Mini Review

Biology, Ecology, and Epidemiology of the Potato Late Blight Pathogen *Phytophthora infestans* in Soil

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Over the 150 years that separate us from the first epidemic of potato late blight in Europe and the onset of phytopathology as a science, *Phytophthora infestans* (Mont.) de Bary has been one of the most extensively studied plant pathogens. It is now commonly perceived as a typical example of an aerial, almost obligate parasite, but the soil stages of its life cycle often have been overlooked. However, sexual or asexual soilborne inoculum is (or could be) significant in three major areas of the biology and pathology of *P. infestans*: (i) survival of the pathogen and production of primary inoculum (19,25,40); (ii) infection of daughter tubers (49, 56); and (iii) variability and evolution of the pathogen (31).

The biology of *P. infestans* in soil was the subject of a number of papers during the late 1800s and early 1900s. The introduction, during the late 1970s, of the A2 mating type to many regions of the world from its original location in central Mexico (29,30,65), giving the fungus the opportunity to produce oospores that could constitute an important source of soilborne inoculum, has recently revived scientific interest in the behavior of the late blight fungus in soil. Thus, the time may be right for a review of the often controversial information available on the ecology and epidemiology of *P. infestans* in soil, proceeding along the three aforementioned areas and providing insights into the current questions and future research needed on these topics.

SURVIVAL AND INFECTIVITY OF *P. INFESTANS* IN SOIL

Both asexual (sporangia, zoospores, mycelium) and sexual organs (oospores), free or associated with potato tubers or debris, may be found in soil at various stages of the *P. infestans* life cycle. Their survival in soil largely conditions primary infections and, therefore, the time and severity of blight outbreaks. Since asexual and sexual organs are largely dissimilar in size, physiology, and resistance to biotic or abiotic stresses, I will focus separately on the survival of these fungal forms in soil before examining their contribution to primary outbreaks of late blight in the field.

Saprophytic survival of asexual inoculum

Three possible sources of asexual, soilborne inoculum of *P. infestans* have been mentioned in the literature: spores produced on foliage that fall to the ground (19), mycelium that survives saprophytically on plant debris (13,21,22), and mycelium and sporangia that survive on infected volunteer or seed potato tubers (20, 50,53).

According to Zan (73), the primary form of asexual inoculum ensuring the survival of *P. infestans* in soil is sporangia, which germinate to produce small, infective mycelia. Mycelium itself can grow readily in sterilized soil or organic debris (21,22,45) but is rarely observed in unsterilized soil (1,45,50,73); it is, therefore, an unlikely candidate for long-term survival of the fungus, contrary to the theory first exposed by Brefeld (13) and later vigorously supported by de Bruyn (21,22). Zan (73) thought that zoospores were too short-lived (approximately 1 day) to be of any significance in survival, but his opinion was questioned by the report of encysted zoospores of *P. palmivora* that survived up to 18 months in soil at intermediate humidity (69). Gregory (37) argued that zoospores, which are diplanetic (i.e., able to reemerge as swimming zoospores after having settled and encysted), may account for infectivity of soil after mycelium has been destroyed. Zoospore survival over several weeks in soil also may provide an explanation for Zan's (73) observation that infectivity could occur in soils in which no mycelium or direct sporangial germination was seen. However, work is still needed to gather evidence implicating zoospores of *P. infestans* in the survival of the fungus in soil.

Soils artificially or naturally contaminated with sporangia remained infective to potato tubers for 15 to 77 days, depending on soil type, moisture content, and pH (1,9,45,50,56,73). As a rule, sandy soils lost their infectious capacity faster than clay or loam soils, and moist soils (i.e., at 20 to 25% of the water-holding capacity) remained infective longer than either water-saturated or dry soils (45,50,73). The shorter persistence of infectivity in sandy soils may be explained by a greater leakage of sporangia and zoospores through the coarse, permeable texture of the soil and by faster drying. Among the different soils studied, very acid soils (pH 3.8 to 4.2) inoculated with *P. infestans* sporangia generally were suppressive to late blight infections (1,9). Low pH was associated with soil suppressiveness to a number of other Phytophthora diseases (5,57); however, in at least one of the soils suppressive to *P. infestans*, pH was not the primary cause of suppressiveness, which was decreased but not eliminated by calcareous amendments raising the pH from 3.8 to 7.6 (2). Germination of *P. infestans* inoculum was lower, and fungal lysis of sporangia was higher in this suppressive soil than in conducive ones (1), suggesting a dual mechanism (fungistasis and inoculum destruction) for suppressiveness. Recent data support the hypothesis of aluminum toxicity, identified as a major determinant of soil suppressiveness to some *Phytophthora* species (7,57), as responsible for fungistasis in this soil (4). On the other hand, the destruction or inhibition of *P. infestans* sporangia in soil by bacteria, actinomycetes, and fungi (1,45) is reminiscent of the microbial antagonism implicated in the suppression of various soilborne Phytophthora diseases (38,47,51,67). Therefore, it is probable that aluminum toxicity and microbial degradation of inoculum act simultaneously, or even complement each other, in suppressive soils.

