

Phytophthora sansomeana

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Contributor: Christopher Evan Detranaltes, Jianxin Ma, Guohong Cai

Phytophthora sansomeana has been shown to be a causal agent of Phytophthora root rot (PRR) in soybean in addition to *P. sojae*. The emergence and spread of a second pathogen causing PRR poses a significant threat to soybean production.

Keywords: Phytophthora sansomeana ; Phytophthora root rot ; soybean

1. Introduction

Since the 1990s concern has been rising about a new causal agent of Phytophthora root rot (PRR) in soybean across the Midwest. *Phytophthora sojae* (Kaufm. and Gerd.) has long been attributed as the cause of PRR in soybean ^[1]. However, in 1990 an outbreak of what was initially thought to be *P. sojae* (then *P. megasperma* (Drechs.) f. sp. *glycinea* (T.L. Kuan and Erwin)) occurred in Indiana ^[2]. Although it is morphologically similar to *P. sojae*, the pathogen failed to produce race-classifiable results when inoculated on a set of *P. sojae* differentials. In 2009, the pathogen isolates from soybean, as well as isolates from Douglas-fir in Oregon and wild carrot, white clover, and white cockle in New York, were recognized as a novel species designated *Phytophthora sansomeana* (E.M. Hansen and Reeser) based on a combination of morphological observations, host interactions, and molecular phylogenetics ^[3]. Isolates from soybean, Douglas-fir, and a few agricultural weeds long suspected to be distinct species from the *P. megasperma sensu lato* clade were all reidentified as belonging to this species.

2. Disease Symptoms and Pathogenicity

2.1. On Soybean

It is now known that the causal agent of PRR outbreaks in soybean may be either *P. sojae* or *P. sansomeana*; how much *P. sansomeana* contributes relative to *P. sojae* in causing PRR epidemics and at what growth stages in soybean remain unknown. When first reported in Wisconsin, *P. sansomeana* was recovered from 49 symptomatic plants (V1–V4) at a rate of only 6% ^[4]. In some Illinois counties, however, *P. sansomeana* (referred to as an unknown *Phytophthora* species ^[5]) was recovered from soil baiting assays as often as 70% of the time ^[6]. Whether all these populations were contributing to PRR is unclear, however fields from which *P. sansomeana* was recovered had histories of seedling disease and at least some of the isolates were pathogenic when inoculated on *P. sojae* differentials. Whether *Rps* genes conferring resistance to *P. sojae* are ineffective at providing resistance to *P. sansomeana* is likely ^{[2][6]} but has not been thoroughly tested.

In the field, distinguishing the two pathogens can be difficult. Late in the growing season, *P. sojae* typically causes a necrosis of the stem that emerges from the soil line and spreads upward after the seedling stage in soybean ^[5]. *P. sansomeana* appears to primarily cause wilting as an aboveground symptom with the stem lesion usually absent in infected hosts ^[3]. An accurate discernment of the two pathogens from field symptoms alone is unlikely before the onset of stem lesion formation and may remain impossible without further diagnostic evidence. There may also be potential for emergent effects from double infection with both *P. sojae* and *P. sansomeana*. Rojas et al. reported the discovery of two field samples testing positive for both species in PCR based assays. They noted that *P. sansomeana* mostly concentrated in the tap roots while *P. sojae* preferentially colonized the lateral roots in both cases ^[7]. Double infection in combination with *P. sojae* may reduce the usefulness of stem lesions as a pathognomonic symptom.

P. sojae is also known to be a potent seed and seedling pathogen implicated in pre-emergence and post-emergence damping off long before the stem lesion symptom is produced. The relative contribution that *P. sansomeana* may make during *Phytophthora*-induced seedling disease outbreaks in the field is unknown. Nevertheless, seed plate assays suggest that *P. sansomeana* causes significantly more severe seed rot scores than *P. sojae* ^[8]. It is therefore possible that *P. sansomeana* plays a greater role as a seed pathogen than in adult plant infections, which could help explain its

relatively low isolation rate from symptomatic hosts later in the season ^[4] despite high frequency of recovery in soil baiting assays ^[6]. The increased availability of conventional and real-time PCR protocols that differentiate between the two species may help to disentangle the relative contributions of each pathogen to PRR ^{[9][10]}

2.2. On Non-Soybean Hosts

On trees *P. sansomeana* causes root and collar rot symptoms of varying severity depending on the host. In conifer species infected trees typically become progressively more chlorotic, stunted, and may die off as infections age ^[11]. In *Abies* species branch flagging and wilting of the younger shoots was observed ^[12]. In Rosaceous trees, fruit production may be stunted or ceased entirely ^[13]. On maize *P. sansomeana* causes wilting and stunting and may be related to emergence problems ^[14], though interestingly, reports from this host lag behind that of soybean in the U.S. and globally. *P. sansomeana* was also recently isolated from leaf lesions in taro ^[15]. Artificial inoculation yielded the same lesions as seen on field hosts on detached leaves. It appears that *P. sansomeana* infects belowground or aboveground tissues on a host-by-host basis.

