Impact of White Pine Blister Rust on Resistant Cultivated *Ribes* and Neighboring Eastern White Pine in New Hampshire

Isabel A. Munck, Northeastern Area State and Private Forestry, United States Department of Agriculture Forest Service, Durham, NH 03824; **Philippe Tanguay**, Canadian Forest Service, Laurentian Forestry Centre, Sainte-Foy, QC, G1V 4C7, Canada; **Jennifer Weimer**, New Hampshire Department of Resources and Economic Development, New Hampshire Division of Forests and Lands, Concord 03302; and **Sara M. Villani** and **Kerik D. Cox**, Department of Plant Pathology and Plant-Microbe Biology, New York State Agricultural Experiment Station, Cornell University, Geneva 14456

Abstract

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To determine the impact of white pine blister rust (WPBR) following the recent breakdown of the *Cr* resistance dominant gene in cultivated *Ribes* spp., 255 plants of 19 *Ribes* cultivars and 445 neighboring eastern white pine (*Pinus strobus*) from 42 sites across New Hampshire were evaluated. Of the 19 *Ribes* cultivars evaluated, 15 were WPBR resistant, and 4 of these were labeled as black currant (*Ribes nigrum*) with the *Cr* gene (*Cr Ribes* cultivars). Incidence of WPBR ranged from 0 to 88% for WPBR-resistant *Ribes* cultivars. Mean WPBR severity was 14 and <6% of leaf area for *Cr Ribes* and partially resistant cultivars, respectively. The presence of *Cronartium ribicola* was confirmed on 17 of the 19 *Ribes* cultivars screened with polymerase chain reaction

analysis and DNA sequencing. Reference accessions of *Cr Ribes* cultivars from the Canadian Clonal Genebank were successfully infected with *C. ribicola* inoculum collected in New Hampshire from *Cr Ribes* cultivars and *P. strobus*, confirming that the vCr race of *C. ribicola* that has overcome the *Cr* resistance dominant gene in cultivated *Ribes* spp. is present in New Hampshire. The probability of finding pine trees with WPBR was greater for trees neighboring infected *Cr Ribes* cultivars (0.18) than trees neighboring WPBR-free *Ribes* cultivars (0.02). Results from this study suggest that the breakdown of *Cr*-based resistance in *Ribes* spp. poses a threat to the white pine resource and to cultivated *Ribes* production.

The causal agent of white pine blister rust (WPBR), *Cronartium ribicola* J. C. Fisch. 1872, has been a threat to both forest resources and agricultural commodities since its introduction to North America in the early 1900s (Ostry et al. 2010; Posey and Ford 1924; Stewart 1906). *C. ribicola* has a complicated lifecycle, alternating between five-needle pine and telial hosts such as currant or gooseberry in the genus *Ribes* (Spaulding 1922a). In the spring, aeciospores produced in pine infect alternate *Ribes* hosts (Geils et al. 2010; Spaulding 1911). Aeciospores are thick walled and pigmented (Kasanen 1997), and are capable of traveling in wind currents for thousands of kilometers (Mielke 1943). Urediniospores, produced on the underside of *Ribes* leaves, mainly from late May to early September, locally infect other *Ribes* plants, thus intensifying the spread of the pathogen among *Ribes* hosts (Geils et al. 2010). Before leaf fall, telia differentiate, and meiosis in teliospores produces basidiospores that infect white pine. Basidiospores are relatively fragile and cannot travel long distances (Zambino 2010). Except in the case of early defoliation, where WPBR has been shown to reduce fruit yield (Pedersen 1998, 2008), the disease is generally not considered a serious economic problem to the *Ribes* industry. However, plant regulatory measures taken to protect the white pine resource could influence *Ribes* cultivation. In contrast, infection by *C. ribicola* in white pine is devastating to pine production and may ultimately lead to tree mortality (Luffman 2000). Indeed infection of pine trees can result in girdling cankers and tree death (Geils et al. 2010; Kearns and Jacobi 2007; Lachmund 1933; Slipp 1953). Young trees die more rapidly because it takes less time for the pathogen to completely girdle their main stems (Geils et al. 2010) but all size classes are killed by WPBR (Martin 1937).

Eastern white pine (*Pinus strobus* L.) is one of the most ecologically, historically, and economically important tree species in eastern North America. White pine provides unique terrestrial habitat elements for wildlife that other large conifer species cannot duplicate (Yamasaki 2003). White pine composes the greatest portion of the growing stock (volume of all living trees in a given area) in Massachusetts (24%), followed by New Hampshire (21%) and Maine (10%) (Widmann and McWilliams 2003). Eastern white pine is also valued in the ornamental and nursery trade (Bergdahl and Teilon 2000). In 2013, the contribution of forest-based manufacturing and forest-related recreation and tourism to the economy in Maine, New Hampshire, Vermont, and New York was over \$33 billion (NESFA 2013). The white and red pine forest type covers approximately 1.25 million ha (10%) of New England's timberland and contributes to the aesthetics sought after by tourists and recreationists.

WPBR was introduced to the United States from Europe more than a century ago via imported plant material (Maloy 1997; Posey and Ford 1924; Spaulding 1922a). The absence of coevolution between the invasive pathogen and its aecial hosts, in addition to the establishment of native white pine and *Ribes* spp. throughout the eastern and western United States, provided a favorable environment for WPBR epidemics (Dalton et al. 2010; Smith et al. 2006). Early management of the disease focused primarily on elimination of its alternate host via widespread *Ribes* eradication. To protect the white pine resource, *Ribes* spp. were quarantined and destroyed throughout the United States from 1917 to 1967 by the Blister Rust Control Program (Benedict 1981; Maloy 1997). Removing the alternate host has long been used to control WPBR because *C. ribicola* must cycle through *Ribes* spp. in order to infect white pine, and delicate basidiospores have a limited infective range (Zambino 2010). These efforts continued for more than 40 years but they were abandoned for a variety of reasons, including increased costs, labor shortages, interagency squabbling, and difficulty in removing certain native *Ribes* spp., especially in the West, where pine stands grew in inaccessible and rugged terrain (Maloy 1997). More recently, fungicide management to control WPBR on susceptible cultivated *Ribes* spp. has demonstrated effectiveness in reducing

Corresponding author: I. Munck; E-mail: imunck@fs.fed.us

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but not eliminating initial urediniospore inoculum in small-scaled research *Ribes* operations (Cox et al. 2008).

