

Approaches to Understanding the Impact of Life-History Features on Plant-Pathogen Co-Evolutionary Dynamics

Jeremy J. Burdon,¹ Peter H. Thrall,¹ and Adnane Nemri¹

Abstract

Natural plant-pathogen associations are complex interactions in which the interplay of environment, host, and pathogen factors results in spatially heterogeneous ecological and epidemiological dynamics. The evolutionary patterns that result from the interaction of these factors are still relatively poorly understood. Recently, integration of the appropriate spatial and temporal context of the metapopulation has permitted an essential conceptual advance stressing the importance of these factors in evolutionary interactions. Yet, a major aspect of host and pathogen biologies that still needs considerable attention is that of the impact of particular life-history traits of both host and pathogen and how these may influence patterns of disease incidence and prevalence and the evolution of host resistance and pathogen infectivity. Here we outline the importance of both empirical comparisons and simulation modelling as a means of uncovering the full impact of host and pathogen life history traits on co-evolutionary dynamics.

Key words: empirical comparisons, simulation models, co-evolutionary dynamics

Introduction

Individual plant species frequently have wide geographic ranges that cover considerable latitude and distinctly different climatic zones. Yet, within particular regions their distribution is usually patchy and individual demes are often associated with local micro-environments showing specific moisture, temperature, and humidity regimes. Over the course of evolutionary history this mixture of broad and narrow scale environmental variation has provided a range of selective pressures across the space-time continuum. In turn this has generated a complex mosaic of genetic variation in the plant with differentiation within and among regions in a wide range of morphological (e.g., leaf shape and thickness), phenological (e.g., timing of bud-break, flowering), biochemical (e.g., lignin/terpene content), and other life-history characters (e.g., growth rate, life span, mating system). In a similar way, the biology and life-history characteristics of pathogens, particularly their responses to environmental cues, often show differentiation among and within regions. Among those traits, host specificity, transmission mode, life cycle complexity, and dispersal ability can have major influence on spread, diversity, and persistence of pathogen populations (Barrett et al. 2008a). At a broad level, these traits control the range of environments, and hence host populations in which pathogens may be found, the genetic structure of pathogen populations and, as a result, the extent to which they exert selective pressure on their host (Barrett et al. 2009).

Pathogens form associations with their hosts that cover part or all of the host's range (although, in some situations, a pathogen's range can exceed that of an individual host species if, as is the case for many necrotrophic species, they are capable of attacking/parasitizing more than one host). The epidemiological and evolutionary interaction that develops between host and pathogen in these associations is influenced by the detailed interplay of life-history differences at the individual patch and metapopulation level. Clearly, as the spatial scale of interactions expands across the full geographic distribution of the host, opportunities for differences in the nature of epidemiological outcomes occur both in different individual population associations and in different parts of the

¹ CSIRO-Plant Industry, GPO Box 1600, Canberra, ACT. 2601 Australia.
Corresponding author: jeremy.burdon@csiro.au.

overall geographic range of the interaction. Given the potential for stochastic colonization/extinction processes in partially isolated local populations and hence differences in the intensity and persistence of consequent selective pressures, this raises the possibility of the development of divergent longer-term evolutionary trajectories in different parts of the geographic range of individual host-pathogen associations.

Assessing the Impact of Host-Pathogen Life-History Features on the Epidemiological and Evolutionary Trajectory of Specific Interactions

How can we start to tease out whether or not life-history features affect the epidemiological and evolutionary trajectories of host-pathogen associations, and, if they do, the role of specific life-history characters? This would be an essential first step towards developing a more general conceptual framework. Importantly, the integration of information on host and pathogen life history has the potential to advance our predictive ability with regard to disease outbreak and emergence (Barrett et al. 2008a).

