

Utilizing genetic information to address *Phytophthora ramorum*/Sudden Oak Death

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In December 2005, geneticists and plant pathologists met to articulate what genetic information is needed to address *Phytophthora ramorum*/Sudden Oak Death and to discuss whether a program to develop genetic resistance within any species was needed (and feasible) at this time. This paper summarizes their findings and discusses genetics-related research and management needs. The Appendix provides research needs, definitions and outlines the steps needed to develop a resistance program.

Summary

Research to elucidate baseline genetic and phenotypic variation in both the pathogen and various host species is essential to adequately understand the future impacts of Sudden Oak Death/*Phytophthora ramorum*. An understanding of susceptibility in each highly impacted host is needed to determine the long-term ecosystem impacts, and to help managers decide whether treatments, regulations and other management activities are needed. This document presents a brief rationale for research and management activities concerning genetic information for the pathogen, its hosts and their interaction. A resistance program may be feasible for hosts affected by this pathogen, but such programs are very expensive and can take decades. Such programs are usually considered only for economically valuable host species or species that would suffer dramatic levels of mortality over substantial portions of their range. Before considering such a program, more information is needed on the potential impacts to hosts and their ecosystems, as well as information on the variation in virulence/aggressiveness of the pathogen and the types of genetic resistance in host species. Public support for any resistance program would be essential.

Recommendations

We propose a genetics/resistance steering committee that will continue to define program needs and progress. Since genetics/resistance information is critical to advance understanding of all aspects of *Phytophthora ramorum*/Sudden Oak Death, we recommend an increased research effort to understand the genetics/resistance for *Phytophthora ramorum*/Sudden Oak Death.

Goals and objectives for a Sudden Oak Death/*Phytophthora ramorum* genetics program

Our goal is to ensure maintenance of all the components of ecosystems impacted by Sudden Oak Death/*Phytophthora ramorum*. To date, tanoak (*Lithocarpus densiflorus*) is the species most susceptible to *Phytophthora ramorum*, so an initial objective is to:

1. Determine if tanoak populations will be lost if action is not taken. More information is needed in the most highly impacted areas of tanoak to determine whether this species might recover without intervention. Information is also needed to determine if Sudden Oak Death/*Phytophthora ramorum*'s impact on tanoak is precluding meeting any management objectives.

Other objectives:

2. Support collection and assembly of adequate genetic data to understand past and current pathogen impacts and incidence so changes in susceptibility and mortality over time and the cause of those changes can be determined. Focus at this point is on host species on the West Coast, but much of this knowledge or the approach taken should be useful to eastern North American ecosystems that could be affected in the future if *P. ramorum* continues to spread.

3. Articulate genetic research needs and priorities for *P. ramorum*.

4. Provide the baseline genetic and other information necessary to potentially undertake a program to develop genetic resistance within an affected species.

Background: Sudden Oak Death and *Phytophthora ramorum*

Sudden Oak Death is a forest disease caused by the plant pathogen *Phytophthora ramorum*. Since 1995, the pathogen has killed over one million native tanoak, coast live oak, and other tree species in California (Meentemeyer, personal communication). Currently *P. ramorum* is found in the wildlands of 14 coastal California counties, from Monterey to Humboldt, as well as Curry County, Oregon and several isolated locations in the United Kingdom and the Netherlands. In California over 3.8 million forested acres are dominated by *P. ramorum* hosts (Waddell and Barrett 2005).

Tree losses continue to occur in both wildland and urban/wildland interface areas, with up to 80 percent of trees affected in some stands. This unprecedented loss of oaks in California is causing dramatic landscape changes that affect ecosystem function, increase fire and safety hazards, and reduce land values.

P. ramorum not only causes deadly cankers on bole hosts, but it also manifests itself as a foliar or twig blight on 84 known plant species, including California bay laurel, Douglas-fir, coast redwood, and numerous ornamentals, such as rhododendron and camellia. Unlike bark cankers, the foliar and twig blight rarely causes the host plant to die. Instead, these hosts allow for the production of large amounts of inoculum, thereby facilitating pathogen spread.