Interest in *Ribes* cultivation in the United States increased in the late 1990s, sparking an inquiry by foresters, pathologists, horticulturists, and regulators to determine the threat of *Ribes* spp. to pine (McKay 2000). The results of the inquiry were varied, and state regulatory agencies differed in their wish to change *Ribes* regulations. Several knowledge gaps arose, including the need to assess incidence of WPBR on pine and the longevity of WPBR resistance in *Ribes* cultivars (McKay 2000). To help regulators make informed decisions and quantify whether WPBR incidence on eastern white pine had changed since the 1900s, a statewide survey was conducted in New Hampshire during 1998 (Lombard and Bofinger 1999). Survey results indicated that WPBR incidence had decreased from 20 to 80%, reported in the early to mid-1900s, to a range of 0.3 to 7.2%, depending on the region (Lombard and Bofinger 1999). Field trials conducted during 1995 and 1996 at the United States Department of Agriculture–Agricultural Research Service (USDA-ARS) National Clonal Germplasm Repository in Oregon identified several WPBR-resistant *Ribes* cultivars (Hummer 1997). As a result of these findings, New Hampshire changed its regulations in 2002 to allow planting of resistant *Ribes* cultivars. By 2009, 19 WPBR-resistant *Ribes* cultivars were available for planting, including 4 European black currant cultivars with the *Cr* dominant resistance gene from *Ribes ussuriense* (*Cr Ribes* cultivars) (Hummer and Finn 1999). None of the cultivars with the *Cr* dominant resistance gene or partial resistance approved for planting in New Hampshire produced uredia after exposure to natural infection in Oregon field trials (Hummer 1997; Hummer and Finn 1999). Additionally, most cultivars did not produce uredia after artificial inoculation with *C. ribicola* urediniospores (Hummer and Picton 2002).

In 2011, WPBR incidence was reported on a *Cr Ribes* cultivar, *R. nigrum* ‘Titania’, in Connecticut (Frederick et al. 2011). A preliminary survey in 2012 indicated the presence of WPBR on ‘Titania’ and other cultivars planted in New Hampshire. These findings caused concern to regulators and forest health managers in New Hampshire, who decided that a more complete assessment of *Ribes* plantings and the impact on local white pine was needed. The objectives of this study were to (i) evaluate WPBR incidence and severity on WPBR-resistant *Ribes* cultivars, (ii) determine WPBR incidence and severity on neighboring eastern white pine and wild *Ribes* spp., (iii) verify presence of *C. ribicola* with molecular diagnostic techniques, and (iv) challenge *Ribes* spp. with *Cr*-based resistance of reliable provenance from the Canadian Clonal Genebank with New Hampshire *C. ribicola* samples.

Materials and Methods

Site selection. A permit specifying location, cultivar, and number of plants must be submitted to the State’s Division of Forests and Lands (NHDFL) to plant *Ribes* spp. in New Hampshire. In total, 43 sites out of 109 permit holders were visited from 31 July to 4 September 2013, when *C. ribicola* uredia or telia were visible on the lower leaf surface of *Ribes* plants and before leaf drop. Thirty-eight sites were randomly selected to assess the incidence and severity of WPBR. An additional five sites were surveyed to obtain descriptive data for *Ribes* cultivars rarely planted in New Hampshire.

***Ribes* sampling in New Hampshire.** Field surveys and collections were conducted by the U.S. Forest Service (USFS) and NHDFL. At each site, disease incidence and severity were assessed for up to five plants per cultivar. The disease severity rating scale developed for grapevine rust (Angelotti et al. 2008) was used to estimate percentage of diseased leaf area on the lower side of leaves for 18 leaves per plant: 3 leaves from the top, middle, and base of each of two randomly selected canes. Plants were considered positive for WPBR if any of the 18 leaves inspected to assess disease severity or any other leaves had *C. ribicola* signs on their lower surfaces. The same methods were used to assess disease incidence and severity for wild or naturalized *Ribes* spp. found in the vicinity of cultivated *Ribes* plants or neighboring white pine trees. At each site, infected leaves from each cultivar were placed in labeled paper envelopes that were pressed in a plant

press until they could be mailed to Cornell for polymerase chain reaction (PCR) analyses. Infected leaves from *Cr Ribes* cultivars ‘Coronet’, ‘Consort’, ‘Crusader’, and ‘Titania’ were collected in the same manner and mailed to Canada for infection assays.