Thrall and Burdon (1997) developed a framework based on three realistic scenarios which illustrate the link between simple host and pathogen life history combinations and spatial considerations (spatial scale of associations; temporal predictability), and how these together are likely to influence the longer-term evolutionary trajectory of associations. Those scenarios recognized the existence of associations in which the scale of pathogen dispersal was likely to be (a) substantially less than that of its host (e.g., soil-borne pathogens); (b) roughly comparable with that of its host (e.g., seed borne viruses, vector-borne floral smuts); and (c) substantially greater than that of its host (e.g., wind-dispersed rusts, persistent aphid-borne viruses). Subsequently, they used simulation modelling to illustrate the importance of spatial scale and the occurrence of multiple demes showing varying degrees of interconnectedness in the longer-term ecological and evolutionary dynamics of hosts and their pathogens (Thrall and Burdon 1999, 2002).

Within pathogens themselves there is also a major life-history dichotomy between biotrophic and necrotrophic fungi. The former essentially rely on living host material for growth and, in its absence, surviving as relatively inert long-lived spores or other protected structures. Necrotrophic pathogens, on the other hand, exploit host tissue that they kill through toxin release. Unlike biotrophs, during off-season periods these pathogens may continue active growth by saprophytic exploitation of dead plant remains. This alternation between parasitic and saprophytic growth phases typically results in switches in the intensity, direction, and indeed the traits under selection – a situation that is markedly contrast to the patterns of selection experience by typical biotrophic pathogens.

Despite these differences, the most apparent way in which to assess the impact of life-history features is to characterize their influence on the epidemiology of the host-pathogen interaction and through this ultimately the reciprocal selective pressures applied by host and pathogen. The longer-term evolutionary consequences of such selective pressures are ultimately manifest in a variety of ways, but perhaps the clearest is through changes in the incidence of resistance in the host and pathogenicity in sympatric pathogen populations. Indeed, plants possess a range of different resistance mechanisms whereby they minimize their susceptibility to pathogens and the extent of damage they incur. Broadly speaking, these mechanisms can be categorized into qualitative or major gene resistance that is typically controlled by single genes with major phenotypic effects, and quantitative resistance that is usually controlled by many loci each with small phenotypic effects. In a classic genetic sense, qualitative resistance is characterized by Mendelian responses while quantitative resistance is typified by continuous variation. In real world host-pathogen systems, some plant populations may be protected against particular pathogens by one or other, or by a combination of these mechanisms.

Heuristic arguments have been developed (Burdon et al. 1996) as to the circumstances that may favor the evolution of one or other of these genetic mechanisms (largely reflecting the predictability of infection and its selective intensity), but as yet no concerted effort has been made to assess their incidence in natural systems and little is known about the selective and evolutionary forces that determine their balance. There are two clear ways in which to explore these possibilities. First, empirically through comparative investigation of situations where various combinations of host and pathogen species are assessed and contrasted, and second, through the development of simulation models that help develop a general conceptual framework for predicting outcomes in broad classes of plant-pathogen interactions.

Empirical Comparisons

In the first of these approaches, a number of combinations can be assessed. Thus:

One Host – One Pathogen; Distinctly Different Environments

Within single host-pathogen associations, correlations between parameters of pathogen dynamics (e.g., prevalence, severity, predictability) and the structure and nature of resistance occurring in contrasting host populations provide insights into the way spatio-temporal variation in selective intensity and predictability can shape host and pathogen population structure and life history features. An example of the insights provided by this sort of comparison is found in the *Populus – Melampsora* interaction occurring on the eastern and western sides of the Rocky Mountains. This interaction is one involving an annual host target (deciduous leaves) and hence one in which the pathogen tends to cycle through massive amplitudes in population size over short periods of time. Because the pathogen cannot survive on this host during the winter season, it is repeatedly forced to re-establish through migration from more benign environments or from alternate hosts. Under these circumstances, race-specific resistance genes which thwart initial entry of pathogen isolates carrying corresponding avirulence genes may provide adequate long-term resistance for the host. On the dry, cold eastern side of the Rocky Mountains in western Canada, this pattern of major gene resistance appears to predominate (Hsiang and Chastagner 1993). In contrast, west of the mountains, the more temperate winter environment means that the pathogen is constantly present, and only multigenic race non-specific resistance is found.