Foliar host infection has not only been identified in wildland settings among infected oaks, but it has also been found in European, United States, and Canadian nurseries. In Europe, more than 825 nurseries, in at least 11 countries, have been found to have the pathogen since 2002, whereas in the United States 278 nurseries have had positive *P. ramorum* detections in 21 states since 2002. In Canada, more than 20 nursery detections have been confirmed. It appears highly likely that *P. ramorum* will become a permanent resident of some of our North American ecosystems.

With many of the hosts being popular ornamental plants that are shipped nationally and internationally, there is concern over areas at high-risk of disease establishment becoming exposed to the pathogen. In response to these challenges, state and federal agencies have implemented *P. ramorum* quarantine regulations to help limit the pathogen's artificial movement.

Is a resistance program needed for any species impacted by *P. ramorum*?

To determine whether a resistance program is needed for any species, we asked:

1. Is this disease/pathogen precluding meeting any management objective?
2. If action is not taken will we lose a component of any ecosystems impacted by Sudden Oak Death/*Phytophthora ramorum*?

P. ramorum is known to infect over 80 species of trees, shrubs and herbaceous plants (APHIS 2006). There are no species currently considered threatened or endangered due to *Phytophthora ramorum*. Mortality is common on tanoak, coast live oak, California black oak, Shreve oak and madrone and has occurred on northern red oak (*Quercus rubra*) and European beech (*Fagus sylvatica*) in Europe. On conifers, stem cankers have been observed on Pacific yew, but symptoms are limited to branch and needle dieback on redwood (*Sequoia sempervirens*), Douglas-fir (*Pseudotsuga menzeisii*), grand fir (*Abies grandis*), white fir (*Abies concolor*), California red fir (*Abies magnifica*). Leaf spot symptoms are common on California bay laurel, buckeye, toyon and other species.

In southwest Oregon and central coastal California the primary tree species impacted by *P. ramorum* are tanoak and coast live oak. Neither of these species is considered to be commercially important. As acorn producers, they are both considered valuable for wildlife food and habitat. In urban environments they are valued for shade, screening, noise reduction and for the woody character they provide for landscapes. Both species have considerable cultural value to California coastal residents, particularly to Native Americans.

It is premature to recommend resistance programs for coast live oak and tanoak, but development of the techniques and protocols needed to evaluate susceptibility and quantify the inherent resistance within the species are needed.

Based on environmental factors (e.g. climate) tanoak's entire native range is considered at elevated risk for *P. ramorum*. For coast live oak, most of its range is at risk, only the very southernmost extent is considered low risk (Meentemeyer and others 2004).

Based on neutral genetic markers, Dodd et al. (2005) reported evidence for significant population genetic structure in coast live oak. Subsequent unpublished data (Dodd personal communication) indicate that coast live oak can be separated into at least 4 groups (north, central and south coastal and central interior) based on neutral molecular markers and into northern and southern groups based on quantitative growth traits. This data would suggest that if a resistance program was ever recommended for coast live oak a minimum of at least four separate breeding populations/programs would be needed to capture the genetic variation present in each of the respective populations. No information currently exists for population genetic structure of tanoak. Additional studies aimed at comprehensively quantifying rangewide levels and partitioning of genetic variation in both species, especially tanoak, are greatly needed. This data should not only include information based on neutral genetic markers, but must also include data on various adaptive traits. Information regarding the population genetic structure for neutral as well as adaptive traits of any species for which a resistance program might be recommended is critical to assure that sufficient levels of genetic variation are represented in any future program.

Although Dodd et al. (2005) reported evidence for significant population genetic structure in coast live oak, little evidence for population differences in susceptibility was observed. It is quite possible that some useful level of resistance may occur within most populations, but for the long-term survival of this species on some of the more heavily impacted sites, an effective population size must be maintained. More information is needed about what constitutes a minimum viable population size for both coast live oak and tanoak. Currently, little is known about the extent and geographic distribution of genetic resistance to *P. ramorum* within tanoak. Preliminary studies have demonstrated differences in resistance among tanoak individuals and populations (Hayden and Garbelotto 2005).