Confirmation of pathogen identity. Pathogen diagnoses were conducted by Cornell University. At each site, composite samples comprising 10 to 33 *Ribes* leaves with putative WPBR pustules were collected from each cultivar and stored at 7°C for genetic confirmation of *C. ribicola*. Prior to amplification of rust DNA using PCR, the rust uredinia, urediniospores, or telia were isolated from leaf tissue by repeatedly pressing a 1-by-1-cm square of single-sided, matt finish, Scotch Magic Tape (3M, St. Paul, MN) lightly to the surface of currant leaves with evidence of rust infection. The tape pieces were subsequently placed in individual 2.0-ml microcentrifuge tubes containing a sterile bead and were dipped in liquid nitrogen for 30 s. Fungal tissue was mechanically disrupted using a TissueLyser System (Qiagen, Germantown, MD), and DNA was extracted using a DNeasy Plant Mini Kit (Qiagen) according to the manufacturer’s instructions. To confirm the identity of *C. ribicola*, primers 15337AG (5′-TTT GTG GAC TTG CAG GTT TG-3′) and 15337AF (5′-CGG ACC AAC TTT CCT AGT CG-3′) for a *C. ribicola* gene encoding a protein with homology to phenol 2-monoxygenase were used to generate an approximately 660-bp fragment from genomic DNA (Brar 2012). PCR reactions were made in 25-μl reaction volumes and contained 1× PCR buffer, 0.4 μM each primer, 2.5 mM MgCl₂, 200 μM each dNTP, 0.625 U of GoTaq Flexi DNA polymerase (Promega Corporation, Madison, WI), and 5 ng of extracted DNA. Amplifications were performed using an iCycler thermal cycler (Bio-Rad Laboratories Inc., Hercules, CA) with the following cycling protocol: 3 min at 94°C; 30 cycles of 30 s at 94°C, 30 s at 50°C, and 1 min at 72°C; followed by a final extension step of 10 min at 72°C. PCR products were separated on a 2.0% agarose gel (Bio-Rad Laboratories Inc.) stained with ethidium bromide in 1× Tris-acetate-EDTA buffer at 100 V for 1 h. Photographs of the gel were taken on a KODAK Gel Logic 200 Imaging System (Eastman Kodak Company, Rochester, NY). A previously confirmed isolate of *C. ribicola* was included each time PCR was conducted in order to detect false-negative results. For a subsample of isolates, additional genetic confirmation was accomplished by sequencing the two internal transcribed spacer (ITS) regions and the 5.8S gene in the nuclear ribosomal repeat using primers ITS1-F and ITS-4, as described previously (Gardes and Bruns 1993).

White pine sampling in New Hampshire. Field surveys and collections were conducted by USFS and NHDFL. During July and September 2013, the 12 closest pine trees within 300 m of cultivated *Ribes* spp. (3 trees in each of the four cardinal directions, if available) were examined for WPBR signs and symptoms at each site. WPBR symptoms included aecial scars, flagging, spindle-shaped cankers, bark discoloration, resinosis, and crown discoloration. A distinction was made between pine with aecial scarring and pine with other symptoms. Distance and cardinal direction from cultivated *Ribes* spp. were determined with the aid of a GPS receiver (GPSmap 60CSx; Garmin International Inc., Olathe, KS) and a compass. Stem diameter at breast height (dbh; 1.3 m above the ground) was recorded for each pine.

During April and May 2014, sites with pine trees containing aecial scars in sites with plants labeled as *Cr Ribes* cultivars infected by *C. ribicola* in August 2013 were revisited to collect aeciospores. At each site, pine trees with aecia within 300 m of *Cr Ribes* cultivars were sampled. Aeciospores were collected from single unruptured aecia by carefully ripping the peridium with a toothpick and inserting the spores into an Eppendorf tube. Up to 10 aecia were sampled per canker, working from the bottom of the canker upward to avoid contamination. Spores from one aecium represented a sample. Samples from up to three cankers per tree were collected. Samples were brought to the laboratory and kept at 4°C until they were shipped to Canada for infection assays within 3 days of their collection.

Plant material for infection assays. Infection assays were conducted by the Canadian Forest Service. The susceptible black currant

control *R. nigrum* 'Ben Lomond' was bought at a nursery in Quebec City. Cuttings from the black currant accessions with the *Cr* gene ('Consort', 'Crusader', and 'Coronet', with accession numbers CN102541, CN102560, and 102561, respectively) were kindly provided by the Canadian Clonal Genebank (Agriculture and Agri-Food Canada) located at the Harrow Research Station (Ontario, Canada). Dormant cuttings were stuck in Rootcubes (Oasis Grower Solutions) and rooted using a hydroponic flood table system (American Hydroponics). Rooted cuttings were transferred into a mix of peat moss, vermiculite, and turf (ratio 4:2:1) and grown under greenhouse conditions with a photoperiod of 16 h/day and 8 h/night at 24 and 20°C, respectively.

Infection assay of *C. ribicola* on detached *Ribes* leaves. Rust from New Hampshire field collections was pathotyped on reference accessions of *Cr Ribes* cultivars 'Consort', 'Coronet', and 'Crusader' from the Canadian Clonal Genebank. Presence of the vCr race of *C. ribicola* that has defeated the *Cr* resistance dominant gene in cultivated *Ribes* spp. was determined by using controlled leaf inoculations of reference accessions of *Cr Ribes* cultivars with urediniospores or aeciospores collected from infected leaves of plants labeled as *Cr Ribes* cultivars in New Hampshire or from neighboring white pine cankers, respectively. Monouredinal *C. ribicola* strains were propagated on detached leaves from the susceptible black currant control 'Ben Lomond' (without the *Cr* gene) and reference accessions *Cr Ribes* cultivars from the Canadian Clonal Genebank (with the *Cr* gene). *Ribes* leaves from leaf plastochron index 6 to 10 were removed from the plant, and pieces from each susceptible and *Cr Ribes* cultivar were placed side by side onto a wet paper towel inside petri dishes. Sterile cotton swabs were used to inoculate leaf abaxial surfaces with the urediniospores or aeciospores collected in New Hampshire from infected leaves of plants labeled as *Cr Ribes* cultivars or pine cankers, respectively. Petri dishes were incubated at 19°C under a 16-h photoperiod and 80% relative humidity. Bulk aeciospores preserved at -80°C and collected in 2008 from cankers in a white pine plantation in Saint-Isidore (Quebec, Canada) were used for negative controls. Leaf pieces were inoculated within 15 and 4 days of uredial and aecial sample collection, respectively. Pathogen growth was assessed 4 weeks postinoculation.