A similar potential direct effect of the environment on pathogen dynamics leading to varying selection pressures on associated hosts is seen in the interaction between *Avena fatua* (wild oats) and the rust fungus *Puccinia coronata* in Australia. In that situation, host and pathogen population pairs occurring in southern and northern New South Wales (NSW) respectively exhibit marked differences in the phenology of their interaction. In northern NSW, winters are warmer than further south and pathogen populations have the opportunity to develop earlier in the host's vegetative stage. As a consequence, earlier, more damaging disease epidemics are likely to ensue with the potential for greater selective impact on their hosts than in the south (Burdon et al. 1983). Consistent with this prediction, surveys reveal a marked increase in the incidence of resistance with major phenotypic effects in the more benign north.

One Host – One Pathogen; Distinctly Different Major Life History Feature Contrast

Mating system is possibly the life-history character that has the biggest single impact on populations of all organisms. It is at the core of the structure of plant populations (Barrett 2002; Barrett et al. 2008) and has been implicated as a determinant of the success of biological control programs to use insect herbivores and fungal pathogens to manage invasive weeds (Burdon and Marshall 1981). Indeed, it has long been hypothesized that differences in recombination systems provide hosts with quite different strategies for controlling their pathogens (cf., the Red Queen hypothesis; Hamilton 1980).

In some cases, lack of the opportunity for completing a full life cycle can present major impediments to the development of pathogen populations, which in turn influences the geographic distribution of resistance in host populations. For example, the rust fungus *Cronartium comptoniae* is

a full-cycle heteroecious rust fungus that naturally alternates between its telial and aecial hosts (*Myrica gale* and *Pinus contorta* respectively). Where the range of these two plant species overlaps, the pathogen is also present and significant levels of resistance are found in *P. contorta* populations. However, the geographic range of *P. contorta* is considerably greater than that of *M. gale* and outside their sympatric range, the pathogen is rarely found; populations of *P. contorta* are essentially susceptible (Hunt and Van Sickle 1984).

The presence or absence of sexual recombination may also have a marked effect on pathogen populations. Evidence for this is seen in two distinct examples – one from agriculture, the other from a naturally occurring interaction. In the first case, the contrast found between populations of wheat stem rust (*P. graminis* f.sp. *tritici*) in the Pacific Northwest and the Great Plains of the United States clearly demonstrates the contribution that sexual recombination makes to population diversity. In the Pacific North West, sexual recombination occurs annually leading to the generation of an enormous number of incipient clonal lineages, each with a distinctive genotype for both neutral (isozyme) and selected (pathogenicity) traits. A sample of 427 isolates collected from that population in 1975 contained 100 distinct pathotypes (Roelfs and Groth 1980), while among a sub-sample of 92 of those isolates (Burdon and Roelfs 1985), no two isolates with the same isozyme multilocus phenotype had the same combination of avirulence genes. When the possibility of sexual recombination is removed from such an interaction, population diversity falls dramatically and the way in which this diversity is distributed also changes markedly. Thus, further east in the Great Plains, the virtual eradication of barberry (the alternate host required to complete the sexual cycle) during the late 1920s generated significant genetic changes. On a yearly average, the number of distinct pathotypes fell by more than half while those that did occur could be grouped into a number of distinct clonal lineages that differ from one another by an average of 11 virulences and various isozyme markers (Roelfs and Groth 1980; Burdon and Roelfs 1985).

A not dissimilar effect is seen in the interaction between the herbaceous perennial *Linum marginale* and an associated rust pathogen (*Melampsora lini*) where differences in the extent of sexual recombination are found between montane and plains populations of both species. In montane regions the rust pathogen is trapped in an asexual cycle (Burdon and Roberts 1995), where it undergoes severe population crashes each winter; its sympatric hosts also show a high level of selfing. In contrast, on the plains, host populations show significant levels of outcrossing (~30 percent; Burdon et al. 1999). Although their sympatric pathogen populations face a summer survival bottleneck, this is ameliorated by the pathogen's ability to survive via telia and subsequently undergo sexual recombination (Barrett et al. 2008b). Comparisons of the resistance and avirulence structure of host and pathogen populations respectively showed significant differences in the diversity of pathogen populations between the two regions and the way in which resistance genes were distributed within and among host populations.

