

Pedicularis and *Castilleja* are natural hosts of *Cronartium ribicola* in North America: a first report

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Summary

White pine blister rust disease, caused by the introduced pathogen *Cronartium ribicola*, has severely disrupted five-needled pine ecosystems in North America. A 100-year effort to manage this disease was predicated in part on the premise that the pathogen utilizes only species of *Ribes* (*Grossulariaceae*) as alternate hosts on this continent. The current study presents the first conclusive demonstration that some species in the family *Orobanchaceae* (*Pedicularis racemosa* and *Castilleja miniata*) are functioning as alternate hosts in a natural ecosystem of North America. This finding has implications for improving our understanding of epidemiology, pathogen adaptation and host–pathogen interactions within white pine blister rust.

1 Introduction

The white pine blister rust fungus, *Cronartium ribicola* J.C. Fisch., introduced into North America in the late 1800s, has caused significant mortality of five-needled (haploxylon) pines and was responsible for widespread disruption of ecosystems where these pines were keystone species (HUNT 2003). The life cycle of *C. ribicola* produces five-spore stages and requires distinct primary and alternate plant hosts. *Cronartium ribicola* was long assumed to utilize only *Ribes* spp. (currents and gooseberries; *Grossulariaceae*) as alternate (telial) hosts in North America. In 1909, this assumption led to initiation of a control programme in the eastern USA based partially on *Ribes* eradication. With the continuing spread of blister rust and detection of the disease in western conifers in 1921 (MIELKE 1943), eradication efforts became continent-wide in scope. *Ribes* plants were removed from over 8.1 million ha in the USA before the federal eradication programme was officially terminated in 1968 (KETCHAM et al. 1968; MALOY 1997) because of low correlations between stand infection rates and residual *Ribes* density after eradication in the eastern (KING 1958) and western (TOKO et al. 1967) USA.

The TOKO et al. (1967) study, which examined a total of 120 stands of western white pine (*Pinus monticola* Dougl.) in the inland north-western USA, found little relationship between annual infection rates of pines and *Ribes* density, regardless of eradication. For example, the ages of cankers were determined in 1966 on an 84-tree sample from a 27-year-old plantation (434-5-01) in northern Idaho. Canker incidence jumped 10-fold from 25 cankers per tree in 1960 to 250 cankers per tree in 1963, while *Ribes* density had been reduced from 99 to 0 bushes/ha in 1955 (from unpublished study data maintained at the USDA Forest Service-RMRS, Moscow, ID, USA). Most stands that had similar increases

in canker incidence after eradication were in the Coeur d'Alene National Forest in northern Idaho, USA.

Asia has been suggested to be the putative centre of diversity for *C. ribicola* and the source of blister rust in Europe, whence it was subsequently introduced into North America (LEPPIK 1970). An Asian centre of diversity is supported by studies that (i) document the extensive presence in Asia of *Cronartium* species, including microcyclic and macrocyclic species that utilize five-needled pines (IMAZU and KAKISHIMA 1995); (ii) distinguish Asian races of *C. ribicola* that differ in their abilities to utilize *Ribes* and hosts in several genera previously classified in the *Scrophulariaceae* (e.g. YOKOTA and UOZUMI 1976; SHAO et al. 1980; YI and KIM 1983; KAKISHIMA et al. 1995) that are now included in the family *Orobanchaceae* (OLMSTEAD et al. 2001) and (iii) indicate high resistance of some native Asian *Pinus* (HOFF et al. 1980) and *Ribes* species (YI and KIM 1983; BRENNAN 1996). These reports support a long-standing, co-evolved relationship of white pine blister rust pathosystems in Asia (MCDONALD et al. 2005).