Statistical analyses. Only data from randomly selected sites and state-approved WPBR-resistant *Ribes* cultivars were used for analyses. *Ribes* plants were grouped by species because of the lack of repetition in each cultivar. A distinction was made between *Cr Ribes* cultivars ('Coronet', 'Consort', 'Crusader', and 'Titania') and 'Willoughby', a cultivar of unknown background selection of *R. nigrum*. To determine the effect of *Ribes* sp. on the incidence of infected *Ribes* plants, data were analyzed using a generalized linear mixed model (PROC GLIMMIX) in SAS (v. 9.3, 2011; SAS Institute Inc., Cary, NC). "Species" was a fixed effect, "site" was a random factor, and the binomial distribution with the logit link function for the response variable "proportion of plants with WPBR" was used. To establish the effect of *Ribes* sp. on WPBR severity on *Ribes* plants, data were analyzed using a linear mixed model (PROC GLIMMIX). "Species" was a fixed effect, "site" was a random factor, and the response variable was "mean percentage diseased leaf area per infected plant".

To explore the effects of variables associated with disease development on WPBR incidence of white pine, data were analyzed using a generalized linear model (PROC GLIMMIX) specifying binomial distribution and the logit link function for the response variables "proportion of pine with aecial scars" or "proportion of pine with aecial scars OR other symptoms". The response variable "proportion of pine with aecial scars" represents a more conservative estimate of WPBR incidence. Analyses were conducted separately for each of these two response variables. The effects of each of the following variables were individually tested: presence of infected *Ribes* spp. with and without *Cr* gene, tree size, distance within 300 m from *Ribes* plants, and cardinal direction from *Ribes* plants. Data from 26 sites with infected *Ribes* spp. only were used for analyses to determine the effect of distance and cardinal direction from infected *Ribes* plants on incidence of infected pine. For all models, when main effects were

significant ($\alpha = 0.05$), a Tukey-Kramer test was used to identify differences among means.

Results

The impact of WPBR on 255 plants of 19 *Ribes* cultivars and 445 neighboring white pine trees from 43 sites was evaluated. Only three sites were managed for commercial *Ribes* production and the remaining locations were private gardens. The mean number of *Ribes* plants grown by commercial growers and private homeowners was 115 and 5, respectively. Most sites (76%) had *Ribes* cultivars infected by *C. ribicola* (Fig. 1). All but two sites had pine trees neighboring cultivated *Ribes* plants. Symptomatic pine trees were observed at most sites (63%) but fewer sites (37%) had trees with aecial scars.

WPBR incidence and severity in *Ribes* spp. surveyed in New Hampshire. WPBR incidence and severity data were obtained for five species and a hybrid for a total of 19 cultivars, of which 15 were on present or previous state-approved lists (Table 1). North American *Ribes* spp. (*R. oxycanthoides* L. and *R. odoratum* H. Wendl.), European *Ribes* spp. (*R. nigrum*, *R. rubrum* L., and *R. uva-crispa*), and a currant × gooseberry hybrid (*Ribes* × *nidigrolaria* Bauer) were represented in the field in New Hampshire. In addition, incidence and severity data were collected for the following four cultivars not approved for planting in the state of New Hampshire: 'Red Lake', 'Blanka', 'Invicta', and 'Pixwell'. 'Titania' was the most frequently planted cultivar (Table 1).

Naturalized and wild *Ribes* spp. were found in only two sites. One location had three naturalized *R. rubrum* plants growing in the woods next to the cultivated *Ribes* sp. Two of these three plants were infected by *C. ribicola* and the mean percent diseased leaf area for these plants was 0.1%. Another location had *R. hirtellum* Michx. growing on an old stone wall surrounding the property. This plant was infected and its mean percent diseased leaf area was 2%. Disease severity of the wild and naturalized *Ribes* spp. was less than the disease severity of *Cr Ribes* cultivars 'Consort', 'Coronet', and 'Titania' and comparable with disease severity of other WPBR-resistant cultivars (Table 1).

Data from the 38 randomly selected sites only was used for comparisons of WPBR incidence and severity among *Ribes* spp. For incidence and severity analyses, observations from 220 and 103 plants, respectively were used. Except for *Cr Ribes* 'Crusader', which was not infected in the field, incidence of WPBR on other *Cr Ribes* cultivars ranged from 19 to 60% by cultivar, suggesting that these plants are either no longer resistant or possibly mislabeled (Table 1). Incidence of WPBR exceeded 50% for most cultivars (10 of 15).

Ribes spp. differed in their susceptibility to WPBR. The main effect of *Ribes* spp. on WPBR incidence ($P = 0.0024$) and severity ($P < 0.0001$) was statistically significant. Plants in the species *R. oxycanthoides*, 'Jahns Prairie' gooseberry, were most likely to be diseased (predicted probability of disease = 0.88; Fig. 2). In contrast, *R. rubrum* cultivars, red and white currants, were least likely to be diseased (predicted probability = 0.25). In comparison, a third of *Cr Ribes* cultivars were predicted to be diseased. *Cr Ribes* cultivars exhibited the greatest percentage of diseased leaf area (15%) compared with other *Ribes* spp. (0.1 to 6%; Fig. 3). *R. rubrum* cultivars had the lowest percentage of diseased leaf area (0.1%).

Confirmation of pathogen identity. Amplification of fungal DNA with primers 15337AF and 15337AR and sequencing of the ribosomal DNA ITS1, 5.8S, and ITS2 regions confirmed the presence of *C. ribicola* in all counties in New Hampshire and *Ribes* cultivars sampled in this study (Fig. 1; Table 1). With the exception of the *Cr Ribes* 'Crusader' and white currant '1301', samples from all other cultivars allowed in New Hampshire were PCR positive for *C. ribicola*, including *Cr Ribes* 'Consort', 'Coronet', and 'Titania' (Table 1).