More data is needed on the pathogen's impact on specific populations to determine if and where collections to maintain genetic diversity need to be made. Quantification of the percentage of tanoak killed in these high mortality areas is needed. Little or no information is currently available on whether the remaining tanoaks in these areas are currently infected, whether they are susceptible and what percentage will die in the future. Information on recent natural (in high mortality areas) and its level of resistance would also be of interest.

Summary of susceptibility

Despite extensive monitoring efforts additional plots are needed to quantify pathogen impacts in watersheds that are severely impacted. Additional baseline data will be needed before any recommendations can be made regarding specific actions to be taken to ensure tanoak's place in the ecosystem. Some of the more important questions that need to be immediately addressed are:

- 1) What percentage of tanoak is dead in the most highly impacted areas?
- 2) What is the temporal progression of mortality in these areas?
- 3) What is predicted for the remaining tanoak?

Mortality estimates have been provided by McPherson and others (2005) tracking disease progression in Marin County. They found mortality of symptomatic trees increased from 2000 to 2003 as follows: *Quercus agrifolia* (n = 668), from 5.8 to 17.4%; *Q. kelloggii* (n = 53), from 3.8 to 9.4%; and *Lithocarpus densiflorus* (n = 164), from 8.3 to 22.2%. Bleeding developed in 40.9% of the initially asymptomatic *L. densiflorus* cohort. By 2003, 24.6% of the initially bleeding *L. densiflorus* cohort had died.

In plots in Marin, Sonoma and Napa Counties, Swiecki and Bernhart (2005) found, among live trees that had *P. ramorum* canker symptoms in 2000, 35% of tanoaks and 26% of coast live oaks had died by 2004.

For both McPherson and Swiecki's plots observations are on-going and future mortality is expected to gradually increase.

Tools available to managers focus on slowing or preventing the spread of a pathogen, mitigating its effect once present in an area, and restoration of a heavily impacted area. All of these can take a long-term concerted effort. Restoration may be needed if prevention or mitigation fails. Restoration will often require utilizing genetically resistant seedlings. A program to develop seedlings genetically resistant to a pathogen is long-term and expensive, and has been undertaken in North America for only a few forest tree species (e.g. Sniezko 2006). Due to the cost and timeframe needed and the lack of information on the epidemic and resistance available, it is premature to recommend creation of a breeding program for tanoak but we are advocating gathering the information needed to responsibly manage these stands and discern their likely futures (see appendix for outline of resistance breeding program). However, research on the availability of genetic resistance in the most susceptible species should take place.

Research Needs

A list of research questions for host genetics, pathogen genetics and host/pathogen genetic interactions is provided in the Appendix. The highest priority, immediate needs are:

1. Development and validation of a reliable test for resistance for various species;
2. Survival and susceptibility data on the specific populations to determine if and where collections to maintain genetic diversity need to be made;
3. Testing of seedlings from acorns of known parentage via inoculation studies so the heritable genetic vs. environmental factors for resistance can be determined;
4. Individuals that survived a SOD epidemic should be screened to determine if they escaped attack or are truly resistant.

Recommendations. The expertise and tools currently exist to answer many of the fundamental genetic questions relating to the pathogen and some of the major hosts. Answers to these genetic questions will present updated predictions of the future extent of mortality as well

as the potential for success in a resistance program. This information will allow land managers more definitive data on which to base management programs. We propose a genetics/resistance steering committee that will continue to define program needs and progress. Genetics/resistance information is critical to advance understanding of all aspects of *Phytophthora ramorum*/Sudden Oak Death. We recommend an increased effort to understand the genetics/resistance for *Phytophthora ramorum*/Sudden Oak Death.