Non-*Ribes* alternate hosts could be one explanation for low correlations between rust incidence and *Ribes* density in some regions of North America (MCDONALD et al. 2004). Common occurrence of *C. ribicola* on non-*Ribes* alternate hosts in North America would have major implications for understanding its epidemiology, adaptation and coevolutionary processes. Until now, evidence that species in the *Orobanchaceae* act as alternate hosts of *C. ribicola* in North America has come only from artificial inoculation studies. In one greenhouse study, aeciospore inoculations of *Castilleja miniata* Dougl. (scarlet paintbrush) plants resulted in lesions with telia, but no uredinia (HIRATSUKA and MARUYAMA 1976). Other controlled inoculations of this and other potential hosts in the *Orobanchaceae* failed to produce sporulating infections (HUNT 1984; PATTON and SPEAR 1989). Histopathology of the early infection process in artificially inoculated *C. miniata* indicated that some rust infections continued to colonize for up to 60 days, but did not produce haustoria or urediniospores (PATTON and SPEAR 1989). Infections on two *Pedicularis* species (*P. resupinata* L., native to eastern Asia and *P. canadensis* L., native to eastern North America) did not persist beyond 6 days. Descriptions of the latter infections are similar to those of non-host responses commonly observed in other host systems (MELLERSH et al. 2002; MYSORE and RYU 2004). However, non-*Ribes* species have not been previously reported as natural telial hosts of *C. ribicola* in North America.

In August and September 2004, naturally occurring leaf lesions bearing telia typical of the genus *Cronartium* were found on four plant species in the *Orobanchaceae* (*Pedicularis racemosa* Dougl., *P. bracteosa* Benth., *C. miniata* and *Castilleja rhexifolia* Rydb.) at a subalpine, whitebark pine (*Pinus albicaulis* Engelm.) site in the vicinity of Roman Nose Lakes Camp Ground (48.634109°N, 116.570817°W, elevation 1800 m). This site is located 30 km west of Bonners Ferry (ID, USA) and had been burned during the Sundance fire approximately 38 years ago. A high proportion of approximately 25-year-old whitebark pine and western white pine were infected with the white pine blister rust fungus. *Pedicularis*, *Castilleja* and *Ribes* species were significantly represented in the shrub and herbaceous vegetation.

Cronartium coleosporioides J.C. Arthur, which causes stalactiform blister rust on lodgepole pine (*P. contorta* Dougl.; VOGLER and BRUNS 1998), occurs near the discovery site. This blister rust alternates to some species in the *Orobanchaceae* (FARR et al. 1995; VOGLER and BRUNS 1998), and teliospores produced on alternate hosts are morphologically indistinguishable from *C. ribicola*. Recently, a rust fungus that appears to be a hybrid between *C. ribicola* and *C. comandrae* Peck was detected on limber pine (*P. flexilis* James; JOLY et al. 2004; HAMELIN et al. 2005). Our objective was to determine whether infections on hosts in the *Orobanchaceae* at the discovery site were of *C. ribicola*, *C. coleosporioides*, or a hybrid species.

2 Materials and methods

Four methods confirmed the identity of the infections and/or tested the ability of several non-*Ribes* species to act as alternate hosts: (i) DNA sequencing of the internal transcribed spacer (ITS)1, 5.8S and ITS2, regions of the ribosomal DNA (rDNA), hereafter referred to as ITS + 5.8S; (ii) alternate host inoculations; (iii) scanning electron microscopy of germinating teliospores and (iv) pine seedling inoculations.

2.1 PCR and rDNA sequencing

The ITS + 5.8S rDNA regions were sequenced for naturally occurring *Cronartium* infections on *R. hudsonianum* Richards and non-*Ribes* alternate hosts obtained from the discovery site during August and September 2004. Single teliospore columns excised from non-*Ribes* alternate host leaves or urediniospores from *R. hudsonianum* served as DNA templates for 30- μ l polymerase chain reaction (PCR) mixtures. Reaction mixtures contained 0.2 mM dNTPs, 4 mM MgCl₂, 0.5 μ M fungal-specific primers ITS1 and ITS4 (WHITE et al. 1990), 1 U of AmpliTaq[®] DNA polymerase (Applied Biosystems, Inc., Foster City, CA, USA) and PCR buffer (supplied with the DNA polymerase). PCR was performed on a MJ PTC-200 thermocycler (Bio-Rad Laboratories, Waltham, MA, USA) using temperature cycling parameters previously described (KIM et al. 2006). PCR product size and band consistency were assessed by electrophoresis on a 1% agarose gel. Samples displaying a visible band were treated using ExoSAP-IT[®] enzyme preparation (USB Corp., Cleveland, OH, USA) according to the manufacturer's protocol. Samples were then submitted for DNA sequencing of the ITS + 5.8S region, which was conducted at the University of Wisconsin Biotechnology Center (Madison, WI, USA) using an ABI 3100 DNA sequencer (Applied Biosystems). Sequence data were checked against the original chromatograms and finally aligned using BIOEDIT v 7.0.1 (HALL 1999).