Two Closely Related But Different Hosts – One Pathogen

While the examples above give hints of the potential impact of mating system on host and pathogen diversity, and have strength as a result of their focus on the same host and pathogen species, their value is simultaneously weakened by the lack of a common environment. In contrast, analogous examples in a common environment could be achieved by comparing patterns of resistance and pathogenicity in two closely related, co-occurring host species which differ markedly in a particular life history feature.

To our knowledge, no such comparisons have yet been made, although a number of potential species comparisons exist. One such example occurs in Australia and New Zealand where the two closely related beach strandline species *Cakile edentula* (tight inbreeder) and *C. maritima* (obligate outcrosser) are both attacked by the necrotrophic pathogen, *Alternaria brassicicola*, making this an ideal system to investigate the evolution of resistance in individual host populations with contrasting recombination constraints (Cousens and Cousens 2011). Furthermore, data already exists for the *C. maritima* – *A. brassicicola* component of this potential comparison that clearly demonstrates the

occurrence of severe pathogen epidemics, and the existence of variation in both host resistance and pathogen pathogenicity (Oliver et al. 2001; Thrall et al. 2001) - both necessary pre-conditions for pathogens to have a detectable role in the evolution of their hosts.

A not dissimilar example is found in the work of Sicard and her colleagues (2007) who assessed evidence for local adaptation in infectivity and aggressiveness in the pathogen *Colletotrichum lindemuthianum* attacking three native Mexican populations of *Phaseolus vulgaris*. This interaction was elaborated with a second association in which the pathogen was the same and the host closely related (*P. coccineus* – *Colletotrichum lindemuthianum*). The two host species differed in a number of life-history features – perenniality, population size and stability, and degree of outcrossing – but, while the pathogen showed evidence of local adaptation in its interaction with the annual *P. vulgaris*, it did not when attacking the longer-lived *P. coccineus* which is also predominately out-crossing.

One Host – Two Contrasting Pathogens

Biotrophic and necrotrophic pathogens exhibit distinct differences in their relationship with their hosts, especially with respect to survival. Biotroph populations (e.g., many rusts and mildews) typically undergo periodic seasonal crashes with individual demes within a metapopulation going extinct or being subject to marked genetic drift from one year to the next. Necrotroph populations, on the other hand, are usually far less affected by environmental change as they typically can survive in a saprophytic phase on dead plant material or generate persistent infections of perennial parts of the plant. These different life history strategies have been predicted to generate different epidemiological patterns and impose distinctly different selective regimes on hosts (Burdon et al. 1996) and their pathogens. Indeed, as demonstrated in the barley scald pathogen *Rhynchosporium secalis*, selection pressures may be quite different between parasitic and saprophytic phases of the life cycle (Abang et al. 2006).

Examples of this category of host–pathogen interaction abound and could include attack by different leaf pathogens [e.g., Meadowsweet (*Filipendula ulmaria*) attacked by *Melampsora* (biotrophic rust) and *Ramularia* (necrotrophic leaf blight)] or by pathogens attacking different organs [e.g., Poplar (*Populus* spp.) attacked by *Melampsora* (leaf rust) and *Hypoxyylon* (stem canker)]. Thus, in the latter comparative set of host and pathogens, depending on the environment, both race-specific and more broadly based non-specific resistance has been detected in the interaction between *Populus* and *Melampsora* (Hsiang and Chastagner 1993, Prakash and Thielges 1987; see discussion above). In contrast, once established, the interaction between *Populus* and the stem canker fungus *Hypoxyylon* is protected from environmental fluctuations, and progress through the tree is primarily determined by characters which are controlled through the combined action of multiple genes (French and Manion 1975).

Simulation Modelling and Meta-Analyses

In contrast to animal host-parasite and agriculturally based plant-pathogen interactions, there has been relatively little use of theoretical models to explore aspects of the impact of life-history characters on the epidemiological or evolutionary trajectory of natural plant-pathogen associations. However, the development of a general conceptual framework is critical for understanding links between different systems. For example, much of the current general theoretical work on animal host-parasite dynamics has been based on models that differentiate between two broad classes of parasites: micro- and macroparasites (Anderson and May 1981). Not only are there qualitative dynamical differences that emerge from these models, but this work has led to the recognition that different factors must be considered for these two broad classes of host-parasite systems.