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Appendix

I. Research Needs

Is there natural resistance to *P. ramorum* in affected native species? Potentially resistant individuals need to be tested against all three pathogen populations (European A1, and several A2 strains). If so, what is the level of natural resistance for tanoak and coast live oak? How many types of resistance are there? How are they geographically distributed? How effective are they? How are they inherited? What types of resistance are of immediate utility versus those that would need further breeding? Is the resistance durable? Answers to these questions will provide valuable data needed to model the future survival of the host species affected.

The first step in determining resistance is the development and testing of a reliable test for resistance. A test that can be done in a relatively short period of time by artificial inoculation of cuttings or seedlings is desirable. The test needs to be validated by comparison to performance of trees in the field. A test site where most affected species would be grown, would be needed.

What is the genetic architecture of the species? What is the life history and reproductive strategy for each species? How much genetic variation exists in the affected tree species? Provenance and common garden studies are needed to understand how widely material could be used for restoration and what type of planting stock is needed. These studies would help determine whether heritable resistance exists and would serve as a foundation if a breeding program is needed in the future.

Can we predict the outcome of the disease, understand the biology and ecology of what happens when a site is heavily impacted by the pathogen?

How likely are other threats (fire) and how would land managers respond to a major species extirpation?

We need to better understand the natural regeneration patterns of the species – can we establish them? Will plants remain disease-free if planted under infected trees? Is there a threshold for inoculum beyond which the stands will not survive?

Will there be undesirable impacts if action is not taken in Big Sur and other areas where tanoak has experienced high levels of mortality? What species will fill the void – native species or invasive exotics? What will be impact on watersheds (erosion, water quality)?

Can levels of resistance be increased to acceptable levels by management activities? Favoring sexual reproduction would increase the odds that resistant individuals would become naturally established. Since tanoak reproduces via acorns, how could this be done? Would eliminating predation increase the number of surviving resistant individuals? What type of site manipulation could be done to promote regeneration? Would favoring clonal reproduction of resistant individuals work? Hunting and killing deer to reduce browse? Temporary fencing? Reducing competition by removal of Douglas-fir? Increasing protection against cutting or killing by other agents? Minimizing seed predation from resistant trees? Is there a way that Agrifos can be used to enhance survival of resistant trees? Are increased measures to prevent fire justified?

- Pathogen Genetics

Compare the behavior and genetic makeup of the North American (A2s) and European populations of *P. ramorum* (A1s) and investigate the potential consequences should A1 strains and new A2 strains become established in North America. Investigate the possibility of sexual recombination.

Determine pathogen origin and how it was introduced and spread in Europe and North America.

Use the elucidation of the *P. ramorum* genome to improve diagnostic tests, understand pathogen virulence, and develop effective treatments.

Monitor pathogen genetic variation in both high impacted areas (lots of inoculum, high chance of mutation & selection) as well as newly impacted areas across North America, if they develop.

Monitor contemporary evolution and adaptation of *P. ramorum* populations in affected stands.

- Host/pathogen genetic interaction

Determine extent of host resistance for coast live oak and tanoak and evaluate application in a resistance program (both for immediate use and those that would need further breeding to reach high levels or frequency of resistance). Quantify infection and mortality rates in heavily impacted areas.

Quantify or characterize the level of expected mortality in infested stands.

Determine if there is a need to establish seed banks for potentially vulnerable or resistant species.

Is there an effect of spore load on resistance? What are the effects of environment on resistance?

Develop field diagnostics kits for *P. ramorum*, i.e. *P. ramorum*-specific ELISA.

Determine nature of resistance (i.e. gene-for-gene vs. multigenic field resistance) and nature of virulence in pathogen (i.e. presence of avirulence genes, effectors, etc.).

- Host Genetics

Investigate seed regeneration rates for *Umbellularia californica* and tanoak.

Determine patterns of variation in adaptive traits for tanoak and coast live oak (common garden tests).

Immediate Needs

-Development and validation of a reliable test for resistance for various species.