The ITS + 5.8S sequences from single teliospore columns from *Cronartium* infections were compared with known sequences for *C. ribicola* (GenBank L76499) and *C. coleosporioides* (GenBank L76500) available from earlier phylogenetic studies of *Cronartium* spp. (VOGLER and BRUNS 1998). Sequences from *Cronartium* spp. collected at the discovery site were deposited into GenBank (Table 1). Voucher specimens of *Cronartium* infections on leaves have been submitted to the Mycological Herbarium of the

Table 1. List of collections from the vicinity of Roman Nose Lakes Campground, ID, USA that have been identified using rDNA internal transcribed spacer sequencing (ITS1, ITS2, 5.8S)

Host plant	Collection date	Pathogen	GenBank number
<i>Pedicularis racemosa</i>	1-9-2004	<i>Cronartium ribicola</i> ¹	AY955829
<i>P. racemosa</i>	1-9-2004	<i>C. ribicola</i> ¹	AY955831
<i>P. racemosa</i>	1-9-2004	<i>C. ribicola</i> ¹	AY955833
<i>P. racemosa</i>	1-9-2004	<i>C. ribicola</i> ²	AY955830
<i>P. racemosa</i>	1-9-2004	<i>C. ribicola</i> ¹	AY955832
<i>P. bracteosa</i>	1-9-2004	<i>C. coleosporioides</i> ¹	AY955834
<i>P. bracteosa</i>	1-9-2004	<i>C. coleosporioides</i> ¹	AY955835
<i>Castilleja rhexifolia</i>	1-9-2004	<i>C. coleosporioides</i> ¹	AY955828
<i>C. miniata</i>	1-9-2004	<i>C. ribicola</i> ¹	AY955827
<i>Ribes hudsonianum</i>	20-8-2004	<i>C. ribicola</i> ¹	DQ017954

¹Specimens deposited at the Department of Plant Pathology Mycological Herbarium, Washington State University, Pullman, WA, USA.
²Specimen deposited at the Institute of Forest Genetics Herbarium, USDA Forest Service Pacific Southwest Experiment Station, Camino, CA, USA.

Department of Plant Pathology (Washington State University, Pullman, WA, USA), and to the Institute of Forest Genetics (USDA Forest Service, Pacific Southwest Research Station, Camino, CA, USA).

2.2 Inoculations of alternate hosts and western white pine

Three sources of aeciospores were used to inoculate alternate hosts. Each source was collected on 1 September 2004 from a different single canker on a different whitebark pine at the discovery site. All sources were used to inoculate plants of *P. racemosa* from the site and single-leaf stem cuttings of *R. nigrum* L. from greenhouse-grown plants. The single-leaf stem cuttings were of the two youngest, fully expanded leaves from rapidly growing plants of a susceptible *R. nigrum* clone developed by C. Heimberger (PATTON 1972). Inoculations of hosts were made using small incubation chambers transported to the site. Each chamber was a 946-ml, clear, polypropylene container (8-cm diameter base, 14 cm height, with tight-fitting lid). The *R. nigrum* cuttings were transported to the site in small pots of moist vermiculite within inoculation chambers. On site, small plants of *P. racemosa* with native soil still attached to the root ball were placed in chambers. For each set of inoculations, *P. racemosa* plants and *R. nigrum* cuttings were removed from chambers, the abaxial surfaces of leaves dusted with aeciospores from a single excised canker by tapping the canker directly over the plants, the inside of the chambers and the adaxial surfaces of leaves misted with distilled water, and the chambers closed to provide 100% relative humidity. Gloved hands and the outside of chambers were wiped with 70% ethanol before and after inoculation with each source to prevent contamination. For negative controls, additional cuttings of *R. nigrum* were briefly removed from chambers, exposed on site and misted with water. Chambers containing the inoculated hosts and controls were transported back to the laboratory in coolers.