A perceived lack of comparable generalizing principles with respect to natural plant-pathogen dynamics is probably due, in part, to a combination of the great range of plant pathogens and their diverse life histories (e.g., multiple hosts, free-living stages, vectors, vertical transmission, mortality vs. fecundity effects, epidemic vs. endemic) and the fact that study of plant-pathogen interactions has tended to arise from agriculture and forestry which focused more on traditional plant pathology approaches than on the tools of population biology. However, similar to the useful sets of

generalizations that have been made for animal diseases (Lockhart *et al.* 1996), it seems likely that plant pathogens can also be divided into broad categories recognizing fundamentally different effects that are likely to transcend taxonomic categories. Thus even very general comparisons between associations involving (i) systemic diseases of plants with varying degrees of vegetative reproduction (Wennström and Ericson 1992); (ii) sporulation and infection characteristics in airborne pathogens (Sache and de Vallavielle Pope 1995); (iii) pathogen dispersal scale (Thrall and Burdon 1997); or differences in host fecundity and mortality effects (Burdon 1993) all illustrate the potential for life history differences to have consequences for co-evolutionary dynamics.

Indeed, an early comparative study assessing the success of biological control programs aimed at reducing the size of invasive weed populations demonstrated that the reproductive mode of the target species was an important factor in the success of biological control with species with low or no recombination being more vulnerable to pathogens than those with high levels of recombination (Burdon and Marshall 1981). More recently, some simple comparative models have been developed to generate insights into the most appropriate types of pathogen to deploy in biological control of weeds. Using a spatially explicit simulation modelling approach built around simple single life-history difference comparisons such as whether a pathogen has its impact solely through host fecundity or host mortality, Thrall and Burdon (2004) found that even with such broad caricatures, there were profound impacts on host population size and persistence, and the potential for evolution of host resistance. Importantly, the effect of these pathogen traits was itself mediated by host traits such as longevity. Other simulation studies, supported by some data from natural plant-pathogen interactions, also predict that increasing host longevity and connectivity of host patches is likely to select for pathogen strains with lower transmission rates as well as for hosts with higher resistance (Carlsson-Granér and Thrall 2002, 2006).

Many host and pathogen life-history traits are likely to have at least some impact on the intensity of ecological and evolutionary forces affecting individual associations. However, the nature and sign of those effects is often less than intuitively obvious, especially when such interactions are considered in realistic ecological settings involving spatially patchy, small, and ephemeral host populations in which genetic drift, extinction, and re-colonization are relatively frequent occurrences. In these circumstances, simulation modelling is a very powerful means of exploring the impact of variation in key host and pathogen life history features on patterns of disease incidence and prevalence and likely changes in the intensity of selective pressures leading to evolution of variation in resistance and virulence. Overall, a simulation modelling approach, particularly one that incorporates both demographic and genetic processes, will help understanding of how interactions between host and pathogen life-histories influence:

- a) pathogen invasion and spread and the degree to which dynamics are endemic vs. epidemic;
- b) levels of impact on host demography and fitness;
- c) conditions favoring the evolution of different genetic systems of resistance and pathogenicity;
- d) the maintenance of polymorphisms; and
- e) correlated life histories of hosts and pathogens that are likely to favor long-term associations.

Conclusions

Individually empirical comparisons and simulation models both have their strengths and weaknesses. While comparisons in which one or more life history traits can be held constant provide powerful clues to the interactive effects of life history characters and host spatial heterogeneity in the long-term development of co-evolutionary trajectories of specific associations, at the same time, empirical testing of the causal relationships between, and polarity of, particular life-history attributes and the structure of host and pathogen populations is always going to be very difficult. Indeed, ultimately, unless sufficient comparisons are available to allow rigorous meta-analyses to be conducted, such studies will remain open to criticism that observed patterns are due to nothing more than chance or uncontrolled variation in other key factors. Stimulation of a broader range of empirical studies on

natural host-pathogen interactions and the development of rigorous meta-analyses represent clear research challenges for the immediate future.