Further investigations of the microorganisms implicated in suppressiveness, their actual contribution to the phenomenon, and their interaction with Al^{3+} toxicity are needed. A direct approach for assessing the relationships between microbial degradation, fungistasis, and suppressiveness needs to be developed, as Hardy and

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Sivasithamparam (38) showed that morphological changes affecting sporangia incubated in soil (lysis, germination rates, etc.) do not correlate with suppressiveness in *P. drechsleri*. Zan (73) reported that infectivity to potato tubers of soil artificially infested is possible without visible germination of *P. infestans* inoculum. The implication of other potential factors, such as inorganic soil particles (44), in soil suppressiveness to *P. infestans* also should be checked.

Oospore survival in soil

The quest for oospores of *P. infestans*, which de Bary pursued in vain for over 15 years (20), ended in 1911 with the observation of oospores in pure cultures of the fungus (16). Many researchers subsequently reported similar observations, but no one was able to locate these spores in nature before Gallegly and Galindo (33) found them in potato plants grown in the Toluca Valley of central Mexico and demonstrated *P. infestans* heterothallism. The absence of oospores in nature in all regions outside central Mexico was a logical consequence of the presence in these regions of only one of the two mating types (A1), although oospore formation might be induced by interspecific pairings of A1 and A2 strains (8,63). Observations of oospores in pure cultures of A1 isolates of *P. infestans* and possible causes of self-fertility have been reviewed and discussed elsewhere (32,60,61).

After the major worldwide population migration of the mid-1970s, resulting in the introduction of the A2 mating type to Europe and most other potato-producing areas of the world (29,30,65), oospore production in planta under controlled or natural conditions has been reported by several authors (25,27,48). Oospores are generally more abundant in the stems of plants infected with a mixture of A1 and A2 isolates than in the leaves of these plants, presumably because stems survive blight attacks longer than leaves. Because oospores were not present in the field outside the center of origin (central Mexico) of the pathogen until recently, their biology, ecology, and epidemiology remain essentially unexplored. *Phytophthora* oospores are thick-walled resting organs that can survive for very long periods (up to 10 years) in soil (47). Recent work has shown that *P. infestans* oospores are able to survive at least one winter in the field under European conditions (25,54). Survival could exceed 2 years in central Mexico (52), but precise data are lacking on the actual duration of persistence of infectivity in soil.

Origin of primary inoculum and foliage infection

Primary inoculum may be composed of both oospores and sporangia, at least in regions where both types of organs are produced in the field. In most cases, primary inoculum is considered to be exclusively or almost exclusively asexual in origin. Infected tubers, as volunteers surviving the winter in the field, as seed tubers planted in the spring, or as discarded tubers stacked in the field in cull piles, are now regarded as the single major source of asexual primary inoculum. Although cull piles are quantitatively the most important and most effective sources of primary late blight infections (10,11), they cannot be regarded as directly related to *P. infestans* survival in soil and, therefore, will not be considered further in this review.

Following the work of de Bary (19,20), evidence of disease spreading from shoots emerging from blighted tubers has been gathered by a number of workers, and comprehensive reviews are available (14,53,55,71). Nevertheless, nearly all aspects of the life cycle of *P. infestans* in diseased tubers, of the role of such tubers as direct or indirect inoculum sources, and of the mechanisms of transmission of the fungus from tuber to foliage are still controversial. Many studies have revealed a low percentage of germination of severely infected tubers and a low proportion of infected shoots from infected tubers (20,23,39,53,62,70), but reports of high emergence rates from infected tubers also exist (12).

The mechanisms of foliage contamination from infected tubers are not completely elucidated yet. Peterson (53) and Robertson (55) considered the main factor to be spores carried out of the ground with growing shoots, whereas other researchers provided evidence of indirect progression of inoculum from the infected tuber to the soil surface and subsequent infection of lower leaves and stem bases by these spores (12,23,40). Many researchers observed blight symptoms exclusively or predominantly above ground (lower leaves or stem base of the plant) on shoots growing from infected tubers (12,14,23,62), but underground infections also may occur (39,53, 56). *P. infestans* is able to sporulate on infected tubers in the soil (20,56), and propagules (sporangia or, most likely, zoospores) can reach the soil surface if water is available; propagules also may serve as a source of inoculum for infection of daughter tubers.