Infection assay of *C. ribicola* on detached *Ribes* leaves. From the last week of July to the first week of September 2013, 15 different plants labeled as *Cr Ribes* cultivars showing signs of WPBR were collected from 12 different sites within 113 km of each other in southern New Hampshire. Urediniospores from 6 of the 15 *Cr Ribes* samples collected in New Hampshire from six different sites, all from plants labeled 'Titania', successfully infected detached leaves of reference accessions of *Cr Ribes* cultivars from the Canadian Clonal Genebank (Table 2). Uredia appeared and developed at the same

speed on *Cr Ribes* cultivars and on the susceptible black currant control 'Ben Lomond' (without the *Cr* gene). During April and May 2014, aeciospores from 328 monoaecia were collected from 45 pine cankers in 10 different sites where infected pine trees were growing next to plants labeled as *Cr Ribes* cultivars with WPBR in August 2013. Aeciospores from 36 aecia from six cankers from a total of five pines in two locations 32 km from each other in New Hampshire

successfully infected detached leaves of reference accessions of *Cr Ribes* cultivars from the Canadian Clonal Genebank (Table 3). Lack of infection for most of the aeciospores collected can most likely be attributed to nonviability at time of inoculation because these aeciospores also failed to infect leaves from the susceptible 'Ben Lomond' control. Bulk aeciospores collected in 2008 from cankers in a white pine plantation in Saint-Isidore (Quebec, Canada) used for negative

Fig. 1. Distribution of sites surveyed in New Hampshire to determine the impact of white pine blister rust on cultivated *Ribes* plants and neighboring white pine trees. Sites positive for *Cronartium ribicola* had infected cultivated *Ribes* spp. The identity of the pathogen was confirmed with molecular diagnostic techniques.

controls successfully infected 'Ben Lomond' leaves but were avirulent on leaves from black currants with the *Cr* gene. These results show that the vCr race of *C. ribicola* that has overcome the *Cr* resistance dominant gene in cultivated *Ribes* spp. is present in both cultivated *Ribes* plants and white pine in New Hampshire.

WPBR incidence in white pine. Of the 385 pines from the 38 randomly selected sites, 37 pines had aecial scars and 74 had aecial scars or other WPBR symptoms such as resinosis, spindle-shaped cankers, flagging, or bark discoloration. Presence of infected *Ribes* plants influenced incidence of WPBR on white pine ($P < 0.01$). White pine trees with WPBR were found more frequently next to Cr *Ribes* cultivars infected with WPBR (probability = 0.18) than next to other WPBR-resistant *Ribes* cultivars infected with WPBR (probability = 0.09) or next to *Ribes* without WPBR (probability = 0.02) (Fig. 4). Distance within 300 m from infected *Ribes* plants also had a significant effect on WPBR incidence in pine, but only when pine trees with aecial scars were included in the analyses ($P = 0.05$). Pine trees within the first 60 m of infected *Ribes* spp. were more likely to be infected than trees further away (Fig. 5). The same trend was observed for pine trees with aecial scars or other WPBR symptoms but the main effect of distance ($P = 0.09$) was not significant at $\alpha = 0.05$ for pine trees with other WPBR symptoms. The effect of cardinal direction downwind from infected *Ribes* plants on incidence of infected pine was not significant at $\alpha = 0.05$ but was significant at $\alpha = 0.1$ ($P = 0.06$ for pine trees with aecia and $P = 0.08$ for trees with aecia or other WPBR symptoms). Consistent with prevailing wind directions in New England, it was more likely to find pines with aecia east (probability = 0.15) or west (probability = 0.19) than north (probability = 0.02) of infected *Ribes* plants. Similarly, it was more likely to find pines with aecia or other WPBR symptoms east (probability = 0.24) or west (probability = 0.31) than north (probability = 0.11) of infected *Ribes* plants. Finally, tree size had a significant effect on WPBR incidence on white pine ($P = 0.0001$). Seedlings (dbh < 2.5 cm) were at least five times more likely to be infected than saw timber-size trees (dbh > 23 cm) (Fig. 6).

Discussion

Cronartium ribicola is one of the most damaging invasive forest pathogens introduced to North America (Aukema et al. 2010; Liebhold

et al. 1995, 2012). In the Northeast, the ubiquitous presence of a very susceptible exotic plant, *R. nigrum*, intensified damage caused by WPBR to native eastern white pine (Benedict 1981). European black currant, consequently, has been regulated since the early 1900s. In the early 2000s, when state regulators were considering relaxing *Ribes* restrictions to allow planting of WPBR-resistant *Ribes* cultivars, Bergdahl and Teilon (2000) cautioned that the resistance might change over time. As predicted, the breakdown of the *Cr* resistance

Fig. 2. Comparison of white pine blister rust incidence among cultivated *Ribes* spp. in New Hampshire. European black currant cultivars 'Titania', 'Consort', 'Coronet', and 'Crusader' are defined as Cr *Ribes* cultivars. Error bars represent standard errors. Values with the same letter are not statistically different ($\alpha = 0.05$).