Similarly, simulation modelling can be extremely powerful in setting up direct comparisons between specific life-history traits and in uncovering counter-intuitive interactions. However, at the same time such models also suffer from an inevitable lack of biological reality, rapidly becoming unwieldy and complex as more variables are added.

The two approaches we advocate here – empirical comparisons and simulation modelling – are essential companion components in developing a strong conceptual framework grouping pathogens with similar ecologies. Doing this will stimulate the development of theory, provide testable hypotheses for empirical studies, and place the relative differences between long-term coevolved systems, recent exotic invasions, novel emerging pathogens, and biological control programs into a generally understandable context.

Acknowledgments

This research was supported by the National Institutes of Health (NIH grant 5RO1 GM074265-01A2).

Literature Cited

- Abang, M.M.; Baum, M.; Ceccarelli, S.; Grando, S.; Linde, C.C.; Yahvaoui, A.; Zhan, J.S.; McDonald, B.A. 2006.** Differential selection on *Rhynchosporium secalis* during parasitic and saprophytic phases in the barley scald disease cycle. *Phytopathology*. 96: 1214–1222.
- Anderson, R.M.; May, R.M. 1991.** The population dynamics of micro-parasites and their invertebrate hosts. *Philosophical Transactions Royal Society of London, Series B, Biological Sciences*. 291: 451–524.
- Barrett, L.G.; Kniskern, J.M.; Bodenhausen, N.; Zhang, W.; Bergelson, J. 2009.** Continua of specificity and virulence in plant host-pathogen interactions: causes and consequences. *New Phytologist* 183: 513–529.
- Barrett, L.G.; Thrall, P.H.; Burdon, J.J.; Linde, C.C. 2008a.** Life history determines genetic structure and evolutionary potential of host-parasite interactions. *Trends in Ecology and Evolution*. 23: 678–685.
- Barrett, L.G.; Thrall, P.H.; Burdon, J.J.; Linde, C.C.; Nicotra, A.B. 2008b.** Population structure and diversity across sexual and asexual populations of the pathogenic fungus *Melampsora lini*. *Molecular Ecology*. 17: 3401–3415.
- Barrett, S.C.H. 2002.** The evolution of plant sexual diversity. *Nature Reviews: Genetics*. 3: 274–284.
- Barrett, S.C.H.; Colautti, R.I.; Eckert, C.G. 2008.** Plant reproductive systems and evolution during biological invasion. *Molecular Ecology*. 17: 373–383.
- Burdon, J.J. 1993.** The role of parasites in plant populations and communities. In: Schulze, E.-D.; Mooney, H.A., eds. *Biodiversity and ecosystem function*. Berlin: Springer-Verlag: 165–179.
- Burdon, J.J.; Marshall, D.R. 1981.** Biological control and the reproductive mode of weeds. *Journal of Applied Ecology*. 18: 649–658.
- Burdon, J.J.; Oates, J.D.; Marshall, D.R. 1983.** Interactions between *Avena* and *Puccinia* species. I. The wild hosts: *Avena barbata* Pott ex Link, *A. fatua* L. and *A. ludoviciana* Durieu. *Journal of Applied Ecology*. 20: 571–585.
- Burdon, J.J.; Roberts, J.K. 1995.** The population genetic structure of the rust fungus *Melampsora lini* as revealed by pathogenicity, isozyme and RFLP markers. *Plant Pathology*. 44: 270–278.
- Burdon, J.J.; Roelfs, A.P. 1985.** The effect of sexual and asexual reproduction on the isozyme structure of populations of *Puccinia graminis*. *Phytopathology*. 75: 1068–1073.
- Burdon, J.J.; Thrall, P.H.; Brown, A.H.D. 1999.** Resistance and virulence structure in two *Linum marginale* - *Melampsora lini* host-pathogen metapopulations with different mating systems. *Evolution*. 53: 704–716.
- Burdon, J.J.; Wennström, A.; Elmqvist, T.; Kirby, G.C. 1996.** The role of race specific resistance in natural plant populations. *Oikos*. 76: 411–416.
- Carlsson-Granér, U.; Thrall, P.H. 2002.** The spatial distribution of plant populations, disease dynamics and evolution of resistance. *Oikos*. 97: 97–110.