Two plants of *C. miniata* that had been obtained from the Deception Creek Experimental Forest near Coeur d'Alene (ID, USA) and maintained in a greenhouse for 2 years were inoculated in the laboratory (USDA Forest Service-RMRS, Moscow, ID, USA) by similarly dusting both plants with aeciospores from a single source used in the previous inoculations. Plants were then misted with distilled water, and enclosed in polypropylene bags. *Ribes nigrum* cuttings in chambers were used as positive and negative controls as previously described.

Hosts inoculated with aeciospores were placed in a lighted, 19°C incubator (VAN ARSDEL et al. 1956) with a 12-h photoperiod. After 48 h, inoculation chamber lids were replaced with a layer of laboratory tissue (Kimberly-Clark, Neenah, WI, USA) secured with a rubber band, and incubation continued at 20°C. Plants of *C. miniata* were unbagged and returned to the greenhouse.

Single-leaf cuttings of *R. nigrum* were inoculated with urediniospores from 11 single pustules that developed on experimentally infected *P. racemosa* plants and represented all three aeciospore sources. Urediniospores were transferred to misted cuttings by touching the tip of a boar bristle to single pustules and wiping the bristle through minute droplets of water at a location marked on the abaxial leaf surface of the cutting. The bristle was examined for spores before, during and after spore transfer, and was also sterilized in 70% ethanol before and after each transfer. Cuttings were incubated at 20°C (VAN ARSDEL et al. 1956) for 24 h before chamber lids were replaced with tissue. Once uredinia developed, the isolates were further multiplied on new cuttings, with the temperature regime for the 12-h photoperiod altered to 20°C day/16°C night to induce formation of telia (VAN ARSDEL et al. 1956).

Basidiospore-producing telia representing six of the 11 single pustule-derived strains and all three aeciospore inoculum sources were used to inoculate young seedlings of western white pine at the cotyledon to primary needle stage. Pieces of leaf tissue with telia were

soaked 4 h at 4°C in sterile distilled water, then free moisture was removed with tissue. Leaf pieces were then affixed, teliospore columns outward, to a plate of 1.5% water agar, and the plate suspended over seedlings in plastic incubation chambers. Seedlings were incubated for at least 48 h at 16°C after basidiospores had been cast, then lids were removed as described above; subsequent incubation was at 20°C.

2.3 Microscopy

Small pieces of leaf tissue from inoculated *P. racemosa* that contained columns of teliospores were soaked for 4 h at 4°C in sterile distilled water, affixed to the side of a block of 1.5% water agar atop a water agar plate, and held at 16°C in the dark until initial stages of teliospore germination were observed at 200× magnification under a compound light microscope. The pieces of agar with attached leaf material were then held over a 4% aq. OsO₄ solution for vapour fixation, lyophilized (Virtis Co., Gardiner, NY, USA), sputter-coated (Technics Hummer V, Anatech, San Jose, CA, USA) with gold, and examined using a scanning electron microscope (Model S-570, Hitachi, Ltd, Tokyo, Japan) at the Electron Microscopy Center, Washington State University, Pullman, WA, USA.

3 Results and discussion

In August 2004, *Cronartium* infections were observed on *Ribes* and four species of *Orobanchaceae* at a northern Idaho site containing *C. ribicola*-infected whitebark and western white pine. Based on ITS + 5.8S sequences, infections on sickletop lousewort (*P. racemosa*; Fig. 1a), scarlet Indian paintbrush (*C. miniata*) and stinking current (*R. hudsonianum*) were identified as *C. ribicola* (Table 1). Infections on *C. rhexifolia* and *P. bracteosa* were identified as *C. coleosporioides*.