Table 1. Descriptive data for currant and gooseberry plants in the genus *Ribes* sampled in New Hampshire to determine white pine blister rust (WPBR) disease incidence and severity of each cultivar

Type of fruit	<i>Ribes</i> spp.	Cultivar	WPBR incidence ^a	Number of plants	Confirmation ^b	WPBR severity ^c	Percentage sites with infected plants	Number of sites
Black currant	<i>Ribes nigrum</i>	'Consort' ^d	19 ± 10	16	Yes	19.4 ± 9.8	29	7
		'Coronet' ^d	60 ± 25	5	Yes	21.1 ± 10.9	50	4
		'Crusader' ^d	0 ± 0	8	No	0 ± 0	0	3
		'Titania' ^d	44 ± 6	72	Yes	16 ± 2.4	38	21
		Willoughby	38 ± 18	8	Yes	0.6 ± 0.3	50	2
Currant × gooseberry	<i>R. odoratum</i>	Crandall	76 ± 10	21	Yes	0.6 ± 0.2	86	7
		<i>R. x nidigrolaria</i>	88 ± 13	8	Yes	1.7 ± 1.3	100	1
Gooseberry	<i>R. oxycanthoides</i>	Jahns Prairie	88 ± 5	43	Yes	1.9 ± 0.5	100	15
		<i>R. uva-crispa</i>	67 ± 33	3	Yes	0.2 ± 0.2	50	2
		Careless	83 ± 17	6	Yes	0.8 ± 0.4	100	3
		Clark	67 ± 33	3	Yes	0.3 ± 0.1	100	1
		Crown Bob	50 ± 50	2	No sample	0.1	100	1
		Invicta	100 ± 0	2	Yes	0.7 ± 0.4	100	1
		Pixwell	24 ± 8	29	Yes	0.1 ± 0	45	11
Red currant	<i>R. rubrum</i>	Red Lake	100	1	Yes	0.1	100	1
		Rolan	25 ± 25	4	No sample	0.1	50	2
		Rondom	29 ± 13	14	No sample	0.8 ± 0.3	20	5
		1301	0 ± 0	9	No	0 ± 0	0	4
White currant	<i>R. rubrum</i>	Blanka	100	1	Yes	1.7	100	1

^a Mean disease incidence ± standard error (SE) for each cultivar. Incidence is defined as percentage of plants with WPBR. A plant (replicate) was considered positive if any of the 18 leaves (subsamples) inspected for severity or other symptomatic leaves had signs of the causal agent of WPBR, *Cronartium ribicola*.

^b Polymerase chain reaction confirmation of *C. ribicola*.

^c Mean disease severity of infected plants ± SE for each cultivar. Disease severity represents the percentage of leaf area with rust pustules produced on the lower side of leaves, including chlorotic halos, necrotic tissue surrounding pustules and coalesced lesions. An estimate was obtained for each plant by calculating the mean percent diseased leaf area for 18 leaves (3 leaves from each of the bottom, middle, and top sections of each of two canes).

^d Cr *Ribes* cultivars.

dominant gene in *R. nigrum* 'Titania' was first reported in 2011 in Connecticut (Frederick et al. 2011).

In this study, we show evidence that the vCr race of *C. ribicola* that has overcome the *Cr* resistance dominant gene in cultivated *Ribes* spp. is present in both cultivated *Ribes* plants and pine trees in New Hampshire. The now susceptible European black currant cultivars with the *Cr* gene (*Cr Ribes* cultivars) pose a threat to neighboring white pine. In the current study, *C. ribicola* inoculum, collected from New Hampshire infected plants labeled as 'Titania', 'Consort', and 'Coronet', successfully infected leaves of reference accessions of *Cr Ribes* cultivars from the Canadian Clonal Genebank. The *C. ribicola* vCr race was also found in eastern Canada in surveys conducted during 2013 in commercial black currant fields in Quebec, New Brunswick, Nova Scotia, and Prince Edward Island (Tanguay 2014). In that recent outbreak, *Cr Ribes* cultivars 'Titania', 'Consort', 'Tisel', and 'Blackcomb' were infected with *C. ribicola* (Tanguay 2014). In the current study, 'Titania' was the most frequently planted (56% sites) *Ribes* cultivar in New Hampshire and it was frequently infected by the *C. ribicola* vCr race (44% plants infected). Other *Cr Ribes* cultivars surveyed in New Hampshire also exhibited a breakdown in WPBR resistance. In all, 19 and 60% of 'Consort' and 'Coronet' plants,

respectively, were infected by *C. ribicola*. Results from the current study suggest that these now susceptible European black currant cultivars pose a threat to local white pine resources, which have greater WPBR incidence in the vicinity of these infected *Cr Ribes* plants compared with pine trees growing next to other infected resistant cultivars or uninfected *Ribes*. The vCr race of the pathogen also caused cankers in the heavily infected white pine natural forest located close to a 'Titania' planting in eastern Canada (Tanguay et al. 2013). Preliminary genetic analyses confirm that the vCr race of the pathogen that has overcome the *Cr* gene in *Ribes* spp. is the result of a new mutation or the genetic recombination of a North American strain of the fungus and not a new introduction of the disease (Tanguay et al. 2013).

In the Northeast, *R. nigrum* was the most important source of inoculum for the spread of WPBR because this extremely susceptible species grew on abandoned farms close to eastern white pine trees (Maloy 1997; Zambino 2010). European black currant is able to produce greater quantities of more vigorous inoculum compared with other *Ribes* spp. (Mielke 1943). Plants generally are taller and have large leaves and, consequently, more leaf area is available for inoculum production (Snell 1942; Spaulding 1922b). In addition,

Fig. 3. Comparison of white pine blister rust severity among cultivated *Ribes* spp. in New Hampshire. European black currant cultivars 'Titania', 'Consort', and 'Coronet' are defined as *Cr Ribes* cultivars. Error bars represent standard errors. Values with the same letter are not statistically different ($\alpha = 0.05$).

Table 3. Growth of *Cronartium ribicola* on susceptible control 'Ben Lomond' (without *Cr* gene) and reference accessions of black currant cultivars with the *Cr* gene (*Cr Ribes* cultivars) from the Canadian Clonal Genebank inoculated with aeciospores from eastern white pine in New Hampshire growing next to infected *Cr Ribes* cultivars or negative control inoculum from Canada

Source ^b	Eastern white pine tree	Canker	Sampled (n) ^c	Number of aecia with infectious aeciospores on ^a	
				Susceptible <i>Ribes</i>	<i>Cr Ribes</i>
Epsom	1	1	5	4	4
	2	1	10	8	8
	3	1	10	9	9
	4	1	10	8	9
	4	2	4	4	4
Concord	1	1	1	0	0
	1	2	1	1	0
	2	1	4	2	2
	3	1	5	0	1

^a *Ribes* leaf samples from susceptible and reference *Cr Ribes* cultivars were inoculated in Canada with New Hampshire inoculum within 4 days of rust collection in the field. Growth of the pathogen was assessed 4 weeks postinoculation. In contrast to susceptible *Ribes* plants, *Cr Ribes* cultivars 'Consort', 'Coronet', and 'Crusader' were not infected by negative control bulk aeciospores collected in 2008 from cankers in a white pine plantation in Saint-Isidore (Quebec, Canada).