-More data is needed on the specific populations to determine if and where collections to maintain genetic diversity need to be made.

-Seedlings from acorns of known parentage need to be tested via inoculation studies so the heritable vs. environmental factors for resistance can be determined.

-Individuals that survived a Sudden Oak Death epidemic should be screened to determine if they escaped attack or are truly resistant.

II. Background on resistance

1. Resistance definition

The definition of resistance is often viewed narrowly, to mean complete immunity, none of the plants will be infected and all will live. Unlike agricultural crops, trees will be on the landscape for decades or even hundreds of years, so durability of resistance is paramount. In this case, it will be essential to assess the different types of resistance, especially types of partial resistance or quantitative resistance which though more difficult to evaluate may allow the best chance for long-term survival of the host and subsequent co-existence with the introduced pathogen. Knowledge of the evolutionary potential of the pathogen can be useful in evaluating the type of resistance that might be needed (McDonald and Linde 2002a, 2002b)

Resistance can be achieved via a single “R” gene or multiple genes. Some types of single gene resistance can be relatively easily overcome through evolution of the pathogen (i.e. in the white pine blister rust pathosystem). Partial resistance of many forms needs to be considered. Some types of resistance may be useful right away, others may need breeding to raise their levels or to combine with other types of resistance. For example in the case of *Phytophthora infestans* gene-for-gene resistance, discovered early on for management of potato late blight, proved to be useless as virulence developed rather quickly. Native *Solanum* species in central Mexico, that coevolved with *P. infestans*, resist severe late blight by using a combination of gene-for-gene and multigenic, field-resistance (Grunwald & Flier, 2005; Grunwald et al. 2002). Thus, it might well be that in the case of tanoaks, gene-for-gene resistance alone will not be effective. Unfortunately, breeding for multigenic field resistance is a slower and costlier.

Resistance can be captured by cloning healthy individuals in heavily infested areas, bred through crossing plants, or molecularly induced via genetic engineering techniques. Since replacement trees for Sudden Oak Death would be used in forest environments where genetically altered materials are not accepted by some members of the public, gene manipulations will not be pursued. Resistance programs for non-native, invasive pathogens using classical screening and breeding methods have made good progress for several tree species in North America (Sniezko 2006).

2. Timeline for resistance programs

Resistance programs for forest tree species are long-term and expensive undertakings. The fastest launch of a forest tree resistance program was in Port –Orford cedar (POC) for resistance to *Phytophthora lateralis*, cause of Port Orford Cedar root disease. The operational program was started in late 1996, and resistant seed for some breeding zones has been available since 2002. However, for other breeding zones, initial field selections, screening, and orchard development are still in early stages. Breeding work has commenced in the advanced breeding zones. Over 10,000 field selections have been evaluated in the first phase of resistance testing for POC, but only a small percentage of them are showing high levels of resistance. The POC resistance program was begun after extensive initial research on screening protocols and to ascertain whether genetic resistance was present to this non-native pathogen. The process for oaks species is projected to take much longer—perhaps 30 years for true oaks and 15 years for tanoaks. Some

improvements should be possible by evaluating the lessons learned in other forest tree resistance programs, as well as from tree improvement programs for oaks in the southern U.S. or elsewhere.

Using other forest tree resistance programs as a baseline, (such as *Phytophthora lateralis* at \$400,000/year) the costs of an operational resistance program for *P. ramorum* could be over \$1 million/year. This does not include the cost for research needed for program support and field material collection costs. Since at least 10 years may be needed to screen material the total program cost overtime might be expected to approach as much as \$10 million. Prioritizing by host species and breeding zones within species will obviously be necessary, and can help reduce costs. Developing protocols using one breeding zone as a prototype may also be helpful in streamlining a program for possible future expansion, as well as determining the actual level of resistance that might be achieved in a given period of time/effort.