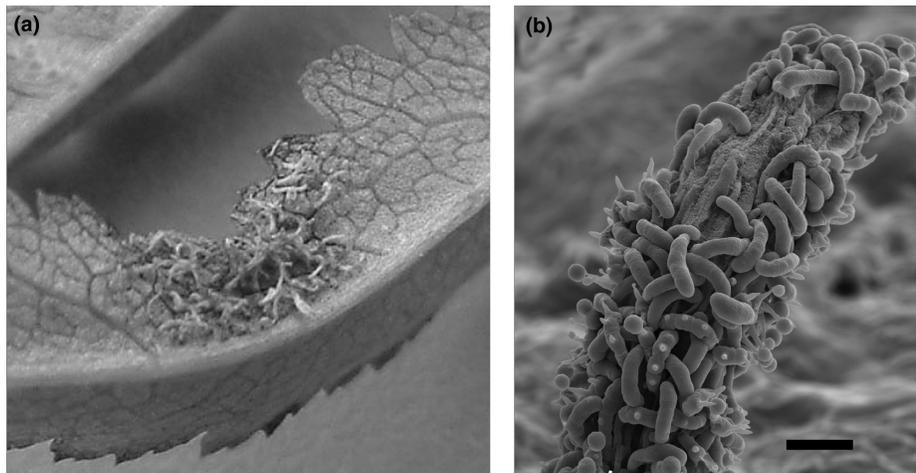


Fig. 1. (a) DNA sequencing of the rDNA internal transcribed spacer region (ITS1, 5.8S, ITS2) identified *Cronartium ribicola* as the rust species causing this telium on the perennial herb *Pedicularis racemosa* (*Orobanchaceae*) collected from the vicinity of Roman Nose Lakes Campground in northern Idaho. The hair-like structures are columns of teliospores (leaf width approximately 1.5 cm, photo credit: John Hanna). (b) Scanning electron micrograph of early stages of germination in a column of teliospores from *P. racemosa* infected with *C. ribicola* by controlled inoculation (bar = 30 μm). Four sterigmata can be seen on each of several allantoid probasidia; four spherical basidiospores per probasidium were observed on basal portions of this teliospore column (not shown)

Laboratory inoculations with *C. ribicola* aeciospores from whitebark pines at the discovery site were all successful and resulted in sporulating infections on *P. racemosa*, *C. miniata* and *R. nigrum*. These infections all produced both uredinia and telia, although urediniospores were rare on *C. miniata*. Negative controls did not become infected. Western white pine seedlings were successfully infected with all six isolates that had been derived from single pustules on experimentally infected *P. racemosa*. Symptoms of *C. ribicola* infection were produced on cotyledons, primary needles and stems. Subsequently, these infections produced pycnia and caused seedling death. The inoculation studies indicate that these *C. ribicola* isolates could complete a viable life cycle, and that these isolates were not specific to a single alternate host genus or to a single five-needled pine host.

Scanning electron microscopy of teliospore germination showed that four sterigmata (Fig. 1b) and four basidiospores were produced per probasidium (not shown), typical of regular meiosis. Thus, telial columns of these *Cronartium* isolates appear to be functionally diploid, and can be assumed to contain the same complement of genetic material as aeciospores from pine and urediniospores from alternate hosts. No double peaks were detected on ITS + 5.8S sequence chromatograms at the 27 informative sites that differentiate *C. ribicola* from *C. coleosporioides*, or at sites that differ between *C. ribicola* and other species of *Cronartium*. This lack of double peaks indicates that the telial columns from *Pedicularis* and *Castilleja* were not first-generation hybrids between *C. ribicola* and *C. coleosporioides*. However, evidence of hybridization in earlier generations could be masked by introgression.

Cronartium ribicola infection of *Pedicularis* sp. and *Castilleja* sp. could indicate a long-standing failure to recognize non-*Ribes* alternate hosts in North America, or even past oversights that ascribed all infections on such hosts to *C. coleosporioides* (see FARR et al. 1995 for host list for this species). Alternatively, *C. ribicola* could be adapting for broader host utilization. Persistent haploid cankers on long-lived pine hosts might facilitate evolutionary change through conventional genetic mechanisms, such as genetic recombination among strains or hybridization between species. Aeciospores with hybrid morphology and DNA markers were recently reported as evidence of interspecific hybridization between *C. ribicola* and *C. comandrae* on the five-needled pine, limber pine (JOLY et al. 2004; HAMELIN et al. 2005). As yet, no evidence has been obtained concerning the viability or non-viability of such interspecific crosses in *Cronartium*, but a hybrid *Melampsora* rust species has been reported as a naturally occurring pathogen of hybrid poplars (NEWCOMBE et al. 2001).