^b Source of New Hampshire inoculum.

^c Number of aecia sampled. Spores from each aecium were used to inoculate a leaf sample from each *Ribes* cultivar.

Table 2. Growth of *Cronartium ribicola* on susceptible black currant control 'Ben Lomond' (without *Cr* gene, Cr-) and reference accessions of black currant cultivars with the *Cr* gene (Cr+, *Cr Ribes* cultivars) from the Canadian Clonal Genebank inoculated with urediniospores from plants labeled as 'Titania' (Cr+) in New Hampshire or negative control inoculum from Canada^a

Source of inoculum	Inoculum location	'Ben Lomond' (Cr-) infected	'Consort' (Cr+) infected	'Coronet' (Cr+) infected	'Crusader' (Cr+) infected
Urediniospores ^b	Durham	Yes	Yes	Yes	Yes
	Concord	Yes	Yes	Yes	No
	Epsom	Yes	Yes	Yes	Yes
	Chichester	Yes	Yes	Yes	Yes
	Temple	Yes	Yes	Yes	Yes
	Hanover	Yes	Yes	Yes	Yes
Aeciospores ^c	Saint-Isidore	Yes	No	No	No

^a *Ribes* leaf samples from susceptible and reference *Cr Ribes* cultivars were inoculated in Canada with New Hampshire inoculum within 3 to 15 days of rust collection in the field. Growth of the pathogen was assessed 4 weeks postinoculation.

^b Urediniospores from 'Titania' leaves from New Hampshire.

^c Aeciospores from pine cankers from Quebec, Canada.

compared with other *Ribes* spp., uredia and telia are larger in size and more abundant (Spaulding 1922a), and basidiospores have a greater survival rate after drying and brief exposure to sunlight on *R. nigrum* (Spaulding and Rathbun-Gravatt 1926).

Susceptible *Ribes* hosts can be infected by aecial or uredinal inoculum sources hundreds of kilometers away and then produce high levels of inoculum that infect less susceptible but locally abundant *Ribes* spp. (Zambino 2010). The native *Ribes* spp. of greatest importance to the initial WPBR epidemic in the Northeast were *R. cynosbati*, *R. rotundifolium*, *R. glandulosum*, and *R. americanum* (Zambino 2010). Today, wild and naturalized *Ribes* spp. are infrequently found in New England and they are typically restricted to stone walls and wet areas (Bergdahl and Teilon 2000; Zambino 2010). For example, in New Hampshire, *Ribes* plants were found in only 9 of 35 (17%) Phase 3 forested, long-term Forest Inventory Analyses plots inventoried from 2007 to 2010 by the USFS (Randall Morin, USFS Northern Research Station's Forest Inventory and Analysis, personal communication). In this study, only 2 of 43 sites had wild or naturalized currant (*R. hirtellum* and *R. rubrum*, respectively). These plants were not vigorous and, although infected by *C. ribicola*, disease severity was small compared with commercial cultivars.

Severity of WPBR in cultivated *Ribes* spp. varies with host genotype, environmental conditions, and pathogen population (Zambino

2010). For example, red currant (*R. rubrum*) 'Rolan' and 'Rondom'; *R. uva-crispa* 'Captivator', 'Careless', 'Clark', 'Crown Bob', 'Jahns Prairie', and 'Pixell'; and *R. hirtellum* were not infected in Oregon field trials (Hummer 1997). In New Hampshire, all of these *Ribes* spp. were infected by *C. ribicola*. In this study and in field trials conducted in New York (Snell 1942), disease severity on red currant and gooseberry (*R. grossularia*) was low. In Finland, red currant 'Jonkheer Van Tets' was highly susceptible to *C. ribicola* (Kaitera and Nuorteva 2006) whereas, in this study, it was not. Differences in environmental conditions among northern Europe and western and eastern North America could explain variation in resistance to WPBR in these *Ribes* spp. These *Ribes* spp., however, were also exposed to different strains of *C. ribicola*. Genetic differentiation among eastern and western populations of *C. ribicola* is very large and is attributed to a barrier in gene flow due to absence of hosts in the Great Plains (Hamelin et al. 2000). In contrast to results from this study, Cr *Ribes* 'Consort', 'Coronet', and 'Titania' were not infected by *C. ribicola* under natural field conditions in Oregon (Hummer 1997) or Vermont (Bergdahl and Teilon 2000). The most likely explanation for the lack of disease in 'Consort', 'Coronet', and 'Titania' in Oregon and Vermont is that the *C. ribicola* vCr race was not present at the time those field trials were conducted.

Although, in the current study, the presence of vCr race of *C. ribicola* in New Hampshire was confirmed, it is also possible that some infected black currant plants surveyed in New Hampshire labeled as Cr *Ribes* cultivars were misidentified. This was the case with 'Consort' accessions purchased at a nursery in Minnesota that became infected by *C. ribicola* but were genetically different from 'Consort' accessions from the USDA-ARS National Clonal Germplasm Repository and the University of Minnesota that were not infected by *C. ribicola* (Burnes et al. 2008). The genetic difference between nursery and reference 'Consort' accessions in that study was attributed to mixing of clones in a nursery or mislabeling. Given the interest in *Ribes* cultivation and the threat posed by WPBR to the white pine resource, the need to confirm the identity of commercially available *Ribes* cultivars through molecular markers is justified and deserves further investigation. For example, it would be very useful to develop PCR-based markers for WPBR resistance similar to the marker developed to identify resistance to the black currant gall mite conferred by a single gene (Brennan et al. 2009). The development of such markers would aid in the identification of mislabeled plants and help plant breeders to identify WPBR-resistant varieties.