3. Outline for a *Phytophthora ramorum* Disease Resistance Breeding Program

Objectives for a comprehensive disease resistance breeding program:

- 1) to identify, test, and deploy (as soon as possible) apparently resistant selections;
- 2) to understand the genetic mechanisms involved in apparent or observed resistant selections;
- 3) to use information gleaned from objectives 1 and 2 to increase levels of resistance via seed production in orchards (or natural stands) or by breeding while maintaining a sufficiently large genetic base and develop strategies to intelligently deploy resistant materials to mitigate against unacceptable losses.

The overall goal would be to intelligently manage for unacceptable economic or ecological losses from *P. ramorum*.

Objective 1. To identify, test, and deploy (as soon as possible) apparently resistant selections.

The first step is to identify candidate materials that may harbor useful resistance for further testing, breeding, and eventual deployment. This material might be immediately deployed or deployed at some later time after further improvement has been conducted. In addition to identifying apparently resistant materials, it is also equally important to identify susceptible individuals. This is usually not a problem but is equally important for two reasons. The first is that we will always need control materials to compare our apparently resistant material against. The second is that these materials will be needed if we are going to use breeding to better understand the genetic nature of any resistances observed, this will be true whether the resistance is qualitative or quantitative.

Resistant selections might come from selections of apparently healthy individuals identified in natural settings where disease incidence is high and selection pressure on the affected species is high, or alternatively they might be chosen from existing breeding and improvement programs. The latter is likely only going to be possible for species that were previously viewed as being economically or ecologically important and hence breeding programs already exist. For these species, it is likely that disease resistance would not have been a selection criterion, but it would

make sense to take advantage of these materials as they would more than likely already contain most of the genetic variation present in the species, since maintaining a diverse genetic base is a priority issue in forest genetic improvement programs. Depending on the frequency of resistance in natural populations, and the selection pressure already exerted by the pathogen in those areas, it may be necessary to screen 1000's or even 10,000's of candidates.

Capturing variation. Since plants can't move, they are generally very well adapted to the sites at/in which they currently grow, they are generally considered to be "locally adapted". This local adaptation needs to be considered in a resistance breeding program, as species will likely need to be re-established in quite diverse areas of their former ranges. Thus, this will require that selections represent each of the various habitats or ecosystems in which the species of interest is currently (or even historically) found, whether disease is currently a problem in these areas or not. Breeding zones which consist of smaller geographic-based units outside of which seed or materials are generally not moved, need to be identified through provenance testing, i.e., common garden studies, but without such prior information such a zonal system could simply be based on some key environmental or geographic features such as min/max temperature zones, min/max rainfall zones, or elevational zones. More zones are always better for capturing all of the potentially useful alleles responsible for adaptive traits, but the total number decided upon will be dictated by logistical, time, and money constraints.

Archiving. Once materials have been selected that likely would represent a fair proportion of the genetic variation present in the species of interest, these materials would need to be archived. Archiving is essential for two reasons. The material needs to be further tested for resistance, and they need to be safeguarded against possible loss. In most cases, archiving could be done either via the collection of seed or more ideally via the grafting of scion onto rootstock in an orchard. The main advantage of seed collection is that seed can often be easily collected but acorns can not be stored for more than a few months. Other disadvantages are that seeds are not genetic clones of the original plant and hence may not have the desired characteristics of the original selection, and in the case of long lived trees species, it may take a considerable amount of time before plants derived from these seeds could be tested and used in operational breeding efforts. Although, there are ways around the latter disadvantage of long juvenile phases such as top grafting or top working of young materials into mature trees to promote early flowering. The main advantages of using grafted materials are that they are an exact genetic clone of the original selection and multiple copies of the selection can be archived. The fact that multiple copies can be archived would hopefully ensure that they are not likely to be lost due to some local disturbance. It is always best to replicate materials at more than one site. However, any replication is good even if it is only at a single locale.