- Carlsson-Granér, U.; Thrall, P.H. 2006.** The impact of host longevity on disease transmission: host-pathogen dynamics and the evolution of resistance. *Evolutionary Ecology Research*. 8: 659–675.
- Cousens, R.D.; Cousens, J.M. 2011.** Invasion of the New Zealand coastline by European sea-rocket (*Cakile maritima*) and American sea-rocket (*Cakile edentula*). *Invasive Plant Science and Management*. 4: 260–263.
- French, J.R.; Manion, P.D. 1975.** Variability of host and pathogen in Hypoxylon canker of aspen. *Canadian Journal of Botany*. 53: 2740–2744.
- Hamilton, W.D. 1980.** Sex versus non-sex versus parasite. *Oikos*. 35: 282–290.
- Hsiang, T.; Chastagner, G.A. 1993.** Variation in *Melampsora occidentalis* rust on poplars in the Pacific Northwest. *Canadian Journal of Plant Pathology*. 15: 175–181.
- Hunt, R.S.; Van Sickle, G.A. 1984.** Variation in susceptibility to sweet fern rust among *Pinus contorta* and *P. banksiana*. *Canadian Journal of Forestry Research*. 14: 672–675.
- Lockhart, A.B.; Thrall, P.H.; Antonovics, J. 1996.** Sexually transmitted diseases in animals: ecological and evolutionary implications. *Biological Reviews of the Cambridge Philosophical Society*. 71: 415–471.
- Oliver, E.J.; Thrall, P.H.; Burdon, J.J.; Ash, J.E. 2001.** Vertical disease transmission in the *Cakile-Alternaria* host-pathogen interaction. *Australian Journal of Botany*. 49: 561–569.
- Prakash, C.S.; Thielges, B.A. 1987.** Pathogenic variation in *Melampsora medusae* leaf rust of poplars. *Euphytica*. 36: 563–570.
- Roelfs, A.P.; Groth, J.V. 1980.** A comparison of virulence phenotypes in wheat stem rust populations reproducing sexually and asexually. *Phytopathology*. 70: 855–862.
- Sache, I.; de Vallavielle Pope, C. 1995.** Classification of airborne plant pathogens based on sporulation and infection characteristics. *Canadian Journal of Botany*. 73: 1186–1195.
- Sicard, D.; Pennings, P.S.; Grandclément, C.; Acosta, J.; Kaltz, O.; Shykoff, J.A. 2007.** Specialization and local adaptation of a fungal parasite on two host species as revealed by two fitness traits. *Evolution*. 61: 27–41.
- Thrall, P.H.; Burdon, J.J. 1997.** Host-pathogen dynamics in a metapopulation context: the ecological and evolutionary consequences of being spatial. *Journal of Ecology*. 85: 743–753.
- Thrall, P.H.; Burdon, J.J. 1999.** The spatial scale of pathogen dispersal: consequences for disease dynamics and persistence. *Evolutionary Ecology Research*. 1: 681–701.
- Thrall, P.H.; Burdon, J.J. 2002.** Evolution of gene-for-gene systems in metapopulations: the effect of spatial scale of host and pathogen dispersal. *Plant Pathology*. 51: 169–184.
- Thrall, P.H.; Burdon, J.J. 2004.** Host-pathogen life-history interactions affect biological control success. *Weed Technology*. 18: 1269–1274.
- Thrall, P.H.; Burdon, J.J.; Bock, C.H. 2001.** Short-term epidemic dynamics in the *Cakile maritima* – *Alternaria brassicicola* host-pathogen metapopulation association. *Journal of Ecology*. 89: 723–735.
- Wennström, A.; Ericson, L. 1992.** Environmental heterogeneity and disease transmission within clones of *Lactuca sibirica*. *Journal of Ecology* 80: 71–77.