At *C. ribicola*'s putative centre of diversity in north-eastern Asia, different *C. ribicola* populations have been shown to infect different combinations of local *Pedicularis*, *Castilleja* and *Ribes* species (e.g. YOKOTA and UOZUMI 1976; SHAO et al. 1980; YI and KIM 1983; KAKISHIMA et al. 1995; McDONALD et al. 2005). In North America, more data are needed on the occurrence of *C. ribicola* infections on *Orobanchaceae* to detect geographical patterns and potential interactions between pathogen and host populations over a range of environments. However, our observation of natural *C. ribicola* infections on *C. miniata* at a site in the Rocky Mountains near the USA/Canadian border supports the results of HIRATSUKA and MARUYAMA (1976), who used similar geographical sources of alternate hosts and *C. ribicola* for their study. The inability of HUNT (1984) and PATTON and SPEAR (1989) to obtain sporulating infections on *Orobanchaceae* (e.g. *C. miniata* from Vancouver Island, British Columbia, Canada) might reflect the use of different regional aeciospore sources, hosts, or inoculation techniques/environments.

Differences in telial host usage have been documented for *C. ribicola* in Asia (see McDONALD et al. 2005). In India and China, the only known telial hosts are *Ribes* spp. Complex regional patterns of host alternation to *Ribes*, *Orobanchaceae*, or both are found in Siberia, Korea and Japan. If differences in inoculation success in North America are

associated with geographical variants of hosts or pathogen, then interactions between *C. ribicola* and its alternate hosts may reflect genetic adaptation, developmental plasticity and/or trait reversion in specific populations. Trait reversion, sometimes referred to as 'evolution in reverse', is well known in evolutionary biology (PORTER and CRANDALL 2003; WEST-EBERHARD 2003). Reversions are derived character states that reproduce states present in ancestral lineages (PORTER and CRANDALL 2003). Patterns of alternate host utilization may fall within a special kind of trait reversion known as recurrence, defined as '...the phyletically disjunct appearance of homologous traits' (WEST-EBERHARD 2003, p. 353). At its centre of diversity, some *C. ribicola* populations may be exhibiting recurrence, evident as alternative phenotypes that infect different alternate hosts. Such recurrence is well known in other types of organisms (WEST-EBERHARD 2003), but it has not yet been attributed to host-range alternation in fungi. Antibiotic resistance in bacteria and host-jumping by certain viruses has also been attributed to mechanisms of trait reversion (PORTER and CRANDALL 2003).

From sequence analysis and an analysis of host relationships, *C. flaccidum* represents an ancestral clade for the genus *Cronartium* (VOGLER and BRUNS 1998). This plurivorous pathogen can utilize 10 diverse families of angiosperms as alternate hosts, including *Orobanchaceae* (*Pedicularis* sp. and *Melampyrum* sp.; MILLAR and KINLOCH 1991; KAITERA 1999). Occurrence of the white pine blister rust fungus *C. ribicola* on *Pedicularis* sp. and *Castilleja* sp. could represent trait reversion if the plurivory in *C. flaccidum* is an ancestral instead of a derived state (VOGLER and BRUNS 1998). Trait reversion might allow additional species or genera in the *Orobanchaceae* or other families to serve as alternate hosts for *C. ribicola*.

The blister rust infections at the discovery site could also represent a hitherto undetected introduction of *C. ribicola* that was separate from the well-documented introduction into Vancouver, British Columbia, Canada in 1910. Separate introductions have been previously proposed to explain anomalies in patterns of blister rust spread in western North America (HUNT 2003). However, introduction history should be reflected in regional patterns of genetic diversity for the pathogen. Ample evidence shows that eastern and western populations of *C. ribicola* are genetically isolated (HAMELIN et al. 2000), but available evidence shows little differentiation in the western population, with the greatest diversity apparent near the presumed centre of origin in Vancouver (P. J. ZAMBINO, R. C. HAMELIN, and N. LECOURS unpublished data). Additional studies are underway to determine relationships among *C. ribicola* populations and the possibility of subsequent cryptic introductions.