3. Genetic Variability

The mitochondrial genome of *P. sansomeana* has been sequenced and annotated ^[16]. Interspecific comparison showed genes of similar structure and order as *Phytophthora* species selected from clades 1, 7, 8, and 9, including conserved respiratory complex, ATP synthase, and ribosomal RNA protein genes. The genome closely resembles that of *Phytophthora ramorum* (clade 8c), differing only in an inversion of the section covering *nad5*, *nad6* and *trnR_{UCU}*. Other members of clade 8 or subclade 8a were not included in the analysis. *P. sansomeana* carries in its mitochondrial genome a unique ORF encoding an unknown protein of 402 amino acids in length not seen in the 10 other *Phytophthora* species included in the analysis. An annotated nuclear genome sequence is in preparation (Cai, unpublished data).

Intraspecific variation in the ITS sequence has been observed across populations of *P. sansomeana*. Hansen et al. reported that isolates of *P. sansomeana* were polymorphic at eight loci in the 826 bp long sequences analyzed between isolates from weedy hosts in New York and isolates from soybeans from the Midwest ^[3]. They noted that isolates from Douglas-fir may have been the result of hybridization between these two populations due to the presence of double peaks in sequence chromatogram that resembled both populations. Whether all *P. sansomeana* strains infecting Douglas-fir are a result of hybridization is unclear, since isolates recovered from weedy-type parent population also readily cause disease when inoculated on Douglas-fir just the same as apparent hybrid strains ^[3].

The species also appears to readily hybridize with other members of *Phytophthora* subclade 8a. Hybrids have been identified from Iran and Bulgaria with *P. sansomeana* acting as the paternal parent in both cases of suspected sexual recombination with the species *P. pseudocryptogea* and *P. kelmania*, respectively ^{[17][18]}. The two hybrids with *P. pseudocryptogea* were isolated from beet, while the hybrid with *P. kelmania* was recovered from *Rhododendron* leaf baits in a freshwater river. All three hybrids produced only asexual sporangia and neither oogonia nor antheridia. Hybrids otherwise shared morphological features of both parents. *P. sansomeana* has been reported to be pathogenic to maize, however the hybrid of *P. sansomeana* and *P. kelmania* was unable to infect maize seedlings, indicating that hybridization may change host specificity or that pathogenicity on maize may be strain specific in *P. sansomeana* ^[17]. Interestingly, in both cases where hybrids have been isolated, *P. sansomeana* has yet to be reported, indicating either that it has been present and so far unreported or that the hybrid originated elsewhere and was transported to the region.

4. Disease Management

Research on the control of *P. sansomeana* is still emerging and several studies have investigated genetic resistance in soybean and chemical control in lab studies. Two small effect quantitative trait loci (QTLs) were found to confer partial resistance to *P. sansomeana* in soybean ^[19]; these two loci showed isolate specificity when tested against seven isolates including one isolate that overcame both QTLs. The authors noted that only one replicate of inoculated trials could be completed per isolate due to seed limitations, so whether this is evidence for a potential virulence/avirulence structure or an artifact of environmental fluctuations between tests is uncertain. Biocontrol measures and other non-chemical management strategies are unstudied in reference to this pathogen.

Seed treatment was found to significantly reduce root rot in soybean seed plate assays after inoculation with *P. sansomeana* ^[8]. Soybean seeds treated with combinations of metalaxyl and ethaboxam, pyraclostrobin and metalaxyl, or mefenoxam alone significantly reduced disease severity compared to nontreated controls. Importantly, these treatments were also effective against *P. sojae* suggesting one seed treatment formulation could control both pathogens. Previous

findings of metalaxyl sensitivity in *P. sansomeana* corroborate the susceptibility of this species to this chemical control measure [20]. *P. sansomeana* isolates recovered from corn are similarly susceptible to both ethaboxam and mefenoxam [21]. Seed treatment may therefore be a useful tool in managing *P. sansomeana* associated losses while genetic resistance and other components of integrated pest management are investigated and developed.

Although both species cause PRR in soybeans, it is unclear if *P. sojae* control measures aside from seed treatment will similarly manage *P. sansomeana* due to fundamental differences in host specificity and the absence of typical race reactions on soybean differentials for *P. sojae* resistance. For instance, crop rotation with corn may be ineffective in reducing primary inoculum available for soybean infections due to the ability of *P. sansomeana* isolates to infect maize [14]. Further, *Rps* genes conferring resistance to *P. sojae* appear to provide no protection against *P. sansomeana*.

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