Artificial inoculation and short-term screening for resistance. Once materials are identified and archived, they will need to be further evaluated to assure that the apparent observed resistance is in fact "real" and not due to the fact that the material may be an escape that was not sufficiently challenged by the pathogen under natural field conditions. This will require the development of an artificial screening procedure that can accurately and reliably assess the relative resistance of the selected materials. At this point in time, in the case of *Phytophthora ramorum*, such a facility would need to be located in the West, and California seems to be the only likely locale since the disease is already present and quarantine issues would be much less

of a concern. The three main requirements of an artificial screening method are that it be accurate, reliable and amenable to high throughput. The following Standard Operating Procedures (SOPs) would need to be developed: 1. generating (and possibly storing) large quantities of inoculum; 2. using single genotype inoculum or bulk inoculum; 3. quantifying inoculum for standardization across runs; 4. defining the amount of inoculum needed to produce a quantifiable level of infection; 5. inoculum delivery onto/into tissues/plants; 6. identifying the tissue to be inoculated; and 7. scoring infected tissues/plants. The actual screening procedures to be used for identification of putatively resistant individuals would still need to be developed and rigorously tested.

Deployment of resistance. Once putatively resistant individuals have been identified, then progeny derived from these selections or clonal replicates of the selections would need to be established in the field to further test the durability of the observed resistance. The level of resistance present needs to be evaluated and a restoration strategy developed. Some proposed strategies for restoration might be as follows from the least labor intensive to the most labor intensive: [1] simply let surviving resistant trees produce offspring for natural regeneration, assuming there are enough of them that are close enough together to interbreed and maintain an acceptable level of genetic variation; [2] plant or graft resistant selections directly into open-pollinated seed orchards to produce diverse populations with increased levels of resistance, seed from which would then be available for deployment and further field testing for durability; or [3] directed breeding or crossing of specific resistant individuals and deployment of “families” for further field testing for both the level and durability of resistance, such an approach is very labor intensive but would provide the most information about the observed resistance/s in terms of heritability and modes of action associated with inheritance, i.e., is resistance primarily qualitatively (single gene) or quantitatively controlled (many genes), if quantitative, are the genes primarily having an additive effect on resistance or do they have a dominant or partially dominant effect.

Objective 2. To understand the genetic mechanisms involved in apparent or observed resistant selections

To better understand the underlying genetics involved in a host/pathogen interaction, one must rigorously control the genetics of both the host and pathogen. In the host this would include using either open-pollinated families or, better yet, specific controlled cross families (as mentioned above), and maybe even in conjunction with clonally replicated materials. It is also necessary that susceptible materials be kept in the system, not only for comparative purposes, but to provide the disequilibrium needed to observe trait variation in the host. This is important for understanding the genetic mechanisms in the host, especially if molecular marker technology is to be used to eventually identify the genes responsible for trait variation. On the pathogen side, in the case of *P. ramorum*, this may or may not be so critical. Although there seems to be considerable variation in *P. ramorum* in terms of pathogenicity/aggressiveness, the genetic data suggests that very few actual genotypes are present. It appears, for the most part, to be a clonally reproducing pathogen, however, natural mutation can play an important role in resistance-susceptibility dynamics, and even more so if resistance in the host is found to be fairly simply inherited, i.e., qualitative or single gene in nature. Durability of any apparent resistance to alternate pathotypes should be investigated, and in any case, if the other mating type becomes

well established and the pathogen population begins sexually reproducing, controlling the genetics of the pathogen, i.e., developing single genotype lines, should be given serious consideration.

Management or mitigating unacceptable losses to disease whose resistance is qualitatively and quantitatively expressed are drastically different. Quantitatively expressed systems are thought to be much more durable since many host genes are assumed to be involved in control of the resistance response and it is unlikely that a “super race” of the pathogen will evolve that will cause unacceptable losses. Whereas, qualitatively controlled systems are often considered to be much less durable. However, even qualitative resistance can be useful depending on the nature of the pathogen involved (McDonald and Linde 2002b).

Objective 3. Increase levels of resistance via breeding while maintaining a sufficiently large genetic base and develop strategies to intelligently deploy resistant materials to mitigate against unacceptable losses.

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