It is interesting that, under field conditions, Cr *Ribes* 'Crusader' and white currant '1301' did not develop WPBR. These plants were relatively rare, growing in three and four of the surveyed locations,

Fig. 4. White pine blister rust (WPBR) incidence of pines in relation to presence of infected *Ribes* plants in New Hampshire. European black currant cultivars 'Titania', 'Consort', and 'Coronet' are defined as Cr *Ribes* cultivars. Values with the same letter are not statistically different ($\alpha = 0.05$).

Fig. 5. White pine blister rust (WPBR) incidence of pine in relation to distance from infected *Ribes* cultivars. Values with the same letter are not statistically different ($\alpha = 0.05$).

Fig. 6. White pine blister rust (WPBR) incidence of pine in relation to tree size. Pine trees were grouped by stem diameter at breast height (dbh; 1.3 m above the ground) into seedling (dbh < 2.5 cm, $n = 74$), sapling (dbh = 2.6 to 12.6 cm, $n = 91$), pole (dbh = 12.7 to 23 cm, $n = 74$), and saw timber (dbh > 23 cm, $n = 172$). Values with the same letter are not statistically different ($\alpha = 0.05$).

respectively. In a couple of these locations, WPBR was absent from white pine and other *Ribes* spp. were not present, which explains why these cultivars did not have WPBR. In other locations, however, both white pine and other *Ribes* cultivars had WPBR. In one of these locations, white pine had WPBR but Cr *Ribes* 'Consort', 'Coronet', and 'Crusader' were disease free, suggesting that the vCr race of the pathogen was absent at this site. In one location for each of the 'Crusader' and '1301' cultivars, both white pine and Cr *Ribes* cultivars were infected but neither 'Crusader' nor '1301' were infected. Infected Cr *Ribes* cultivars at these sites could have been mislabeled. Alternatively, 'Crusader' and '1301' plants could be escaping disease through phenology; for example, by leafing out before *C. ribicola* inoculum is available. Finally, it is possible that the white currant '1301' cultivar possesses a gene mechanism alternative to the Cr dominant gene that is also resistant to the vCr race. To determine whether '1301' plants are escaping disease or absence of disease in these plants is conferred by resistance genes, it would be necessary to challenge '1301' plants under controlled experimental conditions with the vCr race and other *C. ribicola* strains. If, under controlled inoculations, the '1301' cultivar remained disease-free, it might contain WPBR resistance genes of interest to *Ribes* breeders and growers. Similarly, it would be important to discern the mechanisms of WPBR resistance or escape of red currant cultivars that exhibited low disease severity in the field in the current study. Indeed, the *Ribes* gene pool in North America needs to be surveyed for WPBR resistance mechanisms alternative to the Cr dominant gene for resistance (Hummer and Dale 2010). It would be useful to map these genetic resistance traits with molecular markers, as described by Brennan et al. (2009).

At least 10% of the eastern white pine trees sampled in this study were infected with WPBR, which is a greater proportion than the 2.4% incidence previously reported in an extensive statewide survey (Lombard and Bofinger 1999). Other factors previously associated with WPBR such as proximity to susceptible *Ribes* plants, prevailing wind direction, and tree size were also associated with greater disease incidence in the current study. Incidence of pine infection declines sharply with distance from a source (Posey and Ford 1924; Van Arsdel 1960). In this study, proximity to susceptible black currant also significantly increased the probability of WPBR incidence on white pine. Although weather in New England in highly variable, the prevailing wind direction has a westerly component, whereas southeast winds are least common (Keim 1998). Accordingly, infected pine trees were least likely to occur north of inoculum sources. In this study, seedlings were more frequently infected than larger trees. Seedlings are more exposed to infection by *C. ribicola* compared with large trees because they have needles closer to the ground, where more spores from *Ribes* and favorable conditions for infection occur (Lachmund 1933; Mielke 1943).

For more than seven decades, Federal and State agencies have cooperated to remove *Ribes* spp. from selected white pine stands (Maloy 1997). Some stands were worked over up to five times (Benedict 1981). In the production regions with eastern white pine, \$38,632,855 was spent on *Ribes* eradication efforts by 1959 (Benedict 1981). *Ribes* eradication successfully controlled and reduced WPBR in northeastern states (Lombard and Bofinger 1999; Martin 1944; Ostrofsky et al. 1988; Van Arsdel 1960). A 50% reduction in WPBR incidence on white pine was observed in Maine after 70 years of *Ribes* eradication (Ostrofsky et al. 1988). In Maine, WPBR caused 50% of the white pine mortality in untreated areas versus 14% mortality in treated areas. Incidence of WPBR on pine was almost three times greater in untreated areas (9%) than in treated areas (3.8%) (Ostrofsky et al. 1988). In New Hampshire, only 2.4% of white pine trees sampled in an extensive statewide survey were infected by *C. ribicola* (Lombard and Bofinger 1999).

Results from the current study show that disease incidence is greater in eastern white pine in the proximity of Cr *Ribes* cultivars overcome by the *C. ribicola* vCr race. In addition, WPBR incidence and severity were evaluated for 15 WPBR-resistant *Ribes* cultivars under field conditions. This information is useful to states that want consistent data with which to make decisions about plant regulations (McKay 2000). As a result of this investigation, Cr *Ribes* cultivars have been

removed from the list of approved *Ribes* cultivars for planting in New Hampshire.

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