Oospores constitute a potentially threatening source of primary inoculum in areas in which both mating types occur simultaneously, but the factors and mechanisms inducing oospore germination are still poorly understood. Both abiotic and biotic factors affect oospore germination rates in several *Phytophthora* species (6,26, 41,43,47,57). In *P. infestans*, oospore germination rates in vitro can be increased by treatment with $KMnO_4$ (15), organic manure (64), cellulolytic enzymes (66), and digestion by snails (59); however, germination remains generally scarce (approximately 10 to 15%) and always unpredictable. *P. infestans* oospores surviving in soil are able to infect potato plants, yielding infections at the base of the shoots just above soil level (25,52,54,58). However, the spatial and temporal dynamics of oospore germination in soil, if any, are as yet unknown, as is the existence and level of the threshold of germinating spores needed to ensure successful infections. Therefore, further work is needed before the role of oospores can be adequately assessed and integrated into forecasting models.

INFECTION OF DAUGHTER TUBERS

Sporulation of *P. infestans* on infected tubers in soil (20,56) may provide a source of inoculum for infection of daughter tubers. Sato (56) showed that the infectivity of soil in the direct vicinity of infected tubers increased rapidly after tuber burial, but the contribution of this inoculum to long-term survival of the fungus and to infection of foliage and daughter tubers is still unclear. Direct contact between healthy and infected tubers in soil at times has been considered a possible cause of crop contamination and further rotting in storage (45), but inoculum produced on foliage usually is regarded as the major source of tuber infection (42,49,56).

There is no evidence to date to indicate that oospores may serve as secondary inoculum and infect daughter tubers, although this possibility may exist in areas where oospore populations in soil are large and asexual inoculum from the foliage or mother tubers is scarce. In most cases, asexual inoculum from foliage probably would outnumber sexual soil inoculum to such a large extent that infections of daughter tubers by oospores would be negligible. However, infections originating from oospores may be important if the infective isolates happen to be more pathogenic than the asexual genotypes present at the same time.

EVOLUTIONARY SIGNIFICANCE OF *P. INFESTANS* SOIL STAGES

In all regions of the world outside its center of origin, *P. infestans* has evolved asexually during almost 150 years (34) and has adapted successfully and rapidly to a range of selective pressures (resistance genes, systemic fungicides, etc.) imposed by humans (18,72). Efficient mechanisms exist in *Phytophthora* and other Oomycete genera to create variability in asexual populations. Migrations (29,30), as well as mutations and heterokaryosis (46), have been advocated as major factors accounting for the polymorphisms observed in asexual populations of *P. infestans*. However, these mechanisms are mainly relevant during the aerial, epidemic phase of the disease, and the part played by the soil stages in the evolution and adaptation of *P. infestans* revolves prin-

cipally around oospores (31).

The contribution of sexual inoculum to the structure and variability of *P. infestans* populations usually is assessed using measures of deviation from a Hardy-Weinberg equilibrium. The distribution of mating types and isozyme allele frequencies in central Mexico (33,35) is consistent with such an equilibrium, and race, isozyme, and restriction fragment length polymorphism (RFLP) diversity is greater there than in any other population (3,35). This suggests a major contribution of oospores to fungal inoculum in central Mexico. On the other hand, little evidence of the incidence of sexual inoculum on population structure in other parts of the world has been obtained. Diversity usually is limited and linkage disequilibria high (17,28,35), suggesting that *P. infestans* populations as yet are predominantly clonal outside central Mexico. However, recent observations in the Netherlands (24) show that overall diversity is significantly higher and the distribution of mating types and RFLP genotypes is markedly different in private allotment gardens compared to commercial potato fields, indicating that oospores could be a major factor influencing late blight epidemics and population structure. Evidence of the impact of sexual inoculum on the structure and evolution of *P. infestans* populations in Poland (68) and the United States (36) was obtained recently from the detection of gene flow and increased genotypic diversity using molecular markers. Therefore, it is likely that oospores will noticeably influence the composition and evolution of *P. infestans* populations outside its center of origin in the future, but it is still impossible to forecast the magnitude of this influence.

CONCLUSION

This review shows that, in spite of 150 years of research on the biology and epidemiology of the late blight fungus, more questions about the soil stages of the life cycle of *P. infestans* remain unanswered than have been elucidated. This situation is due in part to the limited geographic area in which oospores could be investigated until recently but also to the inherent difficulties of studying a microorganism in soil. The recent worldwide migration of a diverse group of genotypes and the subsequent population displacement that occurred in most potato-growing areas outside central Mexico, as well as the availability of improved genetic markers, provide new opportunities to learn more about the mechanisms and impact of soil stages of the fungus on late blight epidemiology and also on *P. infestans* population genetics and evolution. The coming decade hopefully will yield a number of studies that will indicate whether a reappraisal of the relative importance of the soil and aerial phases of this destructive and fascinating plant pathogen, and, hence, of our ways of controlling it, is warranted.

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