an intermediate stage in which ecological isolation is not complete. The shaded area under the allelic exchange rate illustrates the potential for exchange of loci associated with host specialization. These loci may be freely exchanged among isolates that are not host specific but would become recalcitrant to recombination once strains become isolated, as exchange with poorly adapted loci would lead to loss of fitness and purge from the population. Allelic exchange rate is used here as a measure of exchanges that lead to new allele sequences. Exchanges that would lead to the same sequence, which are expected to be common but are not detectable or likely to be ecologically important, are not included.

Reduced crop yield is, by definition, a result of ineffective management of a disease. Although yield loss is clearly difficult to prevent completely, lack of stakeholder cooperation or trust as well as the difficulties political institutions have in implementing required measures can exacerbate ineffective disease management. For example, conspiratorial and pseudoscientific theories on the establishment of *X. fastidiosa*–caused olive quick decline syndrome in southern Italy have persisted through online communication platforms and potentially hampered public support for robust management responses by the government (25). Importantly, lack of cooperation or trust among stakeholders can be exacerbated when management strategies are developed without adequate public deliberation or stakeholder buy-in; methods for building cooperation and trust among stakeholders have been outlined elsewhere (97).

Vector control, including the use of insecticides, may be a necessary and important response to any vector-borne disease, particularly in the early stages of an epidemic. However, even if vector control is effective, it may not always be needed or economically justified. For example, although imidacloprid application to grapevines in Southern California reduced PD incidence slightly when disease prevalence was not high, the cost of application was likely greater than any modest yield gains given the low disease pressure (37). In addition, populations of nontarget organisms may decline owing to insecticide use, with associated reductions in ecosystem services (20). These harms can be further exacerbated by ineffective use or overuse of insecticides; the latter may also lead to vector resistance to pesticides. In turn, the causes of insecticide overuse are not always straightforward; rather, social and economic forces, including technology diffusion and
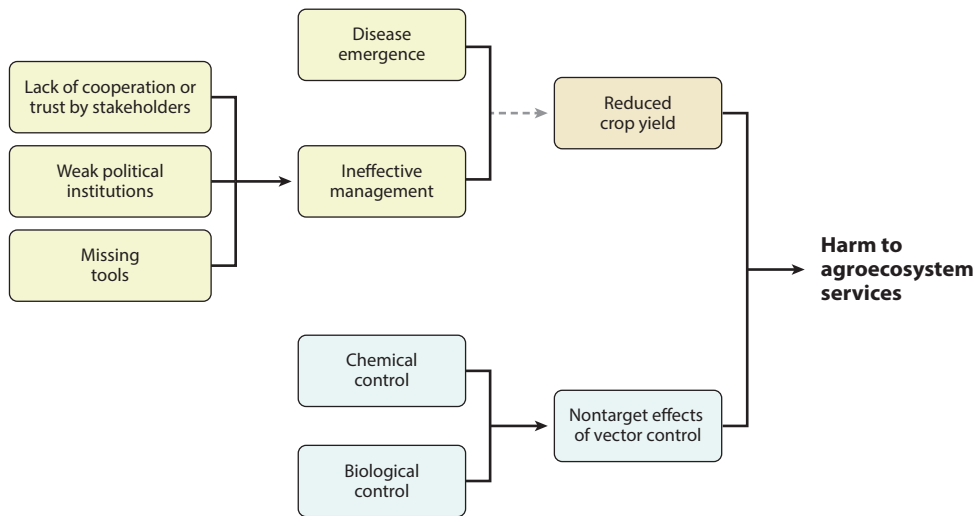
Figure 4

Risk assessment tree (inspired by fault tree analysis) illustrating processes leading to agroecosystem harm caused by *Xylella fastidiosa* diseases. Disease emergence is not the sole reason why *X. fastidiosa* presents a risk to landscapes, and a disease's impacts are mediated by the sociopolitical context. Disease emergence can only cause agroecosystem harm if management is ineffective. In turn, a lack of cooperation or trust by stakeholders, weak political institutions, or missing management tools (e.g., scientific uncertainty and lack of decision-making protocols) can all produce ineffective management. Disease management through vector suppression, using chemical or biological control, can produce harms independent of yield loss, such as nontarget effects. Processes preceded by a closed black arrow can occur through any of the preceding processes (i.e., an OR gate); processes preceded by a dashed grey arrow can occur only through the combination of all proceeding processes (an AND gate).

extension services, market forces, farmer demographics and knowledge, and political institutional constraints, mediate insecticide use (74). Finally, introduced biological control agents aimed at suppressing vector populations as well as new crop genotypes with novel resistance traits could also affect nontarget organisms. However, no evidence exists on the potential importance or prevalence of these impacts for *X. fastidiosa*.

FINAL THOUGHTS

Research on *X. fastidiosa* has been driven by disease epidemics, initially in the United States, then Brazil, and more recently Europe. Accordingly, knowledge has accumulated in leaps rather than incrementally and has focused on a combination of applied disease management priorities and technologies available to scientists. For example, the epidemics in Brazil and the United States at the end of the twentieth century took advantage of genomic tools and led to the development of a thriving community of researchers studying pathogen-plant-insect molecular interactions. However, because *X. fastidiosa* is endemic in the Americas, limited effort has been made to understand patterns of pathogen spread at large (i.e., regional or continental) scales. Similarly, the determinants of pathogen host specificity remain unknown, although this topic may benefit from European epidemics owing to its intrinsic importance for containment and quarantine efforts as well as the now-ubiquitous availability of genomic data.

Accordingly, we have considered *X. fastidiosa* disease emergence in a framework that generates testable hypotheses while identifying current gaps in knowledge or data availability. We are aware

of and acknowledge the difficulty in predicting, for example, how climate change will impact disease prevalence but nevertheless raise points we expect to drive expansion or retraction of geographical ranges. As with many diseases, anthropogenic forces often feature prominently in emergence events of *X. fastidiosa*–associated diseases. Perhaps less clear, however, are the social, economic, and political conditions that mediate the consequences of disease emergence. Finally, we briefly highlight some ways in which the biological aspects of *X. fastidiosa* intersect with such anthropogenic forces to generate disease epidemics and agroecosystem risk. In summary, we hope this review serves as food for thought.

DISCLOSURE STATEMENT

ACKNOWLEDGMENTS

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Contents

Errata

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