Both susceptibility and resistance to *C. ribicola* have been demonstrated in North American populations of five-needled pine and *Ribes* (KINLOCH 1992; McDONALD et al. 2004; ZAMBINO and McDONALD 2004), and host-pathogen interactions should now be examined for host species in the *Orobanchaceae*. If the forms of resistance found in these species represent host resistance instead of non-host resistance, this may suggest previous host-parasite coevolution between *Cronartium* and these hosts in North America. Perhaps an ancient North American pathosystem, disrupted during climate-associated migrations, is now being reconstituted (McDONALD 2004). Landscape scale genetic discontinuities (MANEL et al. 2003) in *Ribes*, *Orobanchaceae* and *Pinus* might reveal co-migrating populations that have functioned as evolutionarily significant units, retaining features of their ancient interactions with the genus *Cronartium* (McDONALD 2004).

Cronartium ribicola infection of species in the *Orobanchaceae* revises our understanding of this pathogen's epidemiological behaviour and of the evolutionary ecology of white pine blister rust pathosystems. Natural infection of *Orobanchaceae* may explain some shortcomings of the 50-year, blister rust control programme that cumulatively cost approximately 1 billion in current USA dollars, as calculated from previously published estimates (MALOY 1997). Successful restoration of impacted ecosystems on local and

regional scales requires an accurate understanding of potential behaviour of epidemics, and the outcome of epidemics in shaping new host and pathogen populations. Knowledge of evolutionary history, coevolutionary processes and landscape genetics of *C. ribicola* and its hosts on a global scale may aid in this understanding. Identifying roles of different alternate hosts and understanding the adaptive and co-evolutionary processes that affect *C. ribicola*'s utilization of alternate hosts may thus facilitate the restoration of disrupted ecosystems by providing a more accurate model of how hosts and pests co-accommodate in environments highly heterogeneous across space and time. Detailed investigation of genetic interactions within this expanded blister rust pathosystem should yield valuable new perspectives about species introductions and host-pest coevolution.

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Résumé

Première mention de Pedicularis et Castilleja comme hôtes naturels de Cronartium ribicola en Amérique du Nord

La rouille vésiculeuse des pins à cinq feuilles, causée par le parasite introduit *Cronartium ribicola*, a gravement perturbé les écosystèmes de pins blancs en Amérique du Nord. Un programme de gestion de cette maladie mis en place depuis 100 ans a été basé en partie sur l'hypothèse que le parasite n'utilise que des espèces de *Ribes* (*Grossulariaceae*) comme hôtes alternants sur ce continent. Cette étude présente la première démonstration solide que quelques espèces de la famille *Orobanchaceae* (*Pedicularis racemosa* and *Castilleja miniata*) fonctionnent comme hôtes alternants dans un écosystème naturel d'Amérique du Nord. Cette observation a des implications pour l'amélioration de la compréhension de l'épidémiologie, de l'adaptation du parasite et des interactions hôte-parasite de la rouille vésiculeuse des pins blancs.

Zusammenfassung

Erstnachweis von Pedicularis und Castilleja als natürliche Wirte von Cronartium ribicola in Nordamerika

Der Weisskiefernblasenrost, hervorgerufen durch das eingeschleppte Pathogen *Cronartium ribicola*, verursacht grosse Schäden in Ökosystemen in Nordamerika, die durch fünfnadelige Kiefern geprägt sind. Während 100 Jahren basierten Versuche, diese Krankheit unter Kontrolle zu bringen, auf der Annahme, dass auf diesem Kontinent das Pathogen nur mit Ribesarten (*Grossulariaceae*) einen Wirtswechsel durchführen kann. Die vorliegende Untersuchung weist erstmals nach, dass einige Arten aus der Familie *Orobanchaceae* (*Pedicularis racemosa* und *Castilleja miniata*) in einem natürlichen Ökosystem Nordamerikas als alternierende Wirte dienen können. Dieser Befund ist von Bedeutung für das Verständnis der Epidemiologie, der Adaptation des Pathogens und der Wirt-Parasit-Interaktionen bei dieser Rostkrankheit.

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